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Direct Risks to Human Health and Welfare

The activist is not the man who says the river is dirty.
The activist is the man who cleans up the river.

Ross Perot (1930–),
U.S. entrepreneur and politician

Much of the original concern with environmental pollution was that it was aesthetically displeasing and a nuisance. Certainly, a few Ancients in Egypt, Rome, and elsewhere did notice the relationship between exposure to pollutants and poor health. However, it was not until the late 1970s that risk was to become the dominant means of determining environmental effects. Thus, this new paradigm called for the comparison of one person's or group's risks to the risk in other groups as a means of determining whether that person or group is being inordinately exposed or placed at risk. An inordinately high exposure or risk is a first indication of injustice. This puts engineers in a pivotal position in environmental justice. Engineers are key players, ''activists'' if you will, as they search for ways to reduce environmental risks to highly exposed and sensitive groups. This proactive role is best articulated in the first canon of our codes of ethics: We must hold paramount the health, safety, and welfare of the public. We spend our careers providing these public services and finding ways to ensure that what we design is safe and does not detract from the public good.

In this chapter we deal with two concepts important to all engineering: risk and reliability. The principal value added by environmental engineers and other environmental professionals is in the improvement in the quality of human health and ecosystems. Environmental professionals do not have a monopoly on risk reduction, and in fact, all engineers can play a role in the enhancement of environmental quality.

Engineers add value when we decrease risk, so risk is one of the best ways to measure the success of engineers whose projects address environmental injustices. By extension, reliability tells us and everyone else just how well we go about preventing pollution, lowering the amounts of pollutants to which people are exposed, protecting ecosystems, and reducing overall risk. What we design must continue to serve its purpose throughout its useful life, in a manner sensitive to public health and environmental quality.

As it is generally understood, risk is the chance that something will go wrong or that some undesirable event will occur. Every time we get on a lawn tractor, for example,

 ISBN: 978-0-471-78707-5

we are taking a risk that we might be in an accident and damage the tractor, get hurt, injure others, or even die in a mishap. The understanding of the factors that lead to a risk is called *risk analysis* and the reduction of this risk (e.g., by following the safety procedures delineated in the owner's manual and staying off steep grades) is *risk management.* Risk management is often differentiated from *risk assessment,* which is comprised of the scientific considerations of a risk.[1] Risk management includes the policies, laws, and other societal aspects of risk.

Engineers engage constantly in risk analysis, assessment, and management. Engineers must consider the interrelationships among factors that put people at risk, suggesting that we are risk analysts. Engineers provide decision makers with thoughtful studies based on sound application of the physical sciences and, therefore, are risk assessors by nature. Engineers control things and, as such, are risk managers. We are held responsible for designing safe products and processes, and the public holds us accountable for its health, safety, and welfare. The public expects engineers to ''give results, not excuses,''[2] and risk and reliability are accountability measures of engineers' success. Engineers design systems to reduce risk and look for ways to enhance the reliability of these systems. Consequently, every engineer deals directly or indirectly with risk and reliability.

Thus, environmental justice embodies the concept of risk and how it can be quantified and analyzed. It also considers ways of reducing risk by conscious and intended risk management and how to communicate both the assessment and management options to those affected.[3]

RISK AND RELIABILITY

Probable impossibilities are to be preferred to improbable possibilities.
Aristotle

Aristotle was not only a moral philosopher and natural philosopher (the forerunner to ''scientist''); he was also a risk assessor. In the business of human health and environmental protection, we are presented with ''probable impossibilities'' and ''improbable possibilities.''

To understand these two outcomes, we must first understand the different connotations of *risk*. Aristotle's observation is an expression of probability. People, at least intuitively, assess risks and determine the reliability of their decisions every day. We want to live in a safe world; but *safety* is a relative term. The ''safe'' label requires a value judgment and is always accompanied by uncertainties, but engineers frequently characterize the safety of a product or process in objective and quantitative terms. Factors of safety are a part of every design. Environmental safety is usually expressed by its opposite term, risk.

Discussion: Probability—The Mathematics of Risk and Reliability

Probability is the likelihood of an outcome. The outcome can be bad or good, desired or undesired. The history of probability theory, like much modern math-

ematics and science, is rooted in the Renaissance. Italian mathematicians considered some of the contemporary aspects of probability as early as the fifteenth century, but did not need, or were unable, to devise a generalized theory. Blaise Pascal and Pierre de Fermat, famous French mathematicians, developed the theory after a series of letters in 1654 considering some questions posed by a nobleman, Antoine Gombaud, Chevalier de Méré, regarding betting and gaming. Other significant Renaissance and post-Renaissance mathematicians and scientists soon weighed in, with Christian Huygens publishing the first treatise on probability, *De Ratiociniis in Ludo Aleae,* which was specifically devoted to gambling odds. Jakob Bernoulli (1654–1705) and Abraham de Moivre (1667–1754) also added to the theory. However, it was not until 1812 with Pierre Laplace's publication of *Théorie analytique des probabilités,* that probability theory was extended beyond gaming to scientific applications.[4]

Probability is now accepted as the mathematical expression that relates a particular outcome of an event to the total number of possible outcomes. This is demonstrated when we flip a coin. Since the coin has only two sides, we would expect a 50–50 chance of either a head or a tail. However, scientists must also consider rare outcomes, so there is a very rare chance (i.e., highly unlikely, but still possible) that the coin could land on its edge (i.e., the outcome is neither a head nor a tail), A "perfect storm" of a confluence of unlikely events is something that engineers must always consider, such as the combination of factors that led to major disasters such as Hurricane Katrina and the toxic cloud in Bhopal, India, or the introduction of a seemingly innocuous opportunistic species (e.g., Iron Gates Dam in Europe) that devastates an entire ecosystem, or the interaction of one particular congener of a compound in the right cell in the right person that leads to cancer. As engineers, we also know that the act of flipping or the characteristics of the coin may tend to change the odds. For example, if for some reason the heads is heavier than the tails side or the aerodynamics is different, the probability could change.

The total probability of all outcomes must be unity (i.e., the sum of the probabilities must be 1). In the case of the coin standing on end rather than being a head or a tail, we can apply a quantifiable probability to that rare event. Let us say that laboratory research has shown that 1 in a million times ($1/1{,}000{,}000 = 0.000001 = 10^{-6}$), the coin lands on edge. By difference, since the total probabilities must equal 1, the other two possible outcomes (heads and tails) must be $1 - 0.000001 = 0.999999$. Again, we are assuming that the aerodynamics and other physical attributes of the coin give it an equal chance of being either a head or a tail, the probability of a head = 0.4999995 and the probability of a tail = 0.4999995.

Stated mathematically, an event (e) is one of the possible outcomes of a trial (drawn from a population). In our coin-toss case, all events, head, tail, and edge, together form a finite *sample space,* designated as $E = [e_1, e_2, \ldots, e_n]$. The lay public is not generally equipped to deal with such rare events, so by convention, they usually ignore them. For example, at the beginning of overtime in a football game, a tossed coin determines who will receive the ball and thus have the first opportunity to score and win. When the referee tosses the

coin, there is little concern about anything other than heads or tails. However, the National Football League undoubtedly has a protocol for the rare event of the coin not being a discernable head or tail. In environmental studies, *e* could represent a case of cancer. Thus, if a population of 1 million people is exposed to a pesticide over a specific time period, and one additional cancer is diagnosed that can be attributed to that pesticide exposure, we would say that the probability of *e* (i.e., $p\{e\}$) $= 10^{-6}$. Note that this was the same probability that we assigned to the coin landing on its edge.

Returning to our football example, the probability of the third outcome (a coin on edge) is higher than "usual" since the coin lands in grass or artificial turf compared to landing on a hard flat surface. Thus, the physical conditions increase the relative probability of the third event. This is analogous to a person who may have the same exposure to a carcinogen as the general population, but who may be genetically predisposed to develop cancer. The exposure is the same, but the probability of the outcome is higher for this "susceptible" person. Thus, risk varies by both environmental and individual circumstances.

Events can be characterized a number of ways. Events may be discrete or continuous. If the event is forced to be one of a finite set of values (e.g., six sides of a die), the event is discrete. However, if the event can be any value [e.g., size of tumor (within reasonable limits)], the event is continuous. Events can also be independent or dependent. An event is *independent* if the results are not influenced by previous outcomes. Conversely, an event affected by any previous outcome is a *dependent* event.

Joint probabilities must be considered and calculated since in most environmental scenarios, events occur in combinations. So if we have *n* mutually exclusive events as possible outcomes from *E* that have probabilities equal to $p\{e_i\}$, the probability of these events in a trial equals the sum of the individual probabilities:

$$p\{e_i \text{ or } e_2 \cdots \text{or } e_k\} = p\{e_1\} + p\{e_2\} + \cdots + p\{e_k\} \tag{4.1}$$

Further, this helps us to find the probabilities of events e_i and g_i for two independent sets of events, *E* and *G,* respectively:

$$p\{e_i \text{ or } g_i\} = p\{e_i\}p\{g_i\} \tag{4.2}$$

For example, a company record book indicates that a waste site has 10 unlabeled buried chemical drums: five drums that contain mercury (Hg), two drums that contain chromium (Cr), and three drums that contain tetrachloromethane (CCl_4). We can determine the probability of pulling up one of the drums that contains a metal waste (i.e., Hg or Cr). The two possible events (Hg drum or Cr drum), then, are mutually exclusive and come from the same sample space; so we can use equation (4.1):

$$p\{\text{Hg or Cr}\} = p\{\text{Hg}\} + p\{\text{Cr}\} = \frac{5}{10} + \frac{2}{10} = \frac{7}{10}$$

Thus, we have a 70% probability of pulling up a metal-containing drum.

If we have another waste site that also has 10 unlabeled, buried drums—three drums that contain dichloromethane (CH_2Cl_2) and seven drums that contain trichloromethane ($CHCl_3$)—we calculate the probability of pulling up a chromium drum from our first site and a $CHCl_3$ drum from the second site. Since the two trials are independent, we can use equation (4.2):

$$p\{\text{Cr and } CH_2Cl_2\} = p\{\text{Hg}\} + p\{\text{Cr}\} = \frac{2}{10} \times \frac{3}{10} = \frac{6}{100}$$

Thus we have 6% probability of extracting a chromium and a dichloromethane drum on our first excavation.

Another important concept for environmental data is that of conditional probability. If we have two dependent sets of events, E and G, the probability that event e_k will occur if the dependent event g has occurred previously can be shown as $p\{e_k|g\}$, which is found using Bayes' theorem:

$$p\{ek|g\} = \frac{p\{e_k \text{ and } g\}}{p\{g\}} = \frac{p\{g|e_k\}p\{e_k\}}{\sum_{i=1}^{n} p\{g|e_i\}p\{e_i\}} \tag{4.3}$$

A review of this equation shows that conditional probabilities are affected by a cascade of previous events. Thus, the probability of what happens next can be highly dependent on what occurred previously. For example, the cumulative risk of cancer depends on the serial (dependent) outcomes. Similarly, reliability can also be affected by dependencies and prior events. Thus, characterizing any risk or determining the reliability of our systems are expressions, at least in part, of probability.

Engineers are comfortable with equations, so another way to present probabilities to characterize risk and reliability is by showing a *probability density function* (PDF) for data. The PDF is created from a probability density; that is, when the data are plotted in the form of a histogram, as the amount of data increases, the graph increases its smoothness (i.e., the data appear to be continuous). The smooth curve can be expressed mathematically as a function, $f(x)$. This is the PDF. The probability distribution can take many shapes, so the $f(x)$ for each will differ accordingly. For example, in environmental matters, distributions commonly seen are normal, log-normal, and Poisson. The normal (Gaussian) distribution is symmetrical and is best known as the *bell curve,* given its shape (see Figure 4.1). The log-normal distribution is also symmetrical, but its x-axis is plotted as a logarithm of the values.

The Poisson distribution is a representation of events that happen with relative infrequency, but regularly.[5] Stated mathematically, the Poisson distribution function expresses the probability of observing various numbers of a particular event in a sample when the mean probability of that event on any one trial is very small. So the Poisson probability distribution characterizes discrete events that occur independent of one another during a specific period of time. This is useful for risk assessments, since exposure-related measurements can be expressed as a rate of discrete events: the number of times that an event happens during a defined time interval (e.g., the frequency (times per week) during

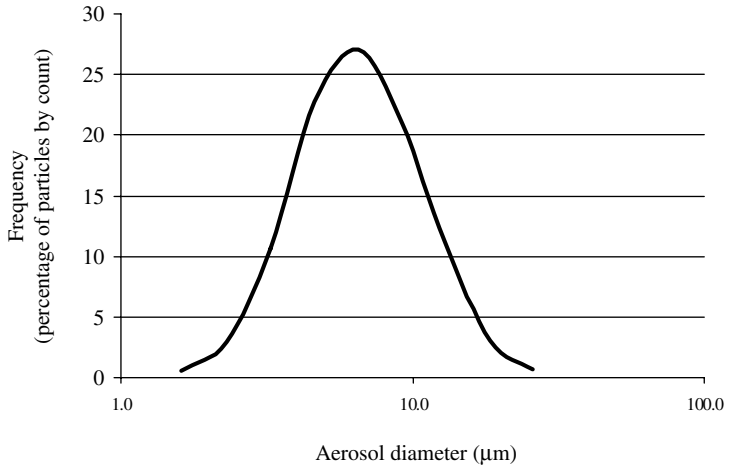


Figure 4.1 Distribution (log normal) of aerosols in the lower troposphere. (Data from L. Silverman, C. E. Billings, and M. W. First, *Particle Size Analysis in Industrial Hygiene,* Academic Press, New York, 1971.)

which a person eats shellfish that contain polychlorinated biphenyls (PCBs) in fish containing methyl mercury concentrations greater than 5.0 mg L^{-1}. The Poisson distribution describes events that take place during a fixed period of time (i.e., a rate), as long as the individual events are independent of each other. As the expected number of events or counts increases (i.e., the event rate increases), so does variability. Obviously, if we expect a count to equal 1, we should have little trouble picturing an observation of 2 or 0. If we expect a count equal to 50,000, counts of 49,700 and 50,300 are within reason. The range and variance of the latter, however, is much larger. The Poisson equation needed to compute the probability of a specific number of counts being observed over a defined time interval is

$$P_{\lambda} = \frac{e^{-\pi}\lambda^{n}}{n!} \tag{4.4}$$

where λ is the average or expected counts or events per unit time and n is the number of encounters.

Thus, the Poisson distribution is useful in a risk assessment to estimate exposures. It may be used to characterize the frequency with which a person (or animal or ecosystem) comes into contact with a substance (e.g., the number of times per day a person living near a wood treatment facility is exposed to pentachlorophenol. Assuming that based on existing data, the expected number of encounters is two per day, applying equation (4.4) with $\lambda = 2$, there is a 9% chance that a person will have 4 (i.e., $n = 4$) encounters with pentachlorophenol on a given day.

Risk itself is an expression of a probability (i.e., the chance of an adverse outcome). It is the probability of a consequence. So any calculation of environmental insult can be based on some use of probabilities.

Biographical Sketch: Abdul Q. Khan

Abdul Qadeer Khan was born in Bhopal, India, in 1936 to a middle-class Muslim family. At the time the population of Bhopal was made up of both Muslim and Hindus who lived an uneasy but peaceful coexistence. When India was partitioned in 1947, forming Pakistan, a huge migration occurred where Muslims moved north to Pakistan, and Hindus moved south to India. Abdul Khan, one of seven children, migrated with his family in 1952. They were harassed, beaten, and robbed during their trip and he ended up walking barefoot to Pakistan. The experience caused him to have a lifelong distrust and hatred of India.

Khan went to the University of Karachi in Pakistan and then to universities in West Germany and Belgium. After graduating with a PhD in metallurgical engineering Khan joined the staff of the Physical Dynamics Research Laboratory, or FDO, in Amsterdam, The Netherlands. FDO was a subcontractor for URENCO, a British-Dutch-German consortium specializing in the manufacture of equipment for the enrichment of uranium.

By 1974 India had developed and demonstrated their nuclear bombs and was threatening Pakistan. Taking advantage of an insurrection in Eastern Pakistan, the Indian army had already soundly defeated the Pakistani forces, resulting in the formation of a new country, Bangladesh.

Demoralized and stinging from their defeat in the Indo-Pakistani War, the political and military leaders of Pakistan concluded that they also had to have nuclear weapons to serve as a deterrent for the threat they perceived to be coming from India. In 1975 A.Q. Khan was reportedly asked by the then Prime Minister of Pakistan to develop a uranium-enrichment program for Pakistan. Using his position at FDO as an engineer working on uranium enrichment machinery, Khan began to steal classified documents and to send them to Pakistan. His actions eventually raised suspicions, but he escaped to Pakistan before he could be arrested. Once safely in Pakistan, he was asked by the government to establish the laboratory that would produce the nuclear device. Using his contacts and an unlimited budget, Khan's lab was able to enrich uranium by the late 1980s leading to the successful detonation of Pakistan's first nuclear device on 28 May 1998. For his efforts Khan became a national hero, living a life of privilege and amassing numerous honors and adulations.

In the late 1990s reports began to be made public accusing Khan and others at his lab of selling nuclear secrets, equipment and material to countries such as Libya, Iran, Malaysia, and North Korea, allegedly in return for tens of millions of dollars. In 2003 the Pakistani government began an investigation, at the prompting of the US government, into Khan and his lab. In January 2004 Khan was placed under house arrest and remains under detention to this day. President Musharraf of Pakistan subsequently pardoned Khan of any wrongdoing citing his service to Pakistan, but this was widely perceived as a face-saving exercise to deflect any blame from government officials, and to salvage the reputation of a popular national hero.

Abdul Khan's story demonstrates how engineers, using their technical skills and blinded by both wealth and nationalism, can forget their primary role of service to society. The sale of nuclear technology to countries that may use this knowledge to terrorize others was unconscionable.

Engineering success or failure is in large measure determined by comparing what we do to what our profession ''expects'' of us. Safety is a fundamental facet of our engineering duties. Thus, we need a set of criteria that tells us when our designs and projects are sufficiently safe. Four criteria are applied to test engineering safety:[6]

1. The design must comply with applicable laws.
2. The design must adhere to ''acceptable engineering practice.''
3. Alternative designs must be sought to see if there are safer practices.
4. Possible misuse of the product or process must be considered and estimated.

The first two criteria are usually more manageable than the latter two. The engineer can look up the physical, chemical, and biological factors to calculate tolerances and factors of safety for specific designs. Laws and regulations are promulgated to protect the public. Crossing these legal thresholds indicates the point at which the engineer has failed to provide adequate protection. Engineering practice standards go a step further. Much of the public and its lawyers would not be able to recognize this type of failure unless other engineers help to judge whether an ample margin of safety has been met. The margin is dictated by sound engineering principles and practice. However, finding alternatives and predicting misuse and mistakes require creativity and imagination. These criteria correspond closely with the five types of failure discussed in Chapter 3. In fact, a corollary to the fourth criteria could be added to contemporary engineering: intentional and willful misuse (e.g., intentional environmental injustices, terrorism), which is now a design criterion that must be considered by every engineer.

If one were to query a focus group as to whether risk can be quantified, the group is usually divided. At first thought, most respondents consider risk not to be quantifiable. The general consensus, at least in our unscientific queries, is that one person's risk is different from another's; risk is in the ''eye of the beholder.'' Some of the rationale appears to be rooted in the controversial risks of tobacco use and daily decisions, such as choice of modes of transportation.

Discussion: Choose Your Route of Exposure

Old man, look at my life, twenty four and there's so much more . . .
Give me things that don't get lost.
Like a coin that won't get tossed. . .
Old Man, Neil Young (1945–)

Young, the songwriter, seems be talking about risk, especially how it changes with age and how, when we are younger, our acceptance of risks may be rather high. Perhaps, since Young was about 25 years old when he wrote *Old Man,* he was displaying cognitive dissidence: on the one hand, wanting to take risks, but on the other, recognizing risk avoidance as a necessity in some matters (i.e., a coin that won't get tossed). An interesting phenomenon that supports this view seems to be taking place on today's college campuses. From

some anecdotal observations, it would appear that students are more concerned about some exposure pathways and routes than others. It is not uncommon at Duke or Bucknell University, for example, to observe a student getting off her bicycle to smoke a cigarette and to take a few drinks of name-brand bottled water.

At first blush, one may conclude that the student has conducted some type of risk assessment, albeit intuitive, and has concluded that she needs to be concerned about physical fitness (biking) and the oral route of exposure to contaminants, as evidenced by the bottled water. For some reason, the student is not as concerned about the potential carcinogens in tobacco smoke as about the contaminants found in tap water. Or is it simply taste . . . or mass marketing?

With a bit more analysis, however, the apparent lack of concern for the inhalation route (tobacco smoke) may not be the case. The behavior may be demonstrating the concept of risk perception. The biking, smoking, and drinking activities seem to illustrate at least two principles regarding increased concern about risk: whether the student maintains some control over risk decisions, and whether the exposures and risks are voluntary or involuntary. The observation may also demonstrate the lack of homogeneity in risk perception. For example, risk perception appears to be age dependent. More frequently than the general population, teenagers and young adults perceive themselves to be invincible, invulnerable, and even immortal. Like most decisions, risk decisions consist of five components:

1. An inventory of relevant choices
2. An identification of potential consequences of each choice
3. An assessment of the likelihood of each consequence actually occurring
4. A determination of the importance of these consequences
5. Synthesis of this information to decide which choice is the best[7]

These perceptions change with age, as a result of experiences and of physiological changes in the brain. However, like the risks associated with the lack of experience in driving an automobile, a young person may do permanent damage while traversing these developmental phases. In fact, this mix of physiological, social, and environmental factors in decision making is an important variable in characterizing hazards. In addition, the hazard itself influences the risk perception. For example, whether the hazard is intense or diffuse, or whether it is natural or human-induced (see Figure 4.2) is a determination of public acceptance of the risks associated with the hazard. People tend to be more accepting of hazards that are natural in origin, voluntary, and concentrated in time and space.[8]

Other possible explanations are risk mitigation and sorting of competing values. The biker may well know that smoking is a risky endeavor and is attempting to mitigate that risk by other positive actions, such as exercise and clean water. Or she may simply be making a choice that the freedom to smoke

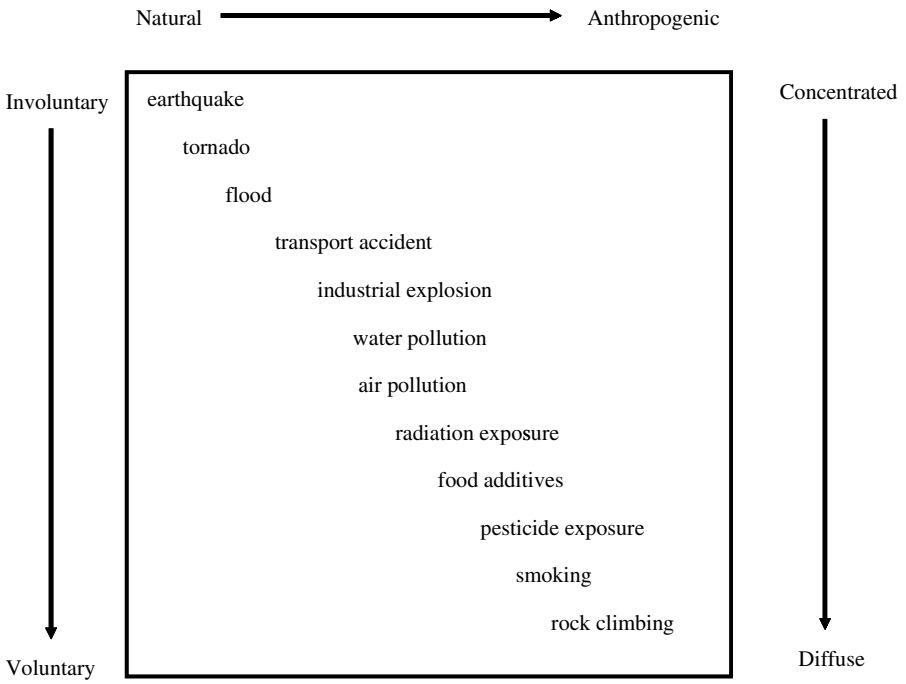


Figure 4.2 Spectrum of hazards. (From D. A. Vallero, *Paradigms Lost: Learning from Environmental Mistakes, Mishaps, and Misdeeds,* Butterworth-Heinemann, Burlington, MA, 2006; adapted from K. Smith, *Environmental Hazards: Assessing Risk and Reducing Disaster,* Routledge, London, 1992.)

outweighs other values, like a healthy lifestyle (students have reported that biking may well simply be a means of transportation and not a question of values at all).

It is likely that all of these factors affect different people in myriad ways, illustrating the complexities involved in risk management decisions.

Demographics is a determinant of risk perception, with certain groups more prone to ''risk taking'' and averse to authority. Teenagers, for example, are often shifting dependencies (e.g., from parents to peers). Later, the dependencies may be transitioning to greater independence, such as that found on college campuses. Eventually, these can lead to healthy, interdependent relationships. Engineers have to deal with these dynamics as a snapshot. Although individual persons may be changing, a population is often more static. There are exceptions, for example, if the mean age of a neighborhood is undergoing significant change (e.g., getting younger), there may a concomitant change in risk acceptance and acceptance of controls (e.g., changes to zoning and land use).

What people perceive as risks and how they prioritize those risks is only partly driven by the actual objective assessment of risk (i.e., the severity of the hazard combined with

the magnitude, duration, and frequency of the exposure to the hazard). For example, the young student may be aware that cigarette smoke contains some nasty compounds but is not directly aware of what these are (e.g., polycyclic aromatic hydrocarbons and carcinogenic metal compounds). She has probably read the conspicuous warning labels many times as she held a pack in her hands, but they really have not ''rung true'' to her. She may never have met anyone with emphysema or lung cancer and may not yet be concerned with the effects on the unborn (i.e., *in utero* exposure). Psychologists also tell us that many in this age group have a feeling of invulnerability. Those who do think about it may also conclude that they will have plenty of time to end the habit before it does any long-term damage.

People seem to have their own ''mathematics'' when it comes to risk. If you visit a local hospital, you are likely to see a number of patients gathered near the hospital entrance in designated smoking areas. Here they are hooked up to IVs, pumps, and other miracles of medical technology and engaging simultaneously in one of the most potent health hazards: smoking. Of course, this is ironic. On the one hand we are assigning the most talented (and expensive) professionals to treat what is ailing them, yet they have made a personal decision to engage in very unhealthful habits. It seems analogous to training and equipping a person at great social costs to drive a very expensive vehicle, all the time knowing that the person has a nasty habit of tailgating and weaving in traffic. Even if the person is well trained and has the best car, this habit will increase the risk. However, there is another way to look at the smoking situation, that is, the person has decided (mathematically) that the sunken costs are dictating the decision. Intuitively, the smoker has differentiated long-term risks from short-term risks. The reason the person is in the hospital (e.g., heart disease, cancer, emphysema) is the result of risk decisions the person made years, maybe decades, ago. The exposure is long in duration and the effect is chronic. So the person may reason that the effects of today's smoking will only be manifested 20 years hence and has little incentive to stop engaging in the hazardous activity. Others see the same risk and use a different type of math. They reason that there is X odds that they will live a somewhat normal life after treatment, so they need to eliminate bad habits that will put them in the same situation 20 years from now. They both have the same data but reach very different risk decisions.

Another interesting aspect of risk perception is how it varies in scale and scope. Vallero recalls sitting in a meeting in the late 1970s among a group of highly trained engineers and scientists from the U.S. EPA and the Kansas Department of Health and Environment. Mind you, these are the two principal federal and state agencies, respectively, charged with protecting the environment. The meeting was to determine the appropriate ways to reduce the ambient concentrations of pollutants, especially particulate matter and carbon monoxide (CO). The meeting was held in a small room, and almost every person was smoking except Vallero. The room was literally smoke-filled. Here we were talking about the best approaches for bringing down ambient levels to the parts per million range (high by today's standards, but at detection limits in the 1970s) in a room that one could almost report as a percent (i.e., 10^{-6} *versus* 10^{-2}). The irony was lost on the participants, probably for good reason. They were not making personal decisions, they were making policy decisions. This is akin to saying ''Do as I say, not as I do.'' Such compartmentalization is not foreign to engineers. We realize that some of our own

homes would not meet many of the standards that we require for our clients' structures (e.g., accessibility, water and air quality, egress/exit, signage).

The compartmentalization concept is brought home some years back in a story shared by a former director of the Office of Management and Budget (OMB). He had been in a budget meeting earlier in the morning, where he was discussing a few multimillion-dollar projects and recommending some million-dollar increases in a number of them. Later, his wife called to update him on the new house they were building. One item was that the window contractor needed an additional $200 above the original estimate. The OMB director was outraged and starting ranting about how important it was to hold the line on such expenses. At this point, he was struck by the irony. Again, the policy decision has a different scale and scope than the individual decision (in the OMB director's case, $\$10^8$ *versus* $\$10^2$). But is this right? Is vigilance a function of size (dollars, risk, number of people), or must it be independent of scale? The neighbors do not care that you have a bigger project or one with much more societal import. This is their only neighborhood, these are their only children, and the risks are the ones that they will have to abide.

Another consideration is whether the scope flavors what we consider to be of value. For example, many communities are overrun with deer populations because suburban developments have infringed on the habitats of deer and their predators, changing the ecological dynamics. Certainly, the deer population increases present major safety problems, especially during rutting season when deer enter roadways and the likelihood of collisions increases. The deer also present nuisances, such as their invasions of gardens. They are even part of the life cycle of disease vectors when they are hosts to ticks that transmit Rocky Mountain spotted fever and Lyme disease. In this sense we may see deer as a ''problem'' that must be eradicated. However, when we come face to face with deer, such as when we see a doe and her fawns, we can appreciate the majesty and value of the individual deer.

Recently, I (Vallero speaking) have been taking numerous actions to make my garden unpleasant to these creatures, with little success (they particularly like eating my tomatoes before they ripen and enjoy nibbling young zucchini plants). I said some rather unpleasant things about the deer population. However, recently I was traveling on a country highway and noticed an emergency vehicle assisting a driver who had obviously crashed into something. The driver and responder were in the process of leaving the scene. Driving 20 meters further, I noticed a large doe trying to come to her feet to run into the woods adjacent to the highway, but she could not lift her back legs. I realized that the people leaving the scene must have concluded that the ''emergency'' was over, without regard to the deer that had been struck by the car. Seeing the wounded creature was a reminder that the individual, suffering animal had an intrinsic value. Such a value is lost when we see only the large-scale problem, without consideration of the individual. Incidentally, by the time I had turned around, another person had already called the animal control authorities. Coming into personal contact with that deer makes it *our* deer, just as knowing that there are too many dogs and cats in the nation does not diminish our devotion to *our* own dog or cat.[9] I still may take measures to prevent the deer herd from destroying my garden, but I would gladly open my garden to *that* deer I pitied on the side of the road.

Thus, we should be aware that what we are saying to people, no matter how technically sound and convincing to us as engineers and scientists, may be simply a din to

our targeted audience, or even an affront to the values they cherish. Their value systems and the ways that they perceive risk are the result of their own unique blend of experiences.

The converse is also true. We may be completely persuaded based on data, facts, and models that something clearly does not cause significant harm, but those we are trying to convince of this finding may not buy it. They may think that we have some vested interest, or that they find us guilty by association with a group they do not trust, or that we are simply ''hired guns.'' They may not understand us because we are using jargon and are not clear in how we communicate the risks. So do not be surprised if the perception of risk does not match the risk you have quantified.

Analyzing each part of Aristotle's seemingly counterintuitive and repetitious quote sheds some light on why risk is such a crucial part of any communication with the public, particularly the risk to those that have been or appear to have been exposed to disproportionately high concentrations of contaminants in their food, air, water, or homes. When the query continues with a bit more information, the number of advocates for the inability to quantify risks begins to dwindle. For example, when the numbers of tobacco users *versus* the incidence of cancer (or other health endpoints) are shown side by side for a population, the strength of association pushes the group to accept that risk as being quantitative. In the same vein, when automobile, rail, bike, and air travel mortality statistics are provided; people see that there is some numeric link between outcomes and behaviors, but they still perceive a greater risk from air travel. People can ''do the math,'' but the math does not hold primacy over what they perceive to be risks. The weight of evidence includes some nonquantifiable factors. For example, they may simply wonder how a multiton metal object can stay airborne. In addition, of the various modes of transportation, air travel on a large plane gives the passenger the least control and fewest options if things go wrong (i.e., a perceived lack of control). So there is always the specter of ''improbable possibilities'' when managing risks.

Perception and Reality

Engineers generally would not disagree that the failure of the Tacoma Narrows Bridge or the devastation wrought by the 1906 earthquake in San Francisco were indeed *disasters.* But characterizing something as a disaster as opposed to the run-of-the-mill ''failures'' depends in large measure on how the public, or at least a substantial part of it, such as the media, perceive it. Failure occurs all the time. In fact, for all human endeavors failure is inevitable. Failure becomes a disaster when events in time and space lead to effects so severe that the events collectively are deemed to be a disaster. An event could also be classified as a disaster when engineers made such a miscalculation or left out some key information. Such mistakes may lead to the public perception that the failure was disastrous, compared to an even more severe outcome that was perceived as less preventable, or even inevitable.

For some events, we do not even recognize them as a disaster until well after the fact. Environmental justice disasters, in particular, may not be noticed for decades. This can be because the actual disease and negative effects are delayed (i.e., called the *latency period.* For example, the onset of cancer symptoms may not be diagnosed until decades

Biographical Sketch: Mary Amdur

Often the most important people in the lives of ordinary citizens are not recognized for their work. Such was the case with Mary Amdur (1922–1998).

She graduated from the University of Pittsburgh and went on to the Harvard Medical School, working with an eminent pulmonologist, Philip Drinker, the inventor of the iron lung that was used to help polio victims. While at Harvard Amdur began to do work in toxicology, especially chronic, sublethal doses of toxins. A principal motivation for her career was the premature death of her father at 40 from lung cancer linked to the steel works in Donora, Pennsylvania. She invented a pump that allowed her to expose guinea pigs to fine mists that carried the toxins into the lungs of the guinea pigs. She showed that young animals are affected sooner and more severely than older animals, and found that the sulfuric acid mist acts synergistically with fine particulates, helping to explain one of the potential ways that people might have died during the Donora air pollution episode. The two researchers also studied the animals that had died during the Donora air pollution episode, and these data confirmed their findings that age is an important factor. They went on to conduct studies with volunteer humans who were subjected to acid mist and who developed the same symptoms as the guinea pigs, clearly proving that acid mist is a dangerous air pollutant.

Unfortunately, the research conducted by Amdur and Drinker was supported financially by the American Smelting and Refining Company, which was not at all pleased with the findings. At one point company representatives allegedly attempted physical intimidation of Mary Amdur, using thugs who crowded her on an elevator and told her not to publish any more papers on the topic. Whatever pressure or intimidation was bought on Drinker seems to have worked. The actual facts are not known, but they resulted in Drinker's firing of Amdur and removal of his name from the papers that they had planned to publish jointly. One paper that at the time was ready for publication in the *Lancet,* the British medical journal, simply disappeared (this was before floppy disks and hard drive backups). Mary Amdur endured this intimidation and soon found a new job in the Harvard School of Public Health, where she stayed for 20 years, continuing to publish prolifically on the toxicology of air pollutants. Her work was well ahead of its time, and it took the world decades to catch up to her (e.g., it was not until the 1990s that the increase in mortality was linked statistically to episodes of elevated particulate levels, interestingly in large part to research by Douglas Dockery and other of Amdur's successors at Harvard[10]). Her courage and perseverance has been an inspiration to many environmental toxicologists.

after exposure to the carcinogen (e.g., asbestos workers may be exposed for decades before signs of mesothelioma or lung cancer). The lag in noticing problems may also reflect the current state of the science. For example, if a contaminant exists in soil at concentrations below those that can be detected by contemporary sampling and analytical

methods, the unsafe conditions will not be reported. This was a factor in some infamous cases, like Times Beach and Love Canal. Even if the levels of dioxin and other contaminants had existed in the 1950s, the science would not have been sufficiently advanced to detect the problem. The lack of recognition of actual and pending disasters may also be the result of understudying and underreporting of the exposures and diseases, such as the relatively recent linkages between childhood exposures to the metal lead and neurological and developmental diseases.

The two problems (i.e., latency period and underreporting) can amplify one another in environmental justice situations. For example, certain workers may not want to jeopardize their livelihoods and are reluctant to report early symptoms of chronic diseases. Scientists historically have been more likely to study certain demographic groups (e.g., healthy workers) and have avoided others (children, women, and minorities). But when the results do flood in, such as the lead studies in the latter part of the twentieth century or the ongoing arsenic exposures in Bangladesh (see Chapter 3), they are perceived to be ''public health disasters.''

So risk perception is a crucial component of risk management. The engineer must be cognizant that sharing the same set of facts will be perceived differently by different groups. One group may see the facts as representing a problem that can easily be fixed, whereas another may perceive the same facts as representing an engineering or public health disaster. A notable example is the State University of New York, Stony Brook,[11] comparison of U.S. transportation fatalities in 1992. The study found that the modes of transportation had similar numbers of fatalities from accidents involving airplanes (775), trains (755), and bicycles (722). The public, however, considered air travel to have much higher risk associated with it than the risk from trains and certainly for bicycles. The researchers concluded that two driving factors may lead to these perceptions: (1) a single event in air crashes leads to a large loss of life, with much media attention; and (2) people aboard a large aircraft have virtually no control over their situation.

The increased anxiety resulting from highly visible failures and lack of control over outcomes leads to the greater perceived risk. These factors also seem to occur for environmental and public health risks. Certain terms are downright scary, like *cancer, central nervous system dysfunction, toxics,* and ominous-sounding chemical names like *dioxin, PCBs, vinyl chloride,* and *methyl mercury.* In fact, the chemicals listed *are* ominous! But some that are less harmful can also elicit anxieties and associated increased perceived risk, even from the well educated and erudite. For example, students at Duke have been asked for some years now as part of a pretest to an engineering ethics course to answer a number of questions. The first two questions on the exam are:

1. The compound dihydrogen monoxide has several manufacturing and industrial uses. However, it has been associated with acute health effects and death in humans, as a result of displacement of oxygen from vital organs. The compound has been found to form chemical solutions and suspensions with other substances, crossing cellular membranes, and leading to cancer and other chronic diseases in humans. In addition, the compound has been associated with fish kills when supersaturated with molecular oxygen, destruction of wetlands and other habitats, and billions of dollars of material damage each year. A prudent course of action dealing with dihydrogen monoxide is to:
   a. Ban the substance outright

b. Conduct a thorough risk assessment, then take regulatory actions
c. Work with industries using the compound to find suitable substitutes
d. Restrict the uses of the substance to those of strategic importance to the United States
e. Take no action except to warn the public about the risks

2. The class of compounds, polychlorinated biphenyls, had several manufacturing and industrial uses during the twentieth century. However, PCBs were associated with acute health effects and death in humans. The compound has been found to form chemical solutions and suspensions with other substances, crossing cellular membranes, and leading to cancer and other chronic diseases in humans. In addition, the compound has been associated with contaminated sediments, as well as wetlands and other habitats, and billions of dollars of material damage each year. A prudent course of action dealing with PCBs is to:
a. Ban the substances outright
b. Conduct a thorough risk assessment, then take regulatory actions
c. Work with industries using compound to find suitable substitutes
d. Restrict the uses of the substances to those of strategic importance to the United States
e. Take no action except to warn the public about the risks

Everything in the question is factually correct. The two questions were intentionally worded similarly and the answers worded identically. The students are well-versed in math and science. On average, their scores on their Scholastic Achievement Tests are above 1400, and most have earned A's in high school or college chemistry, physics, and biology, and are on their way toward completing engineering and other technical degrees. Interestingly, the answers to the two questions differed very little. Most students appear to be influenced by the litany of negative effects to health and safety. The most frequent answer is b: conduct a risk assessment. Students seem to be heeding their teacher's relentless reminders that they get their facts straight before technical decisions (one of the themes of this book). Before we overcongratulate ourselves, as engineering educators, however, many of the students saw no difference between the two questions and several chose a: outright bans on both chemicals, the first of which is water!

Actually, the answers to the two questions should have been very different. We would recommend reply e for water and reply a for the polychlorinated biphenyls (simply because they have been banned since the 1970s following the passage of the Toxic Substances Control Act). We do note that water is not risk-free. In fact, it is a contributing factor in many deaths [drowning, electrocution, auto accidents, falls, especially water in its solid phase (i.e., ice) and workplace incidents such as steam-related accidents]. However, none of us could survive if we banned or placed major restrictions on its use!

Perception may be either higher or lower than actual risk. So, then, engineers must reconcile technical facts with pubic fears. What are the ethics of technical communication when it comes to risks? Like so many engineering concepts, timing and scenarios are crucially determinate. What may be the right manner of saying or writing something in one situation may be quite inappropriate in another. Our communication approaches will differ according to whether we need to motivate people to take action, alleviate undue fears, or simply share our findings clearly, whether they convey good news or bad.

Engineers may wish to avoid the business model in this case. Some have accused certain companies of using pubic relations and advertising tools to lower the perceived risks of their products. The companies may argue that they are simply presenting a counterbalance against unrealistic perceptions. Engineers must take care not to be manipulated by parties with vested, yet hidden interests. An emerging risk management technique is *outrage management,* described by Peter Sandman, a consultant to businesses and governmental agencies.[12] According to Sandman, the first step is to present a positive public image as a ''romantic hero,'' pointing out all the good things the company or agency provides, such as jobs, modern conveniences, and medical breakthroughs. Although these facts may be accurate, they often have little to do with the decisions at hand, such as the type of pollution controls to be installed on a specific power plant near a particular neighborhood. Ethicists refer to such tactics in their extreme as *red herrings.*[13] Another way that a public image can be enhanced is to argue that the company itself is a ''victim,'' suffering the brunt of unfair media coverage or targeted by politicians. If these do not work, some companies have confessed to being ''reformed sinners,'' who are changing their ways. One of the more interesting strategies put forth by Sandman is that companies can portray themselves as ''caged beasts.'' This approach is used to convince the public that even though in the past they have engaged in unethical pollution and unfair practices, the industry is so heavily regulated and litigated against that they are no longer able to engage in these acts. So the public is led to trust that this new project is different from the company's track record. There is obviously some truth underpinning this tactic, as regulations and court precedents have curtailed a lot of pollution. But the engineer must be careful to discern the difference between actual improvement and mere spin tactics to eliminate public outrage.

Holding paramount the health, safety, and welfare of the public gives the engineer no room for spin. On the other hand, the public does often exaggerate risks. Abating risks that are, in fact, quite low could mean unnecessarily complicated and costly measures. It may also mean choosing the less acceptable alternative (i.e., one that in the long run may be more costly and deleterious to the environment or public health). For example, in the preface to the American Council on Science and Health's recent report *America's War on "Carcinogens,"* George M. Gray, formerly the executive director of the Harvard Center for Risk Analysis and currently EPA's Assistant Administrator for Research and Development, warns:

> Public misperception of the magnitude of risks can have two important repercussions. First, people may make bad decisions for themselves and their families. If the costs of organic food, purchased to avoid the hypothetical cancer risks from pesticides, reduce total consumption of fruits and vegetables, a family will clearly be worse off if they ate the recommended amounts of conventionally grown produce. Second, people may exert pressure on government agencies to focus excessively on addressing negligible risks while placing too little effort on reducing larger risks.[14]

Gray's concerns raise the possibility that the members of the public may be wrong in their gauging of a project's risks, thus complicating the task of the engineer in presenting alternatives. In fact, the community may be choosing poorly in their assessment of *risk trade-offs.* The engineer's competency may run up against pleasing the client (both at least tacitly required in most engineering codes). The best alternative, such as siting a landfill in an unpopular location but in an ideal hydrological and environmental

setting, is not simply going to be accepted by the neighbors. Nor can the engineer by *fiat* order the acceptance. This often calls for an arduous process of compromise wherein the engineer does not sacrifice what is dictated by expertise but reasonably and appropriately incorporates the needs of the community.

As shown in Table 4.1, risk assessment relies on problem identification, data analysis, and risk characterization, including cost/benefit ratios. Risk perception depends on thought processes, including intuition, personal experiences, and personal preferences. Engineers tend to be more comfortable operating in the middle column (using risk assessment processes), whereas the general public often uses the processes in the far right column. One can liken this to a ''left-brained'' engineer trying to communicate with a ''right-brained'' audience. It can be done, as long as preconceived and conventional approaches do not get in the way.

Our recent experience in a predominantly African American lower-socioeconomic-status community (i.e., an EJ community) in North Carolina is instructive. During one of the early scoping meetings regarding an environmental assessment, the plans for the early stages of the study were discussed. The engineers and scientists were explaining the need to be scientifically objective, to provide adequate quality assurance of the measurements, and to have a sound approach for testing hypotheses and handling data. We must admit that we thought going into the meeting that the subject matter was pretty ''dry'' and expected little concern or feedback. After the initial nod of approval to begin, we expected the neighborhood interest to pique only when the quality assured and validated data would be shared. However, during the scoping meetings, members of the community expressed concern about what we would do if we ''found something.'' They wanted to know if we would begin interventions then and there. We were not prepared for these questions because we knew that the data were not truly acceptable until they had been validated and interpreted. So we recommended patience until the data met the scientists' requirements for rigor. The neighborhood representatives did not see it that way. At best, they thought we were naïve, and at worst, disingenuous. It seems that they had been ''studied'' before, with little action to follow these studies. They had been told previously some of the same things they were being told at our meeting. ''Trust us!'' We

Table 4.1 Differences between Risk Assessment and Risk Perception Processes

Analytical phase	Risk assessment processes	Risk perception processes
Identifying risk	Physical, chemical, and biological monitoring and measuring the event	Personal awareness
	Deductive reasoning	Intuition
	Statistical inference	
Estimating risk	Magnitude, frequency, and duration calculations	Personal experience
	Cost estimation and damage assessment	Intangible losses and nonmonetized valuation
	Economic costs	
Evaluating risk	Cost–benefit analysis	Personality factors
	Community policy analysis	Individual action

Source: Adapted from K. Smith, *Environmental Hazards: Assessing Risk and Reducing Disaster,* Routledge, London, 1992.

were applying rigorous scientific processes (middle column), which they had endured previously. Their concerns are explained by their experience and awareness (right-hand column). As a result, our flowcharts were changed to reflect the need to consider actions and interventions before project completion. This compromise was acceptable to all parties.

So both ''lay'' groups and our highly motivated and intelligent engineers and scientists can have difficulty in parsing perceived and real risks. The balance between risk assessment and risk perception will probably be a major challenge in many projects, especially in EJ communities. One more cautionary note: Sometimes, perception *is* reality.

To scientists and engineers at least, risk is a quantifiable concept: Risk equals the probability of some adverse outcome. Risks merely result from a straightforward function of probability and consequence.[15] The consequence can take many forms. In the medical and environmental sciences, it is called a *hazard.* Risk, then, is a function of the particular hazard and the chances of a person (or neighborhood or workplace or population) being exposed to the hazard. In the environmental business, this hazard often takes the form of toxicity, although other public health and environmental hazards abound.

Defining Risk[16]

The foregoing discussion is not to taken to mean that there is complete agreement within the scientific community on the meaning of risk. Most definitions *do* include a harmful outcome and the probability of that outcome occurring. That is, many definitions of risk include both the probability of an event *and* the consequences that could result from that event. This is a common definition in planning for catastrophic events, such as nuclear accidents or terrorist attacks. For example, Christine E. Wormuth, Senior Fellow of the International Security Program, has stated:

> In most formal discussions of risk assessment, risk is defined as the product of the probability that a certain event might occur . . . and the consequences that could result from such an event. The probability side of the equation is basically a combination of threats and vulnerabilities.[17]

There is some variation within engineering and technical circles regarding the definition of risk. Recently, Enrico Cameron and Gian Francesco Peloso[18] articulated a somewhat similar definition as that used by Wormuth, but place greater emphasis on the magnitude of the adverse consequence:

> Different events . . . can have adverse effects on human life, health, property, or the environment, and consequently constitute a risk. The concept of risk can be . . . considered as the product between the magnitude of such adverse effects, expressed numerically as the number of deaths, percentage increase in cancer cases, property value loss, and so on, and the likelihood that the event causing will occur or, alternatively, the subsequent likelihood that the consequences themselves will occur.

They later state:

> Risk r will simply be expressed as the product of magnitude m and likelihood l, so with the data coming from risk analysis n risks $r_i = m_i \cdot l_i$ can be calculated.''

Biographical Sketch: Alice Hamilton

Alice Hamilton (1869–1970) graduated from both the Fort Wayne College of Medicine in Indiana and the University of Michigan School of Medicine. Following her medical degree in 1893, she did internships in Munich and Leipzig (being allowed to sit in a class of all men as long as she did not make herself conspicuous).

Some of her early experiences were working in a settlement house in Chicago, where she started educational programs and clinics for the destitute. In 1902 she recognized the connection between waste disposal and typhoid fever and was able to initiate changes in the city health department. In her work with the poor, she noted the connection between unsafe conditions at work and the health of the workers, and in 1910, became the director of the new Occupational Disease Commission.

From there she moved in 1919 to the faculty of the Harvard Medical School, where she founded the program in occupational medicine (and was the only female member of the faculty, and was appointed only on the condition that she not join the faculty club.). She was a leading participant in two occupational controversies, the leaded gasoline debate and the health of the radium dial painters (known as the ''radium girls''). In the leaded gasoline debate, she showed how lead can accumulate in the bones and fought against industry claims that there is a natural threshold of lead in the human body. She fought unsuccessfully the introduction of lead to gasoline in the 1920s, and her work was not vindicated until the 1970s, when lead in gasoline was finally banned (ironically, more for air pollution control reasons than human health). In the radium dial painters controversy, Hamilton's epidemiological studies showed how radiation exposure to women painting glow-in-the-dark watch dials was causing a high incidence of cancer.

Hamilton is acknowledged to be the founder of occupational medicine, and during her long lifetime received many honors and awards. In addition to her work in lead and radium, she paved the way for understanding numerous other environmental contaminants, including mercury (mad hatter's disease), organic solvents, and microbes (e.g., connecting typhoid fever to sewage). In 1944 she was listed in *Men of Science,* which must have caused her to chuckle.

Within this connotation of risk, there is a choice of whether to include the probability of the event or the consequence of the event. In many environmental risk assessments, it is the consequence, such as the added cancer cases or number of deaths (mortality rates) that are included in the risk equation. Also, the shorthand in this text and numerous other environmental risk documents is to present risk as a unitless value, e.g., one in a million or 10^{-6}. However, the consequence is understood. Although the risk is stated as a unitless fraction, such as a cancer risk of 10^{-6}, that numerical expression implies that the units, in fact, are number of cancer cases (i.e., one added case per million people exposed).

The National Research Council's Committee on Risk Perception and Communication[19] has defined risk as:

> . . . the product of a measure of the size of the hazard and its probability of occurrence. Regardless of how numerical estimates are made the essence of the distinction between hazard and risk is that ''risk'' takes probability explicitly into account.

This definition ''adds the hazard and its magnitude the probability that the potential harm or undesirable consequence will be realized.''

The National Research Council's Committee on Risk Characterization[20] has defined risk as:

> A concept used to give meaning to things, forces, or circumstances that pose danger to people or to what they value. Descriptions of risk are typically stated in terms of the likelihood of harm or loss from a *hazard* and usually include: an identification of what is ''at risk'' and may be harmed or lost (e.g., health of human beings or an ecosystem, personal property, quality of life, ability to carry on an economic activity); the hazard that may occasion this loss; and a judgment about the likelihood that harm will occur.

The importance of both the type of adverse outcome and its probability of occurrence is succinctly captured by Rasmussen:[21]

> The term risk usually expresses not only the potential for an undesired consequence but also how probable it is that such a consequence will occur. . . . . A mathematical definition of risk commonly found in the literature is
>
> Risk (Consequence/unit time) = Frequency (event/unit time)
>
> $\times$ Magnitude (consequence/event)

Thus, there are numerous ways to express risk quantitatively. All have an expression of probability and either explicitly or implicitly an expression of consequence.

The difference between hazard and risk can be demonstrated by two students in an engineering ethics class. Jan has made A's in all of her engineering and elective courses, including prerequisites for the ethics course. She has taken copious notes, has completed all of her homework assignments, and participates in study groups every Thursday evening. Dean, on the other hand, has taken only one of the three prerequisite courses, receiving a D. He has completed only half of his homework assignments and does not participate in study groups. Jan and Dean share the same hazard (i.e., flunking the course). However, based on the data, we would consider their risks of flunking to be very different, Dean's being much greater. Of course, this does not mean that Dean *will* flunk, or even that Jan *will* pass. It merely indicates that the probability is more likely that Dean will fail the course than will Jan. Even an A student has the slim chance of failing the course (e.g., may experience testing anxiety, may have personal problems the week of the final), just as a failing student has a slim chance of passing the course (e.g., becomes motivated, catches up on homework, reaches a state of illumination, correctly recognizes a pattern on the answer sheet). This is why there is seldom a ''sure thing'' (i.e., 100% probability) in risk assessment. However, the risk difference between Jan and Dean can be very large: say, 0.0001 for Jan and 0.85 for Dean.

The example also illustrates the concept of risk mitigation. For example, if Dean does begin to take actions, he can decrease the probability (i.e., risk). Perhaps by partic-

ipating in a study group he decreases the risk of flunking to 50%, and by also catching up on his homework, the risk drops to 20%. These two risk abatement actions lowered his risk by 65%.

To illustrate further the difference between hazard and risk, let us consider an environmental example: a ''highly exposed person'' *versus* a person with very low exposure. Leinad works in a lead foundry, is removing lead-containing paint from his home walls, drinks from a private well with average lead concentrations of 10 mg L^{-1}, and in his spare time breaks down automobile batteries to remove the lead cores. Enraa is of the same gender and age as Leinad, but Enraa's only exposure to lead is from the public drinking water supply, which on average is 0.001 mg L^{-1}. Lead is well known to be neurotoxic: It causes damage to the central and peripheral nervous systems of mammals, including humans. The hazard in this instance is neurotoxicity. The hazard is identical for Leinad and Enraa, nervous system disorders. However, the neurotoxic risk to Leinad is orders of magnitude higher than the neurotoxic risk to Enraa.

The chemical concentration is part of the risk equation. However, the actual exposure (beyond mere ambient concentration or even dose) is influenced by activities (e.g., working, touching, drinking, and breathing in different situations). Several of Leinad's activities would be greater than the 99th exposure percentile. A good source of information about such activities is the U.S. Environmental Protection Agency's *Exposure Factors Handbook,*[22] which summarizes statistical data on the different activities and other factors related to how people are exposed to contaminants, including:

- Drinking water consumption
- Soil ingestion
- Inhalation rates
- Dermal factors, such as skin area and soil adherence factors
- Consumption of fruits and vegetables, fish, meats, dairy products, and homegrown foods
- Breast milk intake
- Human activity factors
- Consumer product use
- Residential characteristics

The handbook provides the recommended exposure values for the general population as well as for highly exposed and environmentally susceptible subpopulations. Such differences are especially crucial for environmental justice projects. Often, the default is to calculate average exposures and risks, but actual conditions may be at levels one or two standard deviations higher than measures of central tendency (mean, median, or mode), out in the tail of the distribution. After all, environmental justice communities are, by definition, exposed to contaminants disproportionately compared to the general population. Certain minority subpopulations have higher body burdens of persistent toxicants than the burdens found in the general population. For example, subsistent fishing and hunting is more common in Inuit populations in the Arctic regions of North America. Tissue concentrations of PCBs and toxic compounds in fish and top predators (e.g., polar bears) have increased dramatically in the past five decades.[23] Thus, the PCB body burden of the Inuit has also increased. Merely advising a change in activities, such as no longer

hunting or fishing, may not only be infeasible (e.g., these may substantially represent the food source), but such recommendations may militate against traditional, even religious or spiritual, mores of the people. Thus, decisions about acceptable levels of exposures to PCBs for Inuit people must take into account the already elevated levels, and risk management must incorporate numerous social factors.

Risks appear in a complex social arrangement; they do not occur in a vacuum. Taking care of one risk can, if we are lucky, ameliorate another risk, such as when pollution control equipment removes particles and in the process also removes heavy metals that are sorbed to the particles. This means that not only are risks to heart and lung diseases reduced, but neurological risks are also reduced because of the decrease in exposures to lead, mercury, and other neurotoxic metals. Conversely, reducing one risk can, if we are unlucky, increase other risks, such as when solid waste is incinerated, eliminating the possibility of long-term risks from contaminated groundwater while escalating the concentrations of products of incomplete combustion in the air as well as creating bottom ash with very high concentrations of toxic metals.

Another problem occurs when one exposed group is exchanged for another. For example, to address the concern of possible exposures of building inhabitants to asbestos in building materials, we are likely to create occupational asbestos exposures to workers called in to remove the materials. This is always a consideration in engineering management: that is, the acceptable amount to which workers in a remediation or emergency response project should be exposed. Obviously, risk abatement measures such as respirators, protective clothing, and other measures must be part of any hazardous situation. Another example of risk shifting is that of environmental justice situations, when the overall population risk is lowered by moving contaminants to sparsely populated regions, but the risk to certain groups is in fact increased. This type of risk shifting can also occur internationally, such as when a nation decides that it does not want its population or ecosystems to be exposed to a hazardous substance but still allows the manufacture and shipping of the substance outside its borders[24] (see the discussion of risk shifting in Chapter 3).

Another example of risk shifting is a decision made in one part of world's impact on another remote region. Consider *persistent organic pollutants* (POPs), most of which are organochlorine compounds. In addition to the global problem of long-range transport, these compounds present abundant lessons on how to address local problems with risk trade-offs. As mentioned, subsistence anglers and hunters receive heavy doses of these substances in their food. Pregnant and lactating women in these regions often have elevated concentrations of PCBs, dioxins, and other POPs in their fats and breast milk. What can we learn from this? First, the engineer must ensure that recommendations are based on sound science. Although seemingly obvious, this lesson is seldom easy to put into practice. Sound science can be trumped by perceived risk, such as when a chemical with an ominous-sounding name is uncovered in a community, leading the neighbors to call for its removal. However, the toxicity may belie the name. The chemical may have very low acute toxicity, has never been associated with cancer in any animal or human studies, and is not regulated by any agency. This hardly allays the neighbors' fears. The engineer's job is not done by declaring that removal of the chemical is not necessary, even though the declaration is absolutely right. The community deserves clear and understandable information before we can expect any capitulation.

Second, removal and remediation efforts are never entirely risk-free. To some extent they always represent risk shifting in time and space. A spike in exposures is possible during the early stages of removal and treatment, as the chemical may have been in a place and form that made this less available until actions were taken. Due in part to this initial exposure, the concept of *natural attenuation* has recently gained greater acceptance within the environmental community. However, the engineer should expect some resistance from the local community when they are informed that the best solution is to do little or nothing but to allow nature (i.e., indigenous microbes) to take its course (doing nothing could be interpreted as intellectual laziness!).

Third, the mathematics of benefits and costs is inexact. Finding the best engineering solution is seldom captured with a benefit/cost ratio. Opportunity costs and risks are associated with taking no action (e.g., the recent Hurricane Katrina disaster presents an opportunity to save valuable wetlands and to enhance a shoreline by *not developing and not rebuilding* major portions of the Gulf region). The costs in time and money are not the only reasons for avoiding an environmental action. Constructing a new wetland or adding sand to the shoreline could inadvertently attract tourists and other users who could end up presenting new and greater threats to the community's environment.

Health costs are also not simply a matter of benefits *versus* cost. In addition, they often require that one risk be traded for another. Stakeholders must be fully aware of the pros and cons to make informed decisions.

Discussion: Informed Consent

If you have had a medical procedure recently, you were probably asked to sign a form that says that you understand the risks associated with the procedure. The probability of each adverse outcome (harm) is delineated with a percentage or some other expression of odds. For example, your operation may have a 1 to 5% chance of fever and extended hospital stay, a 0.1% chance of some hearing loss, and a 0.0001% chance of death. In other words, epidemiologists have found that complications in the type of surgery you are about to receive results in the death of 1 in a million cases. This is not the same as *your* risk, which is a function of your own vulnerabilities and strengths. It is probably a general reflection of *all* cases. If you are 25 years of age and in good health, your individual risk of death is much lower than that of an 89-year-old cancer patient. So your stratum of the population may have a 1 in a 100 million chance of death, and the elderly, ill stratum a 1 in 500 chance. However, there is still a chance that the older person will live and you won't. In statistics, *you* don't matter.

So if such information is so easily misunderstood, why is it given to everyone (old, young, educated, illiterate, citizen status, etc.)?[25] The answer is something known as *informed consent.* Much of this goes back to aftermath of Nazi Germany's unethical treatment of prisoners of war, especially the "medical" procedures to which they were subjected. Other unethical medical treatments took place in the United States under the banner of "research," such as the withholding of treatment and dishonesty of researchers in "treating"[26]

syphilis in African Americans in Tuskegee, Mississippi (which began in 1930 and lasted 42 years), mistreatment of mentally handicapped patients, and sterilization of poor women of childbearing age. Such inhumane and inhuman practices cried for increased scrutiny and accountability of medical and other scientific research involving human subjects. One of the mandates that has become well established is that patient, client, or subject be thoroughly informed about any risks. As we see in this and the next chapter, however, risk assessment and management are quite complicated, even for those of us in the business of risk.

Certainly, people need to be informed about the risks of important decisions, and medical decisions are right near the top of most decisions we make. However, what is the sufficient amount of information needed to make such a decision? If we want to ask a third-grader to be included in a clinical trial, prudence and practical experience tells us that she will not be sufficiently prepared intellectually and morally for such a decision. We delegate such decisions to her guardian, as defined by regulation and the courts. However, who says that the guardian is sufficiently informed?

There is another problem that does not seem to get much attention. How many people are needlessly frightened away from certain necessary procedures because they see these percentages? When is professional care compromised in the name of full disclosure? Full disclosure is valuable only if the information is interpreted properly and precisely by the person making the decision.

We hope only a small number of people are so easily impressionable and susceptible. Is it possible that those seeing these odds of harm may simply give up or be so overcome with fear that the medical procedure is compromised? Of course, this is not an argument for the "ignorance is bliss" approach, but it is a warning that physicians (and engineers) are in a position of trust and have an enormous impact on their patients' (and clients') attitudes and outlooks.

One of the most difficult risk numbers for engineers to explain is that of the 100-year flood. Informed consent dictates that we tell people living in a certain part of town that the likelihood of a flood reaching their property line in one such flood is expected every 100 years. However, the people living there know good and well that they have had five floods in the past 20 years! We have listened to officials from U.S. Army Corps of Engineers, the Federal Emergency Management Administration, and various state agencies trying to explain this conundrum to local people. They may start by invoking the "geologic time" argument (i.e., yes, your five floods may have occurred over the past 20 years, but in a few million years there will be many more 20-year spans with no floods). This may be followed by a short course in statistics. The 100-year flood is a mathematical construction. It is a measure of central statistical tendencies, exactly like an arithmetic mean. Although we can sympathize with the engineer trying to explain this concept, we are not the least bit surprised when the local people are not impressed.

Informed consent spills over into all of our engineering specifications and accountability. Liners that leak, water treatment that fails, odorless landfills that stink, and pesticides that lead to previously unexpected health effects are all

fuel for the public's skepticism and discontent. We informed, hopefully with the best available data and knowledge, but subsequent events show that we missed a few things. Sometimes, these are important. Sometimes, they are the difference between success and failure. But the public may believe that their consent was breached. They may feel that it was really "misinformed consent."

A tool needed to address contravening risk is optimization, with which engineers are quite familiar. Unfortunately, the greater the number of contravening risks that are possible, the more complicated our optimization routine becomes.

The concept of risk trade-off is a very common phenomenon in everyone's life. For example, local governments enforce building codes to protect health and safety. Often, these added protections are associated with indirect, countervailing risks. For example, the costs of construction may increase safety risks via income and stock effects. The *income effect* results from pulling money away from family income to pay the higher mortgages, making it more difficult for the family to buy other items or services that would have protected them. The *stock effect* results when the cost of the home is increased and families have to wait to purchase a new residence, so they are left in substandard housing longer.[27] Such countervailing risks are common in environmental decisions, such as arguments for greater amounts of open space and green areas in communities, with the overall effect of increasing median housing costs and making housing less affordable. Arguing for major environmental standards is tantamount to arguing for increased risks from income and stock effects by imposing increased environmental controls. In fact, some people opposing higher-density housing are in effect calling for less standing housing stock. Many of these same people would sign petitions calling for more affordable housing, but in the interests of protecting the environment in their own neighborhoods, they are in fact making housing less affordable. This is but one of example of how the planner and engineer are frequently asked to optimize two or more conflicting variables in environmental justice situations.

Reliability: A Metric of Socially Responsible Engineering

Like risk, reliability is an expression of probability, but instead of conveying something bad, it expresses the likelihood of a good, or at least a *desired,* outcome. *Reliability* is the extent to which something can be trusted. A system, process, or item is reliable as long as it performs the designed function under the conditions specified during a certain time period. In most engineering applications, reliability means that what we design will not fail prematurely. Or, stated more positively, reliability is the mathematical expression of success; that is, reliability is the probability that something that is in operation at time zero (t_0) will still be operating until the designed life (time $t = t_t$). As such, it is also a measure of an engineer's social accountability. People in neighborhoods near the proposed location of a proposed facility want to know if it will work and will not fail. This is especially true for those facilities that may affect the environment, such as incinerators, treatment facilities, landfills and power plants. Similarly, when environmental cleanup is being proposed, people want to know how certain the engineers are that the cleanup will be successful.

Biographical Sketch: Herbert Needleman

The heavy metal lead (Pb) is an extremely potent neurotoxin (i.e., a substance that damages nerve cells). Like several other heavy metals, lead interferes with physiological processes because, when ionized, divalent lead (Pb^{2+}) acts in many ways like divalent calcium (Ca^{2+}). Due to its larger size and other chemical differences, however, Pb^{2+} induces biological effects that differ from those of Ca^{2+}. For example, during gestation and in early childhood, the developing brain is harmed when Pb^{2+}, competing with Ca^{2+}, induces the release of a neurotransmitter in elevated amounts and at the wrong time (e.g., during basal intervals, when a person is at rest). Thus, at high lead exposures, a person may have abnormally high amounts of brain activity (when it should be lower), and conversely, when a neural response is expected, little or no increase in brain activity is observed. This may induce chronic effects when synaptic connections in the brain are truncated during early brain development.

Lead also adversely affects the release of the transmitter glutamate, which is involved in brain activities associated with learning. The *N*-methyl-D-aspartate (NMDA) receptor seems to be selectively blocked when lead is present. Other lead effects include the activation of protein kinase C (PKC) because PKC apparently has a greater affinity for lead than for the normal physiological activator, divalent calcium. This, complicates and exacerbates the other neurotransmitter effects and harms the cell's chemical messaging (i.e., second-messenger systems), synthesis of proteins, and genetic expression.

All these neurological effects, especially in the developing brain, began to be documented in earnest by the medical community only within the last half century. Enter Herbert Needleman, for whom the pervasive effects of lead and its compounds on the health of children has been a lifelong concern. Needleman, a pediatrician at the University of Pittsburgh Medical Center, discovered that a correlation existed between the amount of lead in the teeth of infants and their intelligence at age 16, as measured by their IQ scores. His research has shown a dose–response correlation between lead dose and IQ: that is, the higher the lead content, the lower the IQ in these teenagers. In a series of follow-up studies, Needleman determined that lead poisoning had long-term implications for a child's attentiveness, behavior, and school success.

But Needleman was not the type of scientist who simply published his papers and waited for others to implement his findings. He recognized that immediate action was required while scientific assessments continued (advice we have often heard from members of the environmental justice communities[28]). Needleman initiated a campaign to remove tetraethyllead from gasoline, to phase out lead-based paints, and to reduce exposure in houses where kids can chew on the paint chips.[29] The results have been dramatic, with average blood lead levels in this country dropping an estimated 78% from 1976 to 1991.

Not surprisingly, the lead industry has been highly critical of Needleman and his research, and even has alleged scientific fraud and misconduct charges, against which Needleman has defended himself successfully. Throughout his professional life, Needleman has remained a consistent advocate for the cause of eradicating pediatric lead poisoning.

The probability of a failure per unit time is the *hazard rate,* a term familiar to environmental risk assessment, but many engineers may recognize it as a *failure density,* or $f(t)$. This is a function of the likelihood that an adverse outcome will occur, but note that it is not a function of the severity of the outcome. $f(t)$ is not affected by whether the outcome is very severe (such as pancreatic cancer and loss of an entire species) or relatively benign (muscle soreness or minor leaf damage). The likelihood that something will fail at a given time interval can be found by integrating the hazard rate over a defined time interval:

$$P\{t_1 \leq T_f \leq t_2\} = \int_{t_1}^{t_2} f(t)\, dt \tag{4.5}$$

where T_f is the time of failure. Thus, the reliability function $R(t)$ of a system at time t is the cumulative probability that the system has not failed in the time interval t_0 to t_t:

$$R(t) = P\{T_f \geq t\} = 1 - \int_0^t f(x)\, dx \tag{4.6}$$

Engineers must be humble, since everything we design *will* fail. We can improve reliability by extending the time (increasing t_t), thereby making the system more resistant to failure. For example, proper engineering design of a landfill barrier can decrease the flow of contaminated water between the contents of the landfill and the surrounding aquifer (e.g., a velocity of a few micronmeters per decade). However, the barrier does not eliminate failure completely [i.e., $R(t) = 0$]; it simply protracts the time before the failure occurs (increases T_f).[30]

Equation (4.2) illustrates built-in vulnerabilities such as unfair facility siting practices or the inclusion of inappropriate design criteria; like cultural bias, the time of failure is shortened. Like pollution, environmental injustice is a type of inefficiency. If we do not recognize these inefficiencies upfront, we will pay by premature failures (e.g., lawsuits; unhappy clients; a public that has not been well served in terms of our holding paramount their health, safety, and welfare).

Reliability engineering, a discipline within engineering, considers the expected or actual reliability of a process, system, or piece of equipment to identify the actions needed to reduce failures, and once a failure occurs, how to manage the effects expected from that failure. Thus, reliability is the mirror image of failure. Since risk is really the probability of failure (i.e., the probability that our system, process, or equipment will fail), risk and reliability are two sides of the same coin. Recall from our discussion of the five types of failure in Chapter 3 that it may come in many forms and from many sources. Injustice is a social failure. A tank leaking chemicals into groundwater is an engineering failure, as is exposure to carcinogens in the air, water, and food. A system that protects one group of people at the expense of another is a type of failure. So if we are to have

reliable engineering, we need to make sure that whatever we design, build, and operate is done with fairness. Otherwise, these systems are, by definition, unreliable.

The most common graphical representation of engineering reliability is the *bathtub curve* (Figure 4.3). The U shape indicates that failure is more likely to occur at the beginning (infant mortality) and near the end of the life of a system, process, or equipment. Actually, the curve indicates engineers' common proclivity to compartmentalize. We are tempted to believe that the process begins only after we are called on to design a solution. Indeed, failure can occur even before infancy. In fact, many problems in environmental justice occur during the planning and idea stage. A great idea may be shot down before it is born.

Injustices can gestate even before an engineer becomes involved in a project. This "miscarriage of justice" follows the physiological metaphor closely. Historically, certain groups of people have been excluded from preliminary discussions, so that if and when they do become involved, they are well beyond the "power curve" and have to play catch-up. The momentum of a project, often being pushed by project engineers, makes participation very difficult from some groups, so we can modify the bathtub distribution accordingly. Figure 4.4 shows that the rate of failure is highest during gestation. This may or may not be the case, since identifying the number of premature failures is extremely difficult to document with any degree of certainty.

Another good way to visualize reliability as it pertains to socially responsible engineering is to link potential causes to effects. *Cause-and-effect diagrams* (also known as *Ishikawa diagrams*) identify and characterize the totality of causes or events that contribute to a specified outcome event. A *fishbone diagram* (see Figure 4.5) arranges the categories of all causative factors according to their importance (i.e., their share of

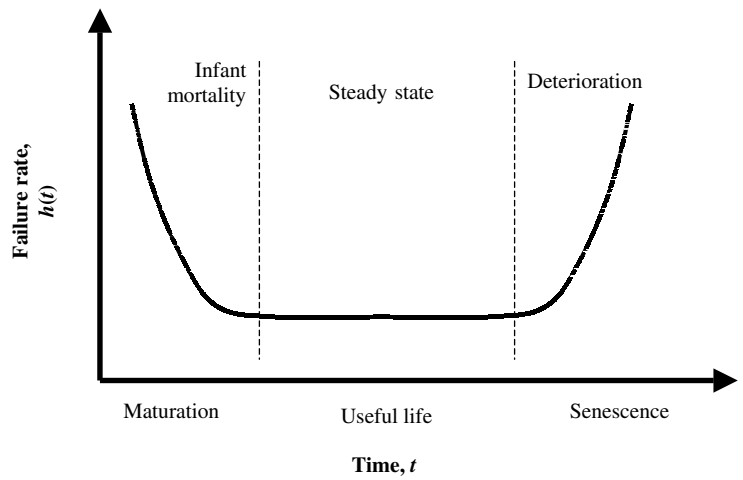


Figure 4.3 Prototypical reliability curve: the bathtub distribution. The highest rates of failure, $h(t)$, occur during the early stages of adoption (infant mortality) and when the systems, processes, or equipment become obsolete or begin to deteriorate. For well-designed systems, the steady-state period can be protracted (e.g., decades). (From D. A. Vallero, *Paradigms Lost: Learning from Environmental Mistakes, Mishaps, and Misdeeds,* Butterworth-Heinemann, Burlington, MA, 2006.)

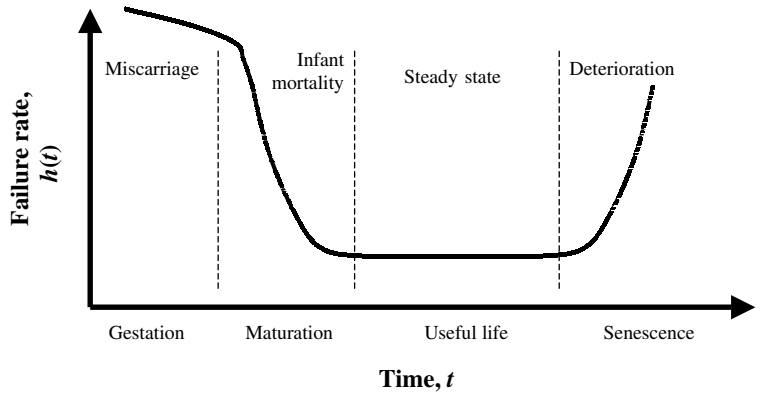


Figure 4.4 Prototypical reliability curve with a gestation (e.g., idea) stage. The highest rate of failure, $h(t)$, can occur even before the system, process, or equipment has been made a reality. Exclusion of people from decision making or failure to get input about key scientific or social variables can create a high level of hazard. (From D. A. Vallero, *Paradigms Lost: Learning from Environmental Mistakes, Mishaps, and Misdeeds,* Butterworth-Heinemann, Burlington, MA, 2006.)

the cause). The construction of this diagram begins with the failure event to the far right (i.e., the ''head'' of the fish), followed by the ''spine'' (flow of events leading to the failure). The ''bones'' are each of the contributing categories. This can be a very effective tool in explaining failures to communities, especially if the engineer constructs the diagrams with input from neighbors. Even better, the engineer may construct the diagrams in real time in a community meeting. This will help prevent recurring accidents, releases, and other failures in which contributing causes have been ignored (an all too common occurrence in environmental justice communities, whose members often lament that ''nobody listened'').

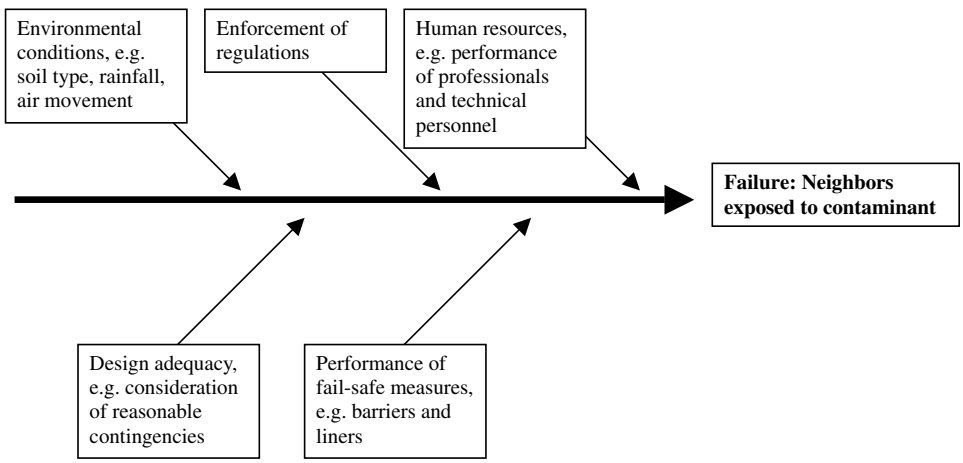


Figure 4.5 Fishbone reliability diagram, showing contributing causes to an adverse outcome (exposure to an environmental contaminant).

The premise behind cause-and-effect diagrams such as the fishbones and fault trees is that all the causes have to connect through a logic gate. This is not always the case, so another more qualitative tool may need to be used, such as the Bayesian belief network (BBN). Like the fishbone, the BBN starts with a failure (see Figure 4.6). Next, the most immediate contributing causes are linked to the failure event. The next group of factors that led to the immediate causes is then identified, followed by the remaining contributing groups. This diagram helps to catalog the contributing factors and also compares how one group of factors affects the others. Again, this can be an effective tool for gathering information about causes from neighbors as well as from government agencies, industries, and other stakeholders.

The engineering and scientific communities often use the same terms for different concepts. This is the case for reliability. Environmental engineering and other empirical sciences commonly use the term *reliability* to indicate quality, especially for data derived from measurements, including environmental and health data. In this use, reliability is defined as the degree to which measured results are dependable and consistent with respect to the study objectives (e.g., stream water quality). This specific connotation is sometimes called *test reliability,* in that it indicates the consistency of measured values over time as well as, how these values compare to other measured values, and how they differ when other tests are applied. Like engineering reliability, test reliability is a matter of trust. As such, it is often paired with test validity, that is, just how near the true value (as indicated by some type of known standard) the measured value is. The less reliable and valid the results, the less confidence scientists and engineers have in interpreting and using them. This is very important in engineering communications generally, and risk communications specifically.

The engineer must know just how reliable and valid the data are and must communicate this properly to clients and the public. This means that however discomfiting,

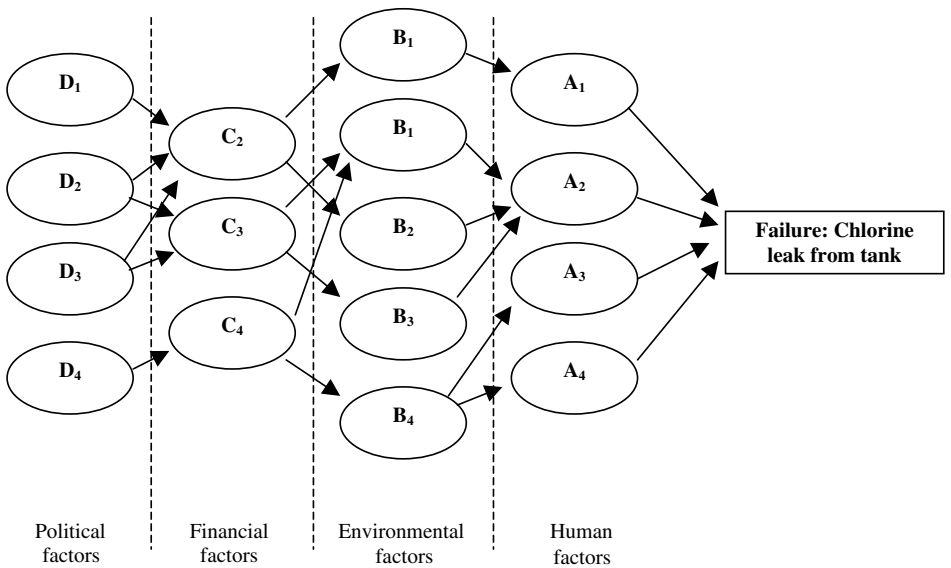


Figure 4.6 Bayesian belief network, with three groups of contributing causes leading to a failure.

the engineer must ''come clean'' about all uncertainties. Uncertainties are ubiquitous in risk assessment. The engineer should take care not to be overly optimistic or overly pessimistic about what is known and what needs to be done. Full disclosure is simply an honest rendering of what is known and what is lacking for those listening to make informed decisions. Part of the uncertainty involves conveying the meaning; we must communicate the potential risks clearly. A word or phrase can be interpreted in many ways. Engineers should liken themselves to physicians writing prescriptions. Be completely clear; otherwise, confusion may result and lead to unintended, negative consequences.

As evidenced from our discussions of disasters, risk trade-offs, and reliability, the concept of safety is laden with value judgments. Thus, ethical and just environmental decisions must rely on both sound science and quantifiable risk assessment, balanced with social fairness. In the following section we introduce a number of the elements of sound science, together with case studies to demonstrate what happens when credible risk assessment is not matched with justice.

THE ELEMENTS OF ENVIRONMENTAL RISK

Risk is a quantifiable engineering concept, and in its simplest form, risk (R) is the product of the hazard (H) and the exposure (E) to that hazard:

$$R = H \times E \tag{4.7}$$

Environmental risk assessment consists of a number of steps, described below.

Hazard Identification

A *hazard* is anything with the potential for causing harm. Ice is a slipping hazard. Sharps (e.g., syringe needles) are infection hazards. Pesticides are health hazards. A hazard is an intrinsic property of a substance, product, or process (i.e., a concept of potential harm). For example, a chemical hazard is an absolute expression of a substance's properties, since all substances have unique physical and chemical properties. These properties can render the substance to be hazardous. Conversely, equation (4.7) shows that risk can only occur with exposure. So if you are walking on a street in the summer, the likelihood of your slipping on ice is near zero. Your total slipping risk is not necessarily zero (e.g., you could step on an oily surface or someone may have thrown out ice from a freezer). If you are not in a medical facility, your infection risk from sharps may be near zero, but your total infection risk is not zero (e.g., you may be exposed to the same infection from a person sneezing in your office). If you do not use pesticides, your pesticide health risk is also lower. However, since certain pesticides are persistent and can remain in the food chain, your exposure is not zero. Also, even if your pesticide exposure is near zero, your cancer risk is not zero, since you may be exposed to other cancer hazards.

Engineers and scientists working in environmental areas consider a number of hazards; the most common is toxicity. Other important environmental hazards are shown in Table 4.2, such as landfills, storage facilities, and hazardous waste sites. Hazards can be

expressed according to the physical and chemical characteristics, as in Table 4.2, as well as in the ways they may affect living things. For example, Table 4.3 summarizes some of the expressions of biologically based criteria of hazards. Other hazards, such as flammability, are also important to environmental engineering. However, the chief hazard in most environmental justice cases has been toxicity.

The first means of determining exposure is to identify *dose,* the amount (e.g., mass) of a contaminant that comes into contact with an organism. Dose can be the amount administered to an organism (called the *applied dose*), the amount of the contaminant that enters the organism (the *internal dose*), the amount of the contaminant that is absorbed by an organism over a certain time interval (the *absorbed dose*), or the amount of the contaminants or its metabolites that reach a particular ''target'' organ (the *biologically effective dose* or simply *bioeffective dose*), such as the amount of a hepatotoxin (a chemical that harms the liver) that finds its way to liver cells or a neurotoxin (a chemical that harms the nervous system) that reaches the brain or other nervous system cells. Theoretically, the higher the concentration of a hazardous substance that comes into contact with an organism, the greater the adverse outcome expected. The pharmacological and toxicological gradient is called the *dose–response curve* (see Figure 4.7). Generally, increasing the amount of the dose means a greater incidence of the adverse outcome.

Dose–response assessment generally follows a sequence of five steps:[31]

1. Fitting the experimental dose–response data from animal and human studies with a mathematical model that fits the data reasonably well
2. Expressing the upper confidence limit (e.g., 95%) line equation for the mathematical model selected
3. Extrapolating the confidence limit line to a response point just below the lowest measured response in the experimental point (known as the *point of departure*): the beginning of the extrapolation to lower doses from actual measurements
4. Assuming that the response is a linear function of dose from the point of departure to zero response at zero dose
5. Calculating the dose on the line that is estimated to produce the response

The curves in Figure 4.7 represent those generally found for toxic chemicals.[32] Once a substance is suspected of being toxic, the extent and quantification of that hazard is assessed.[33] This step is frequently referred to as a *dose–response evaluation* because this is when researchers study the relationship between the mass or concentration (i.e., dose) and the damage caused (i.e., response). Many dose–response studies are ascertained from animal studies (*in vivo* toxicological studies), but they may also be inferred from studies of human populations (epidemiology). To some degree, ''Petri dish'' (i.e., *in vitro*) studies, such as mutagenicity studies like the Ames test[34] of bacteria, complement dose–response assessments, but they are used primarily for screening and qualitative or, at best, semiquantitative analysis of responses to substances. The actual name of the Ames test is the Ames *Salmonella*/microsome mutagenicity assay, and it shows the short-term reverse mutation in histidine-dependent *Salmonella* strains of bacteria. Its main use is to screen for a broad range of chemicals that induce genetic aberrations leading to genetic mutations. The process works by using a culture that allows only those bacteria whose genes

Table 4.2 Hazards Defined by the Resource Conservation and Recovery Act

Hazard type	Criteria	Physical/chemical classes in definition
Corrosivity	A substance with an ability to destroy tissue by chemical reactions.	Included are acids, bases, and salts of strong acids and strong bases. The waste dissolves metals and other materials, or burns the skin. Examples include rust removers, waste acid, alkaline cleaning fluids, and waste battery fluids. Corrosive wastes have a pH $<$ 2.0 or $>$ 12.5. The U.S. EPA waste code for corrosive wastes is D002.
Ignitability	A substance that oxidizes readily by burning.	This group includes any substance that combusts spontaneously at 54.3°C in air or at any temperature in water, or any strong oxidizer. Examples are paint and coating wastes, some degreasers, and other solvents. The U.S. EPA waste code for ignitable wastes is D001.
Reactivity	A substance that can react, detonate, or decompose explosively at environmental temperatures and pressures.	A reaction usually requires a strong initiator [e.g., an explosive like TNT (trinitrotoluene)], confined heat (e.g., saltpeter in gunpowder), or explosive reactions with water (e.g., Na). A reactive waste is unstable and can react rapidly or violently with water or other substances. Examples include wastes from cyanide-based plating operations, bleaches, waste oxidizers, and waste explosives. The U.S. EPA waste code for reactive wastes is D003.
Toxicity	A substance that causes harm to organisms. Acutely toxic substances elicit harm soon after exposure (e.g., highly toxic pesticides causing neurological damage within hours after exposure). Chronically toxic substances elicit harm after a long period of time of exposure (e.g., carcinogens, immunosuppressants, endocrine disruptors, and chronic neurotoxins).	Toxic chemicals include pesticides, heavy metals, and mobile or volatile compounds that migrate readily, as determined by the toxicity characteristic leaching procedure (TCLP): a TC waste. TC wastes are designated with the waste codes D004 through D043.

Table 4.3 Biologically Based Classification Criteria for Chemical Substances

Criterion	Description
Bioconcentration	The process by which living organisms concentrate a chemical contaminant to levels exceeding the surrounding environmental media (e.g., water, air, soil, or sediment).
Lethal dose (LD)	A dose of a contaminant calculated to expect a certain percentage of a population of an organism (e.g., minnow) exposed through a route other than respiration (dose units are milligrams of contaminant per kilogram of body weight). The most common metric from a bioassay is the lethal dose 50 (LD_{50}), wherein 50% of a population exposed to a contaminant is killed.
Lethal concentration (LC)	A calculated concentration of a contaminant in the air that when respired for 4 hours (i.e., exposure duration = 4 h) by a population of an organism (e.g., rat) will kill a certain percentage of that population. The most common metric from a bioassay is the lethal concentration 50 (LC_{50}), wherein 50% of a population exposed to a contaminant is killed. (Air concentration units are milligrams of contaminant per liter of air.)

Source: P. A. Vesilind, J. Peirce, and R. F. Weiner, *Environmental Engineering,* 3rd ed., Butterworth-Heinemann, Boston, 1993.

revert to histidine interdependence to form colonies. As a mutagenic chemical is added to the culture, a biological gradient can usually be determined. That is, the more chemical that is added, the greater the number and size of colonies on the plate. The test is widely used to screen for the mutagenicity of new or modified chemicals and mixtures. It is also a ''red flag'' for carcinogenicity, since cancer is a genetic disease and a manifestation of mutations.

The toxicity criteria include both acute and chronic effects, and include both human and ecosystem effects. These criteria can be quantitative. For example, a manufacturer of a new chemical may have to show that there are no toxic effects in fish exposed to concentrations below 10 mg L^{-1}. If fish show effects at 9 mg L^{-1}, the new chemical would be considered to be toxic.

A contaminant is acutely toxic if it can cause damage with only a few doses. *Chronic toxicity* occurs when a person or ecosystem is exposed to a contaminant over a protracted period of time, with repeated exposures. The essential indication of toxicity is the dose–response curve. The curves in Figure 4.7 are sigmoidal because toxicity is often concentration dependent. As the doses increase, the response cannot stay mathematically linear (e.g., the toxic effect cannot double with each doubling of the dose). So the toxic effect continues to increase but at a decreasing rate (i.e., decreasing slope). Curve A is the classic cancer dose–response; that is, any amount of exposure to a cancer-causing agent may result in an expression of cancer at the cellular level (i.e., no safe level of exposure). Thus, the curve intercepts the *x*-axis at 0.

Curve B is a classic noncancer dose–response curve. The steepness of the three curves represents the potency or severity of the toxicity. For example, curve B is steeper

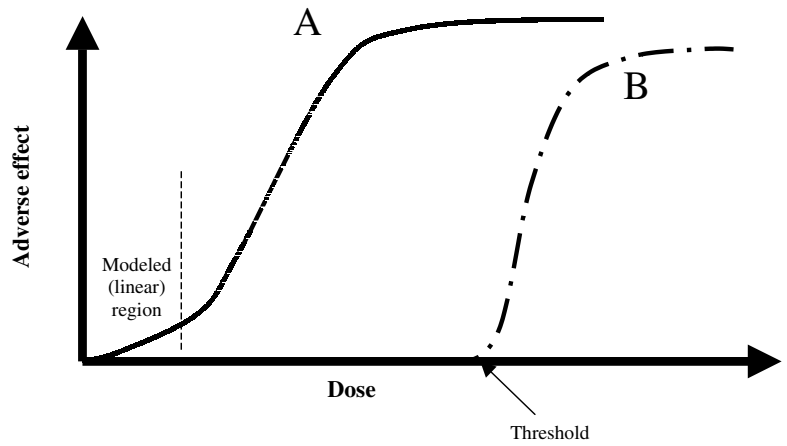


Figure 4.7 Prototypical dose–response curves. Curve A represents a no-threshold curve, which predicts a response (e.g., cancer) even if exposed to a single molecule (one-hit model). As shown, the low end of the curve, below which experimental data are available, is linear. Thus, curve A represents a linearized multistage model. Curve B represents toxicity above a certain threshold [no observable adverse effect level (NOAEL) is the level below which no response is expected]. Another threshold is the no observable effect concentration (NOEC), which is the highest concentration where no effect on survival is observed ($NOEC_{survival}$) or where no effect on growth or reproduction is observed ($NOEC_{growth}$). Note that both curves are sigmoidal in shape because of the saturation effect at high doses (i.e., less response with increasing dose). (Adapted from D. A. Vallero, *Environmental Contaminants: Assessment and Control,* Elsevier Academic Press, Burlington, MA, 2004.)

than curve A, so the adverse outcome (disease) caused by chemical in curve B is more potent than that of the chemical in curve A. Obviously, potency is only one factor in the risk. For example, a chemical may be very potent in its ability to elicit a rather innocuous effect, like a headache, and another chemical may have a rather gentle slope (lower potency) for a dreaded disease such as cancer.

With increasing potency, the range of response decreases. In other words, as shown in Figure 4.8, a severe response represented by a steep curve will be manifested in greater mortality or morbidity over a smaller range of dose. For example, an acutely toxic contaminant's dose that kills 50% of test animals (i.e., the LD_{50}) is closer to the dose that kills only 5% (LD_5) and the dose that kills 95% (LD_{95}) of the animals. The dose difference of a less acutely toxic contaminant will cover a broader range, with the differences between the LD_{50} and LD_5 and LD_{95} being more extended than that of the more acutely toxic substance.

The major differentiation of toxicity is between carcinogenic and noncancer outcomes. The term *noncancer* is commonly used to distinguish cancer outcomes (e.g., bladder cancer, leukemia, or adenocarcinoma of the lung) from other maladies, such as neurotoxicity, immune system disorders, and endocrine disruption. The policies of many regulatory agencies and international organizations treat cancer differently than noncancer effects, particularly in how the dose–response curves are drawn. As we saw in the introduction to the dose–response curves, there is no safe dose for carcinogens. Cancer dose–

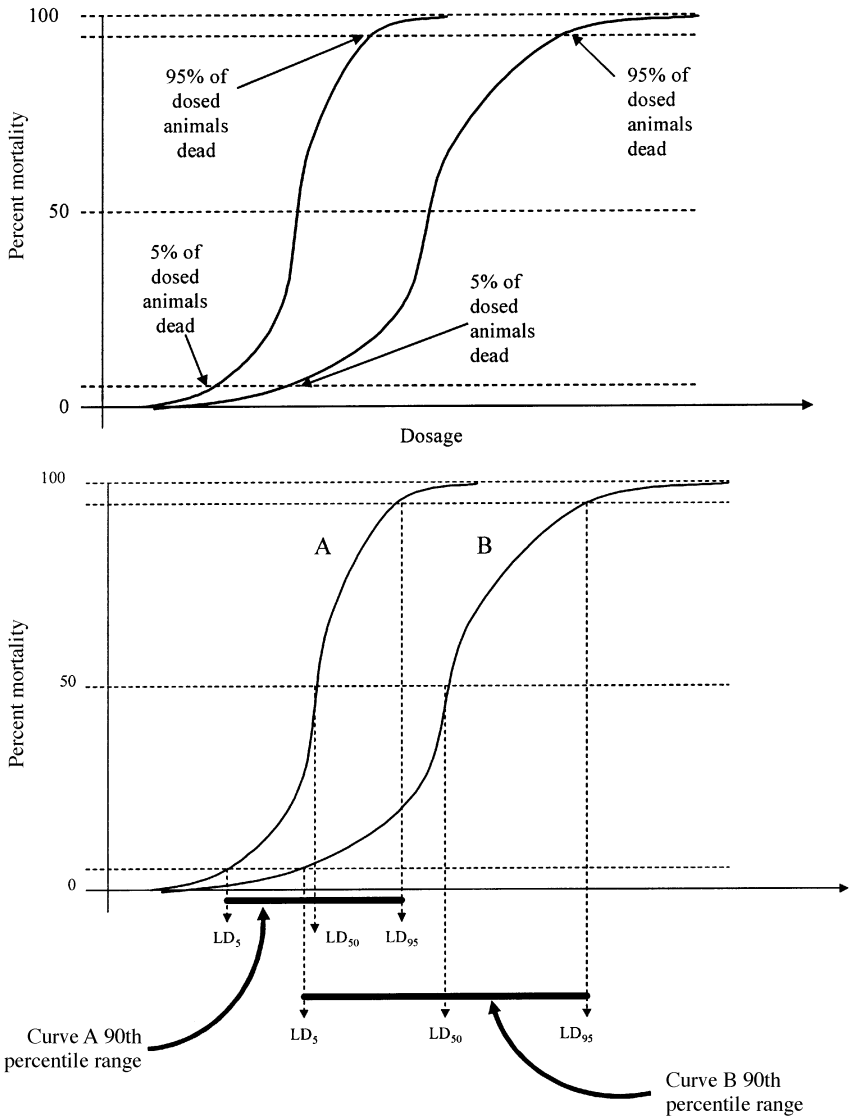


Figure 4.8 The greater the potency or severity of response (i.e., steepness of the slope) of a dose–response curve, the smaller the range of toxic response (90th percentile range shown in bottom graph). Also, note that both curves have thresholds and that curve B is less acutely toxic based on all three reported lethal doses (LD_5, LD_{50}, and LD_{95}). In fact, the LD_5 for curve A is nearly the same as the LD_{50} for curve B, meaning that about the same dose, contaminant A, kills nearly half the test animals, but contaminant B has killed only 5%. Thus, contaminant A is much more acutely toxic. (From D. A. Vallero, *Environmental Contaminants: Assessment and Control,* Elsevier Academic Press, Burlington, MA, 2004.)

response is almost always a nonthreshold curve (i.e., no safe dose is expected, whereas theoretically at least, noncancer outcomes can have a dose below which the adverse outcomes do not present themselves). So for all other diseases, safe doses of compounds can be established. These are known as *reference doses* (RfDs), usually based on the oral exposure route. If the substance is an air pollutant, the safe dose is known as the *reference concentration* (RfC), which is calculated in the same manner as the RfD, using units that apply to air (e.g., μg m^{-3}). These references are calculated from thresholds below which no adverse effect is observed in animal and human studies. If the models and data were perfect, the safe level would be the threshold, known as the *no observed adverse effect level* (NOAEL).

The term *noncancer* is very different from *anticancer* or *anticarcinogens. Anticancer procedures* include radiation and drugs that are used to attack tumor cells. *Anticarcinogens* are chemical substances that work against the processes that lead to cancer, such as antioxidants and essential substances that help the body's immune, hormonal, and other systems to prevent carcinogenesis.

In reality, the hazard identification and dose–response research is inexact and often has much uncertainty. Chief reasons for this uncertainty include variability among the animals and people being tested, as well as differences in response to the compound by different species (e.g., one species may have decreased adrenal gland activity, while another may show thyroid effects). Sometimes studies indicate only the lowest concentration of a contaminant that causes the effect—the *lowest observed adverse effect level* (LOAEL)—but the NOAEL is unknown. If the LOAEL is used, one is less certain how close this is to a safe level where no effect is expected. Often, there is temporal incongruence, such as most of the studies taking place in a shorter time frame than in real world exposures. Thus, acute or subchronic effects have to be used to estimate chronic diseases. Similarly, studies may have used different ways to administer the doses. For example, if the dose is oral but the pollutant is more likely to be inhaled by humans, this route-to-route extrapolation adds uncertainty. Finally, the data themselves may be weak because the study may lack sufficient quality, or the precision, accuracy, completeness, and representativeness of the data are unknown. These are quantified as *uncertainty factors* (UFs). *Modifying factors* (MFs) address uncertainties that are less explicit than UFs. Thus, any safe level must consider these uncertainties, so the RfD moves closer to zero; that is, the threshold is divided by these factors (usually, multiples of 10):

$$\text{RfD} = \frac{\text{NOAEL}}{\text{UF} \times \text{MF}} \tag{4.8}$$

Uncertainty can also come from error. Two errors can occur when information is interpreted in the absence of sound science. The first is the *false negative,* reporting that there is no problem when one in fact exists. The need to address this problem is often at the core of the positions taken by environmental and public health agencies and advocacy groups. They ask such questions as the following:

- What if the leak detector registers zero, but in fact, toxic substances are being released from the tank?
- What if this substance really does cause cancer but insufficient testing is the basis for this conclusion?

- What if people are being exposed to a contaminant, but via a pathway other than the ones being studied?
- What if there is a relationship that differs from the laboratory when this substance is released into the real world, such as the difference between how a chemical behaves in the human body by itself as opposed to when other chemicals are present (i.e., the problem of ''complex mixtures'')? This can either make for more toxicity (synergism) or less toxicity (antagonism).

The other concern is, conversely, the *false positive*. This can be a major challenge for public health agencies with the mandate to protect people from exposures to environmental contaminants. For example, what if previous evidence shows that an agency had listed a compound as a potential endocrine disruptor, only to find that a wealth of new information is now showing that it has no such effect? This can happen if the conclusions were based on faulty models or on models that work well only for lower organisms but subsequently developed models have taken into consideration the physical, chemical, and biological complexities of higher-level organisms, including humans. False positives may force public health officials to devote inordinate amounts of time and resources to deal with so-called *non-problems.* False positives also erroneously scare people about potentially useful products. False positives, especially when they occur frequently, create credibility gaps between engineers and scientists and the decision makers. In turn, the public, those whom we have been charged to protect, lose confidence in us as professionals.

To reduce the occurance of both false negatives and false positives, environmental risk assessment is in need of high-quality scientifically based information. Put in engineering language, the risk assessment process is a *critical path* in which any unacceptable error or uncertainty along the way will decrease the quality of the risk assessment and, quite likely, will lead to a bad environmental decision.

Reliable risk assessment begins with an understanding of the intrinsic properties of compounds, which render them more or less toxic. For example, polycyclic aromatic hydrocarbons (PAHs) are a family of large, flat compounds with repeating benzene structures. This structure makes them highly hydrophobic (i.e., fat soluble) and difficult for an organism to eliminate (since most blood and cellular fluids are mainly water). This property also enhances the PAHs' ability to insert themselves into the deoxyribonucleic acid (DNA) molecule, interfering with transcription and replication. This is why some large organic molecules can be mutagenic and carcinogenic. One of the most toxic PAHs is benzo[*a*]pyrene, which is found in cigarette smoke, combustion of coal, coke oven emissions, and numerous other processes that use combustion.

After a compound is released into the environment, its chemical structure can change substantially. Further, compounds change when taken up and metabolized by organisms. For example, methyl parathion, an insecticide used since 1954, has been associated with numerous farmworker poisonings. It has also been associated with health problems in environmental justice communities. Methyl parathion can cause rapid, fatal poisoning through skin contact, inhalation, and eating or drinking. Due to its nature, it can linger in homes for years after its application. People living in low-income housing projects are exposed disproportionately to methyl parathion. Although methyl parathion is heavily restricted, residents and landlords have been able to obtain it, since it is one of the most

effective ways to deal with cockroaches. Exposures have led to illnesses and even reports of death. In addition, the parent compound breaks down after the pesticide is applied. It may become less toxic, but it can also be transformed to more toxic metabolites, a process known as *bioactivation.* Figure 4.9 shows how methyl parathion can change in the environment, and Figure 4.10 illustrates the metabolism of methyl parathion in rodents.

Like many environmental toxicants, methyl parathion degradation involves catalysis. Organic catalysts, such as hydrolases, are known as enzymes. Note that these reactions can generate by-products that are either less toxic (i.e., detoxification) or more toxic (i.e., bioactivation) than the parent compound. For methyl parathion, the metabolic detoxification pathways are shown as 2 and 3 in Figure 4.10 and the bioactivation pathway as 1. Methyl paroxon is more toxic than methyl parathion. Note that these reactions occur within and outside an organism, so a person may be exposed to the more toxic by-product some time after the pesticide has been applied. In other words, it is possible that

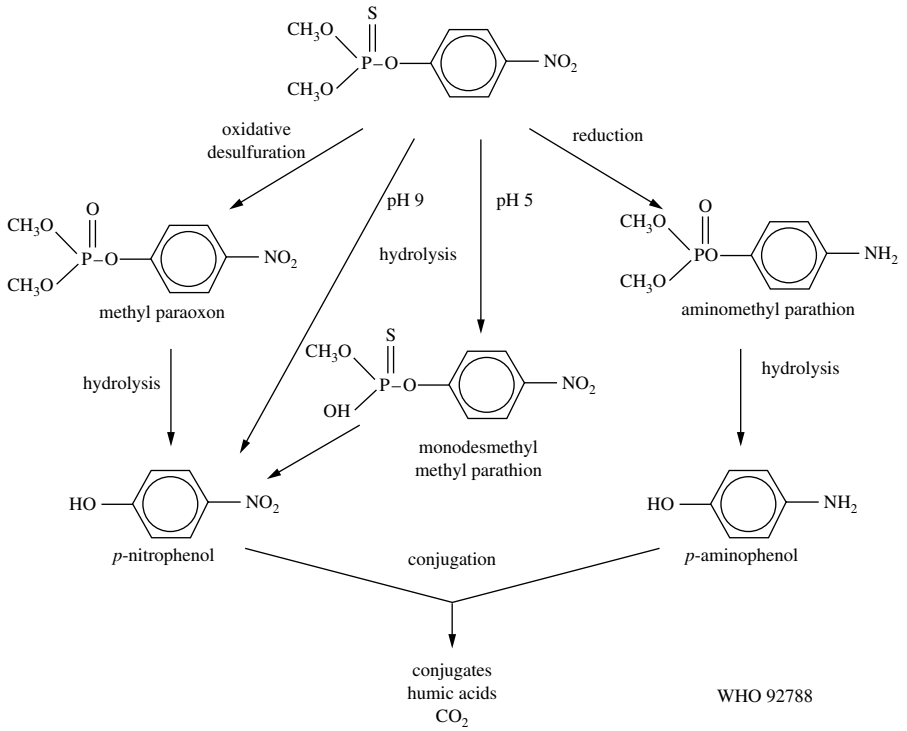


Figure 4.9 Proposed pathway of methyl parathion in water. Environmental factors, including pH, available oxygen, and water, determine the pathway. [From World Health Organization, International Programme on Chemical Safety, *Environmental Health Criteria 145: Methyl Parathion,* WHO, Geneva, 1993; A. W. Bourquin, R. L. Garnas, P. H. Pritchard, F. G. Wilkes, C. R. Cripe, and N. I. Rubinstein, Interdependent Microcosms for the Assessment of Pollutants in the Marine Environment, *International Journal of Environmental Studies,* 13(2):131–140, 1979; and R. Wilmes, Parathion-methyl: Hydrolysis Studies, Bayer AG, Institute of Metabolism Research, Leverkusen, Germany, 1987, 34 pp. (unpublished report PF 2883, submitted to WHO by Bayer AG).]

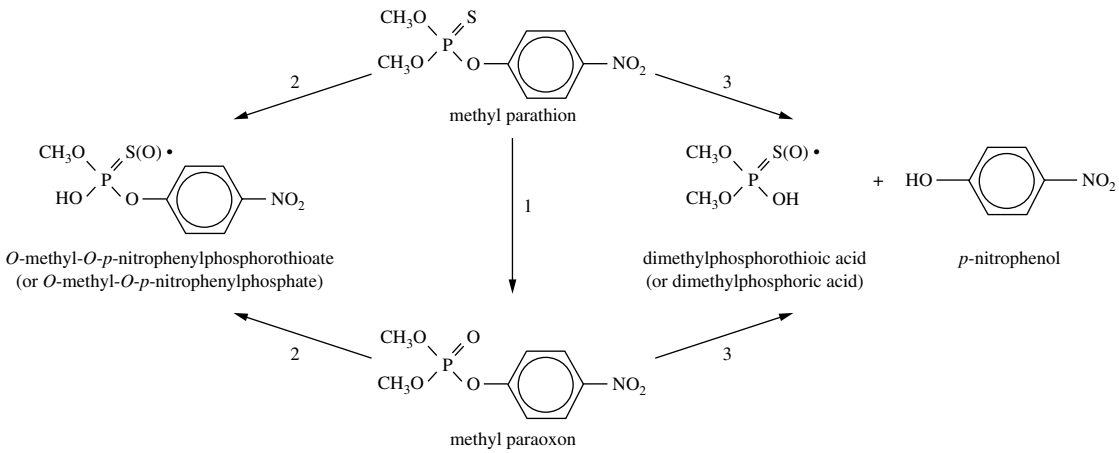


Figure 4.10 Sometimes, chemicals become more toxic as a result of an organism's (in this instance, rodents) metabolism. For example, methyl parathion's toxicity changes according to the degradation pathway. During metabolism, the biological catalysts (enzymes) make the molecule more polar by hydrolysis, oxidation, and other reactions. Bioactivation (pathway 1) renders the metabolites more toxic than the parent compound, and detoxification (pathways 2 and 3) produces less toxic metabolites. The degradation product, methyl paraoxon, may be metabolized in the same pathways as those for methyl parathion. This results in the oxygen analog, designated as (0)*. [From International Agency for Research on Cancer, Methyl Parathion, in *Miscellaneous Pesticides,* IARC, Lyon, France, 1983, pp. 131–152 (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 30).]

the risk of health effects is increased with time until the less toxic by-products (e.g., *p*-nitrophenol) replace the more toxic substances (e.g., methyl paroxon).

Figure 4.11 indicates that the exposure and toxicity properties are affected by extrinsic conditions, such as whether the substances are found in the air, water, sediment, or soil, along with the conditions of these media (e.g., oxidation-reduction, pH, and grain size). For example, the metal mercury is usually more toxic in reduced and anaerobic conditions because it is more likely to form alkylated organometallic compounds, such as monomethyl mercury and the extremely toxic dimethyl mercury. These chemically reduced mercury species are likely to form when buried under layers of sediment where dissolved oxygen levels approach zero. Ironically, engineers have unwittingly participated in increasing potential exposures to these toxic compounds. With the good intention of attempting to clean up contaminated lakes in 1970s, engineers recommended and implemented dredging programs. In the process of removing the sediment, however, the metals and other toxic chemicals that had been relatively inert and encapsulated in buried sediment were released to the lake waters. In turn, the compounds were also more likely to find their way to the atmosphere (see Figure 4.11). This is a lesson for engineers to take care in considering as many physical, chemical, and biological characteristics of the compound as possible, as well as the environment where it exists.

Some of these important physical and chemical characteristics that are part of hazard identification are listed in the Appendix. For the most toxic substances, the principal components of a hazard are its persistence, its ability to accumulate in organisms, and its ability to elicit a biological response. Let us consider each of these components briefly.[35]

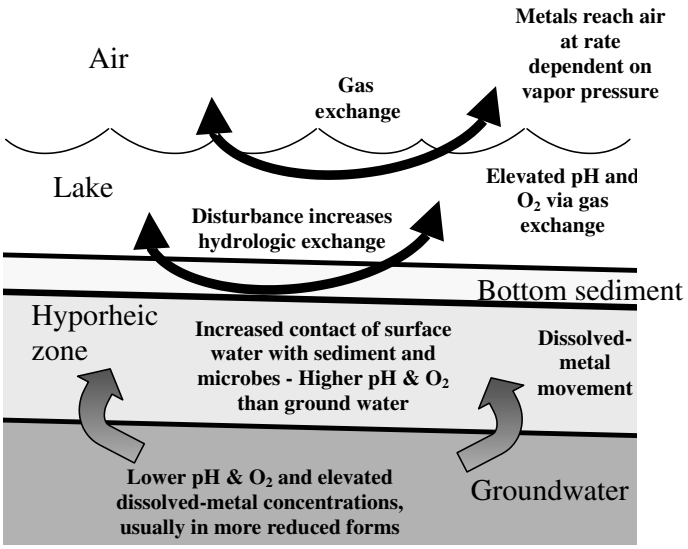


Figure 4.11 Exchanges and reactions involve all environmental media, including groundwater, sediment, and surface water. Human activities such as dredging can increase the exchanges between media and enhance the mobility of dissolved metallic compounds. (Adapted from U.S. Geological Survey and D. A. Vallero, *Environmental Contaminants: Assessment and Control,* Elsevier, Academic Press, Burlington, MA, 2004.)

Persistence

The likelihood that a substance will cause a problem is influenced by how long it lasts, so persistence is important in both parts of the risk equation (i.e., hazard and exposure). Substances that once released remain in the environment are more likely to continue to cause problems or to be a threat. Persistence is commonly expressed as the chemical half-life ($T_{1/2}$) of a substance (i.e., the time it takes to degrade one-half of the mass). The U.S. Environmental Protection Agency considers a compound to be persistent if its $T_{1/2}$ value in water, soil, or sediment is greater than 60 days, and very persistent if the $T_{1/2}$ value is greater than 180 days. In air, the compound is considered persistent if its $T_{1/2}$ value is greater than two days. Some of the most notoriously toxic chemicals are also very persistent.

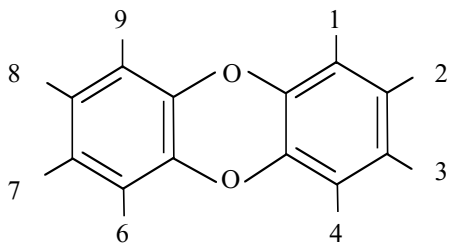
CASE STUDY: TIMES BEACH

Pick up an organic chemistry textbook published before 1975 and you might find that it estimates the half-life of dioxin to be a few months. In other words, if 20 milligrams (mg) of dioxin is released, in a few months you will have only 10 mg. Unfortunately, these estimates were based on best-case conditions in a laboratory, such as a thin film exposed to light over a large surface area. When organic compounds such as dioxin reach the environment, such rosy

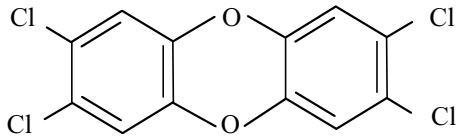
scenarios are the exception, not the rule. For example, PCBs, dioxins, and other aromatic compounds have a strong affinity for soil, especially the clay-sand organic matter. This means that instead of a few months, the half-lives can be increased to hundreds of years. When dioxins were found at Love Canal in New York and shortly after at numerous other sites, chemists and environmental scientists were thrust into hyperdrive in an effort to understand these substances. As is often the case, outrage and frustration were the norm for neighbors where the compound was discovered in soil. The experiences at Love Canal, Times Beach, Missouri and the Valley of the Drums in Kentucky were key events that led to the passage of the Comprehensive Environmental Response, Compensation and Liability Act, better known as the Superfund, in 1980.

Times Beach, about 17 miles west of St. Louis, was an unlikely spot for a hazardous waste controversy. Up to the 1970s, it was a popular resort community along the Meramec River. With few municipal resources, the roads in the town were not paved and dust on the roads was controlled by spraying oil. For two years, 1972 and 1973, the contract for the road spraying went to a waste oil hauler named Russell Bliss. The roads were paved in 1973 and the spraying ceased.

Bliss obtained his waste oil from the Northeastern Pharmaceutical and Chemical Company in Verona, Missouri, which manufactured hexachlorophene, a bactericidal chemical. In the production of hexachlorophene, the company had to remove and dispose of considerable quantities of dioxin-laden waste. A significant amount of the dioxin was contained in the "still bottoms" of chemical reactors, and the company found that having it burned in a chemical incinerator was expensive. The company was taken over by Syntex Agribusiness in 1972, and the new company decided to contract with Russell Bliss to haul away the still bottom waste without telling Bliss what was in the oily substance. Bliss mixed it with other waste oils, and this is what he used to oil the roads in Times Beach, unaware that the oil contained high concentration of dioxin (greater than 2000 ppm), including the most toxic congener, 2,3,7,8-dibenzo-*p*-dioxin (TCDD).



dioxin structure



2,3,7,8-dibenzo-*p*-dioxin (TCDD)

Bliss also sprayed oil to control dust, especially in horse arenas. He used the dioxin-laden oil to spray the roads and horse runs in nearby farms. In fact, it was the death of horses at these farms that first alerted the Centers for Disease Control to sample the soil at the farms. They found the dioxin but did not make the connection with Bliss. Finally, in 1979, the U.S. EPA became aware of the problem when a former employee of the company told them about the sloppy practices in handling the dioxin-laden waste. The EPA converged on Times Beach in "moon suits" and panic set in among the populace. The situation was not helped by the message from the EPA to the residents of the town. "If you are in town it is advisable for you to leave, and if you are out of town do not go back." In February 1983, on the basis of an advisory from the Centers for Disease Control, the EPA permanently relocated all of the residents and businesses at a cost of $33 million. Times Beach was by no means the only problem stemming from the contaminated waste oil. Twenty-seven other sites in Missouri were also identified by the EPA as being contaminated with dioxins.

The concern with dioxin, however, may have been overstated. As a previous accident in Seveso, Italy, had shown, dioxin is not nearly as acutely toxic to humans as originally feared, causing some to conclude that is unlikely that the damage to human health in Times Beach was anywhere near the catastrophe originally anticipated. Even some EPA officials later admitted that the evacuation and bulldozing of the community was probably unnecessary. But given the knowledge of dioxin toxicity in 1979, the decision to detoxify the site was not unreasonable. For one thing, the carcinogenicity of TCDD was later better established and found to be very high (slope factors $> 10^5$ for inhalation, ingestion, and dermal routes). The psychological toll of such decisions is more difficult to measure. Some years after the decision, a former resident was said to have committed suicide because, according to his wife, he was unable to cope with losing his home. Later, a local official who was party to issuing the advisory admitted the uncertainties of the social costs and that the health danger was possible, but not certain.

After everyone had moved out of Times Beach, the houses were razed and Syntex Corporation was required to build an incinerator for burning the contaminated soil. The Superfund site was eventually decontaminated at a cost of over $200 million, and the site now is a beautiful riverside park.

The concept of persistence elucidates the notion of trade-offs that are frequently needed as part of many responses to environmental insults. It also underlines the importance that good science is necessary but never sufficient to provide an acceptable response to environmental justice issues. Let us consider the pesticide DDT [1,1,1-trichloro-2,2-bis(4-chlorophenyl)ethane ($C_{14}H_9Cl_5$)]. DDT is relatively insoluble in water (1.2 to 5.5 mg L^{-1} at 25°C) and is not very volatile (vapor pressure: 2.0×10^{-7} mmHg at 25°C).[36] Looking at the water solubility and vapor pressures alone may lead one to believe that people and wildlife are not likely to be exposed in the air or water. However, the compound is highly persistent in soils, with a $T_{1/2}$ value of about 1.1 to 3.4 years, so it may still end up in drinking water in the form of suspended particles or in the air sorbed to fine particles. DDT also exhibits high bioconcentration factors (on the order of 50,000 for fish and 500,000 for bivalves), so once organisms are exposed, they tend to increase body burdens of DDT over their lifetimes. In the environment, the parent DDT is metabolized mainly to DDD and DDE.[37]

The physicochemical properties of a substance determine how readily it will move among the environmental compartments (i.e., to and from sediment, surface water, soil, groundwater, air, and in the food web, including humans). So if a substance is likely to leave the water, it is not persistent in water. However, if the compound moves from the water to the sediment, where it persists for long periods of time, it must be considered environmentally persistent. This is an example of how terminology can differ between chemists and engineers. Chemists often define persistence as an intrinsic chemical property of a compound, while engineers see it as both intrinsic and extrinsic (i.e., a function of the substrate, energy and mass balances, and equilibria). So engineers usually want to know not only about the molecular weight, functional groups, and ionic form of the compound, but also whether it is found in the air or water, and the condition of the substrate in the media (e.g., pH, soil moisture, sorption potential, organic matter content and microbial populations). The movement among phases and environmental compartments is known as *partitioning*. Many toxic compounds are semivolatile (i.e., at 20°C and 101 kPa atmospheric pressure, vapor pressures = 10^{-5} to 10^{-2} kPa) under typical environmental conditions. The low vapor pressures and low aqueous solubilities mean that they will have will low fugacities; that is, they lack a strong propensity to flee a compartment, (e.g., to move from the water to the air). The most common water-to-air fugacity measure is the *Henry's law constant*. Henry's law states that the concentration of a dissolved gas is directly proportional to the partial pressure of that gas above the solution:

$$p_a = K_H[c] \tag{4.9}$$

where K_H is the Henry's law constant, p_a is the partial pressure of the gas, and $[c]$ is the molar concentration of the gas; or

$$p_a = K_H C_W \tag{4.10}$$

where C_W is the concentration of gas in water.

Henry's law expresses the proportionality between the concentration of a dissolved contaminant and its partial pressure in the open atmosphere at equilibrium. That is, the Henry's law constant is an example of an *equilibrium constant,* which is the ratio of concentrations when chemical equilibrium is reached in a reversible reaction, the time

when the rate of the forward reaction is the same as the rate of the reverse reaction. Most of the time, when a partitioning coefficient is given, it is assumed to be an equilibrium constant. For environmental partitioning, the amount of chemical needed to reach equilibrium is usually very small (i.e., very dilute solutions and other mixtures).

A direct partitioning between the air and water phases is the air–water partitioning coefficient (K_{AW}):

$$K_{AW} = \frac{C_A}{C_W} \tag{4.11}$$

where C_A is the concentration of a gas in the air. The relationship between the air–water partition coefficient and Henry's law constant for a substance is

$$K_{AW} = \frac{K_H}{RT} \tag{4.12}$$

where R is the gas constant (8.21×10^{-2} L · atm mol^{-1} K^{-1}) and T is the temperature (K).

Under environmental conditions, most toxic substances have very low K_H values since K_H is proportional to the concentration of a dissolved contaminant and its partial pressure in the atmosphere at equilibrium. There are many exceptions, however; such as the relatively water soluble compounds with high vapor pressures (e.g., alcohols, benzene, toluene, and many organic solvents). Since Henry's law is a function of aqueous solubility and vapor pressure, estimating the tendency for a substance's release in vapor form, K_H is a good indicator of the fugacity from the water to the atmosphere.

Another common expression of partitioning is the octanol–water coefficient (K_{ow}). The K_{ow} value indicates a compound's likelihood to exist in the organic *versus* aqueous phase. A rule to keep in mind is that ''like dissolves like.'' The configuration of a molecule determines whether it is polar or nonpolar. *Polar compounds* are electrically positive at one end and negative at the other. The water molecule, for example, is highly electropositive at the hydrogen atoms and electronegative at the oxygen atom. Other molecules, like fats, are not polar (i.e., do not have strong differences between the positive and negative ends). If a relatively *nonpolar compound* is dissolved in water and the water comes into contact with another substance (e.g., octanol), the nonpolar compound will move from the water to the octanol. Its K_{ow} reflects just how much of the substance will move from the aqueous and organic solvents (phases) until it reaches equilibrium. For example, if at a given temperature and pressure a chemical is at equilibrium when its concentration in octanol is 100 mg L^{-1} and in water is 1000 mg L^{-1}, its K_{ow} is 100 divided by 1000, or 0.1. Since the range is so large among various environmental contaminants, it is common practice to express log K_{ow} values. So, for example, in a spill of equal amounts of two insecticides, DDT (log $K_{ow} \approx 7$) and methyl parathion (log $K_{ow} \approx 3$), the DDT has much greater affinity for the organic phases than does the methyl parathion (four orders of magnitude). This does not mean than a greater amount of either of the compounds is likely to stay in the water column, since they are both hydrophobic, but it does mean that they will vary in the time and mass of each contaminant moving between phases. The time it takes to reach equilibrium (i.e., the kinetics) is different (see Figure 4.12).

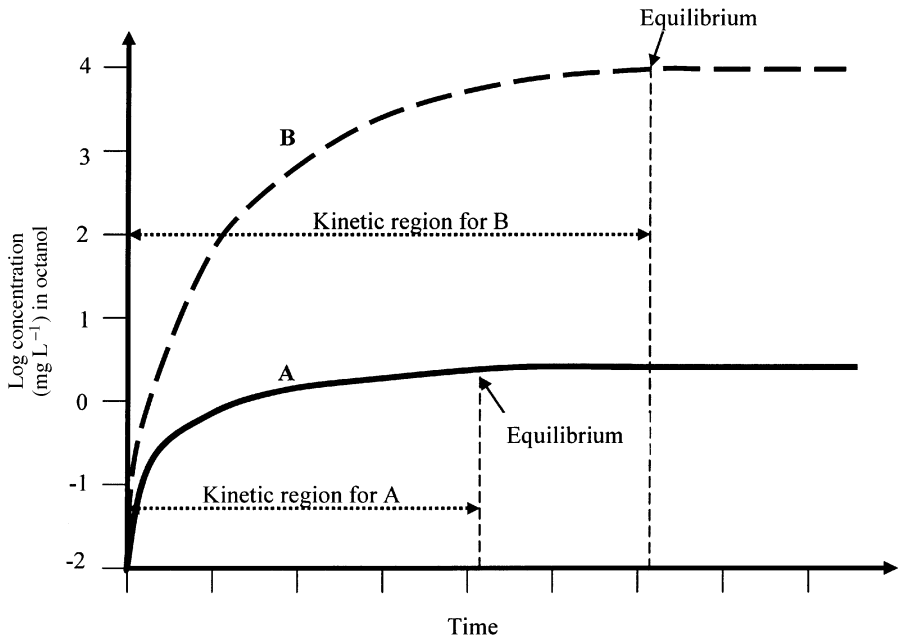


Figure 4.12 Hypothetical diagram of the relative concentrations of two compounds in octanol with time. In this instance, compound A is more soluble than compound B in octanol. Note the steeper slope for the compound A kinetics compared to the kinetics for compound B. This is an indication that the compound A's affinity for the organic phase is greater than that of compound B. The time units are not shown since these can vary considerably depending on the environmental conditions.

Even low K_H and K_{AW} compounds, however, can be transported long distances in the atmosphere when sorbed to particles. Fine particles can behave as colloids and stay suspended for extended periods of time, explaining in part why low-K_H compounds can be found in the most remote locations relative to their sources, such as in the Arctic regions. This is important, for example, when explaining to indigenous populations why they may be exposed to contaminants that are not produced near them.

Sorption is the partitioning of a substance from the liquid to the solid phase and is an important predictor of a chemical's persistence. If the substrate has sufficient sorption sites, such as many clays and organic matter, the substance may become tightly bound and persistent. The properties of the compound and those of the water, soil, and sediment determine the rate of sorption. The soil partition coefficient (K_d) is the experimentally derived ratio of a contaminant's concentration in the solid matrix to the contaminant concentration in the liquid phase at chemical equilibrium. Another frequently reported liquid-to-solid phase partitioning coefficient is the organic carbon partitioning coefficient (K_{oc}), which is the ratio of the contaminant concentration sorbed to organic matter in the matrix (soil or sediment) to the contaminant concentration in the aqueous phase. Thus, K_{oc} is derived from the quotient of a contaminant's K_d value and the fraction of organic matter (OM) in the matrix:

$$K_{oc} = \frac{K_d}{OM} \tag{4.13}$$

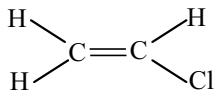
Many toxic substances are expected to be strongly sorbed, but K_{oc} varies from substrate to substrate.

It is important to keep in mind the difference between chemical persistence and environmental persistence. For example, one can look at Henry's law, solubility, vapor pressure, and sorption coefficients for a compound and determine that the compound is not persistent. However, in real-life scenarios, this may not be the case. For example, there may be a repository of a source of a nonpersistent compound that leads to a continuous, persistent exposure of a neighborhood population. Or a compound that is ordinarily not very persistent may become persistent under the right circumstances (e.g., a reactive pesticide that is tracked into a home and becomes entrapped in carpet fibers). The lower rate of photolysis (degradation by light energy) indoors and the sorptive characteristics of the carpet twill can lead to dramatically increased environmental half-lives of certain substances.

CASE STUDY: FROM CANCER ALLEY TO TOXIC GUMBO[38]

Even before the devastation that followed Hurricane Katrina, the lower Mississippi River industrial corridor was a disaster waiting to happen. It is home to a predominantly low-income minority (African American and Hispanic American) community who are being exposed to many pollutants.[39] This 80-mile-long region, known as Cancer Alley, between Baton Rouge and New Orleans has experienced releases of carcinogens, mutagens, teratogens (birth-defect agents), and endocrine disruptors in the atmosphere, soil, groundwater, and surface water. More than 100 oil refineries and petrochemical facilities are located in this region. It has been reported that per capita release of toxic air pollutants is about 27 kilograms (kg), nine times greater than the U.S. average of only 3 kg.[40] The U.S. average 260 kg of toxic air pollutants per square mile is dwarfed by the more than 7700 kg per square mile in the industrial corridor.

One particular carcinogen of concern is vinyl chloride. In the 1970s, cases of liver cancer (hepatic angiosarcoma) began to be reported in workers at polymer production facilities and other industries where vinyl chloride was present. Since then, the compound has been designated as a potent human carcinogen (inhalation slope factor = 0.3 kg · day mg^{-1}).



vinyl chloride

Vinyl chloride may at first glance appear to be broken down readily by numerous natural processes, including abiotic chemical and microbial degradation (see Figure 4.13), but numerous studies have shown that vinyl chlo-

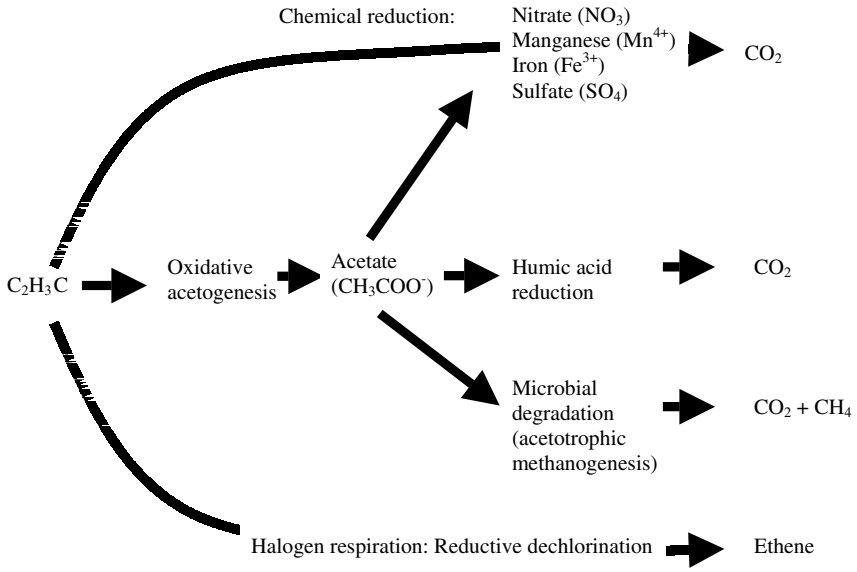


Figure 4.13 Biodegradation pathways for vinyl chloride. (From U.S. Geological Survey, Microbial Degradation of Chloroethenes in Ground Water Systems, Toxic Substances Hydrology Program: Investigations, http://toxics.usgs.gov/sites/solvents/chloroethene.html, accessed November 29, 2004.)

ride concentrations can remain elevated over long periods of time. In fact, under environmental conditions, vinyl chloride can be extremely persistent, with an anaerobic $T_{1/2}$ value in soil greater than two years. It can also be difficult to treat with conventional engineering methods. For example, aerobic degradation in sewage treatment plants and surface water in an isolated bacteria culture with vinyl chloride concentrations of 20 to 120 mg L^{-1} needs a minimum of 35 days to degrade the compound completely. Nontraditional treatment methods, such as attack by hydroxyl radicals, can significantly reduce the half-life.[41] In heavily polluted areas such as Cancer Alley, vinyl chloride repositories can remain intact for decades, serving as a continuous potential source. These repositories can actually be compounds other than vinyl chloride, but which break down to form the compound (e.g., chloroethylene solvents degrade to vinyl chloride). With its high vapor pressure (2300 mmHg at 20°C) and high aqueous solubility (1100 mg L^{-1}), the chances of people being exposed via the air or drinking water once vinyl chloride is formed can be considerable.

Local environmental and neighborhood groups have begun arming themselves with environmental data, such as the emissions and other release information in the Toxic Release Inventory (TRI) that show the inordinately high toxic chemical release rates near their communities. Local communities have challenged nearby industries with possible health effects linked to

chemical exposures. For example, residents in Mossville, Louisiana, argue that several health problems in their community could be linked to chemical releases by 17 industrial facilities located within 1 kilometer of the community. These confrontations led to a number of advocates writing the 2000 report, *Breathing Poison: The Toxic Costs of Industries in Calcasieu Parish, Louisiana,* which called for "pollution reduction, environmental health services, and a fair and just relocation for consenting residents."[42] These efforts have gained the attention of national media and regulatory agencies and have been emblematic of the environmental justice movement.

Hurricane Katrina: The Perfect Storm and the Toxic Gumbo

In many ways, the category 4 hurricane that made landfall along the Gulf coast of Louisiana, Mississippi, and Alabama on August 29, 2005 was literally and metaphorically the *perfect storm.* That is, all of the conditions for a disaster occurred simultaneously. Even worse, engineering failures of every type preceded, co-occurred, and followed the storm. The vulnerability of New Orleans was no surprise to many scientists and engineers. For example, in *Geological Hazards* (Greenwood Press, Westwood, CT), earth scientist Timothy Kusky warned that "if a category 4 hurricane ever hits New Orleans directly, the dikes will be breached and destroyed, and thousands will perish." In fact, the cities on the delta of the Mississippi River (or any river system for that matter) are engaged in a constant struggle against nature.

The perfect storm required the confluence of meteorological events (high-intensity hurricane, 100-year flood), sociological conditions (traditionally poor and mistrusting populace), and political mistakes (botched evacuation planning, delayed response due to red tape and bureaucracy, corruption, and lack of coordination among responding agencies). In addition to the loss of life and property, the immediate- and long-term environmental effects have slowly begun to be fully understood.

Possibly the best characterization was by a nonscientist, New Orleans native singer Aaron Neville. In an appearance on the *Tonight Show* a week after the storm, Neville described the scenario as a *toxic gumbo.* The floodwaters contain the typical pathogens and vectors (e.g., rats and insects) following flooding. In addition, due to the industries and commercial enterprises in the region, numerous petroleum refineries and pipelines, pesticide manufacturers, chemical plants, and other sources of toxic pollutants were added to the exposures. Again, this is no surprise, since the Gulf has some of the highest exposures to toxic contaminants in the nation, (as we have just discussed with regard to vinyl chloride). Further exacerbating the contamination is the amount of time it took to pump out the water. The longer the water remained in the New Orleans basin, the greater the number of pipe breaks, line failures, and chemical releases. Fires started and gases were released in the days following the hurricane, so that thousands of first responders from around the nation had to be called in to suppress fires and repair lines; all this while trying to evacuate people and begin recovery.

We are reminded of the old adage, "When you are up to your neck in alligators, it is too late to think about draining the swamp." Conversely in this case, we would have to restate the adage as: "When you are up to your neck in toxic gumbo, it is too late to think about saving the wetlands." Wetlands are a natural part of hydrologic systems, especially in deltas and backwater areas. Humans had to make a cognizant (using the word advisedly) decision to destroy these natural systems and replace them with structures such as buildings and roads. This is doubly bad for the hydrology since the systems now have vulnerable land uses (and thus, people using the land), and the ability to infiltrate and remove water is exponentially decreased (due to the great increase in impervious surfaces, the loss of plant life, and the elimination of the integration of surface water and groundwater systems). Before the development, the streams were connected to each other and to the aquifers, allowing for efficient water removal. So the engineers and planners who allowed and even designed these new land uses were key players in the disaster.

Another aspect of the perfect storm was poor city and regional planning. In fact, the New Orleans disaster shares some common elements with one of the worst industrial disasters in Bhopal, India. First, engineers and planners seem to adopt a "one size fits all" mentality. Like Bhopal, where a U.S. company transplanted a Western type of pesticide plant in a completely different culture, with little regard for the social differences, the design and siting of industrial facilities in the Mississippi Delta were little different from that in an upland. Second, land-use planning failed in not considering the possible impact on people living next to a facility. The discontinuity should have been obvious. In Bhopal, squatters lived right at the company property line. In New Orleans, entire neighborhoods were in the shadow of heavy industry. Third, the adjacent residents had little voice in decisions that directly affect their health. This would have been the case even without Katrina, although in a less acute manner, since the residents were being exposed to contaminants every day. Fourth, the agencies that were supposed to be protecting public health and the environment were actually collaborating with those who were presenting the threat. In Bhopal, the local and national Indian governmental agencies were more concerned about encouraging industry than about protecting the most vulnerable. Unfortunately, it appears that this may also have been the case in New Orleans.

The major similarity between the aftermaths of Bhopal and Katrina is the disproportionate effect on those who were already at a cultural and social disadvantage. One interesting observation is the disproportionate impact on women and children in New Orleans (some preliminary estimates were that four times more women than men were among the "refugees" and those who lost their lives). This may well be the result of socioeconomic conditions, such as the unstable conditions of lower-socioeconomic-status families, including higher percentages of single heads of households. Characterizing such vulnerabilities *in any community* must be part of emergency and contingency planning.

So then, what should engineers do now? We might be tempted to respond by what we do best, that is, design and build. That is only the right thing to do if we think first. Designing and building must be the outgrowth of good planning and a thoughtful consideration of the events that led to the disaster. For example, "hardening" the levees and dams is not the solution, at least it is not the entire solution. Hindsight does approach 20/20. It seems obvious now that those engineers and planners who called for stronger levees deserve credit, but this is but a small part of the solution. Such engineering projects are successful only when they are integrated into an overall plan. There are positive signs in this regard. Numerous engineering experts have called for the construction of wetlands; and many are calling for aggressive and well-enforced land-use controls.

The United States has a checkered past when it comes to planning. Some have feared that land-use planning is too much like a Soviet-style centralized planning program. We have often foregone strong land-use controls, even in vulnerable settings like wetlands, coast lines, and sensitive habitat, in the interest of unfettered uses by landowners. This is understandable, and some would argue that it is guaranteed by the Constitution. Land-use controls are *de facto* takings as eminent domain, which must only be for the public good and for which the landowner is justly compensated. The New Orleans and other Gulf disasters demonstrate that wise land-use planning would certainly have provided for the public good. But the sticky issue is just how the landowners should be compensated. Actually, zoning and other ordinances have stood the tests of legal challenges for several decades, so it seems that a major reason for the lack of strong planning is a mix of politics and economics. Unfortunately, to get elected often requires immediacy. It takes much courage to run on long-term issues such as wetland protection, especially when others are offering short-term benefits (e.g., attracting industry, which in turn reduces taxes and offers jobs). The good news for engineers and other environmental professionals is that we are called to make the right decisions irrespective of politics. In reality, however, engineers all too often are beholden in some way to politics and economics (e.g., as line and staff employees in governmental agencies, as contractors to the city, and as beneficiaries of the short-term decisions). However, a glance at our first professional canon trumps these influences. We must hold paramount the health, safety, and welfare of the *public,* not the politicians and our bosses.

Actually, the concept of prevention is built into many engineering systems. For example, public impoundments usually proscribe any residential and most commercial buildings in the 100-year floodplain. Recall that the 100-year flood is a purely statistical concept. The Federal Emergency Management Agency characterizes a *100-year flood* as one whose magnitude is expected to be equaled or exceeded once on the average during any 100-year period. Thus, it is not a flood that will occur once every 100 years, but it is the flood elevation that has a 1% chance of being equaled or exceeded each year. It is less a temporal concept than a hydrological phenomenon.

Thus, the 100-year flood could occur more than once in a relatively short period of time, even several times in a single year. The 100-year flood is the standard applied by most federal and state agencies; for example, it is used by the National Flood Insurance Program (NFIP) as the benchmark for floodplain management and to determine the need or the eligibility for flood insurance. It is used by the U.S. Army Corps of Engineers and the Bureau of Reclamation to prevent encroachment near impoundments and lakes. It is also a common standard for local, regional, and state land-use planning. The challenge is that people become used to artificial "100-year" floods. Had the dikes and levees not existed in New Orleans, for example, much of the area would be in a "zero-year" floodplain (i.e., under water).

So, is it wise engineering practice to build another, better dike and levee system? As usual the answer depends on one's perspective. We recommend that a larger view in time and space be used instead of what is politically expedient. Or as Benjamin Franklin would suggest, should we apply an ounce of prevention (land-use proscriptions) instead of a future pound of cure (repairing and rebuilding again in the vulnerable delta)? Prevention comes in various forms and scales. The largest scale is planetary. The intensity of the storm begs the question of what is the role of global climate change, if any, in breeding large hurricanes and other extreme meteorological events. From a purely thermodynamic perspective, one would suspect the answer to be "yes." If the buildup of greenhouse gases has led to greater amounts of stored energy (i.e., infrared—heat—converted from incoming solar radiation), there is more energy that needs to be released. Hurricanes are simply the result of two energy systems on Earth, heat and motion. The heat is derived almost completely from the sun and is converted and transferred through complex systems in the atmosphere. Motion is the result of the Earth's rotation (i.e., the Coriolis effect), where the air is deflected, so that in cyclones (low-pressure systems) and anticyclones (high-pressure systems) are formed. When greater amounts of heat are formed, the mechanical and thermodynamic systems must become more intense, and if this logic is correct, the storms that result will probably become increasingly violent. Of course, there is much debate but a general consensus in the scientific community about the grounding assumption; that is, does the buildup of carbon dioxide, methane, and other gases in the Earth's atmosphere *really* increase global temperatures? Like many uncertainties in science, this is *important if true.*

It is not that engineers will not have plenty to design and build, such as constructed wetlands and hardened facilities within the floodplain. The key is finding the proper balance of knowing what to build and what to avoid building. Perhaps the biggest lesson for engineers is the need to approach everything we do from a perspective of sustainability. When we design, plan, and build, how does this fit with and affect other parts of the systems within which our projects will exist? We must do complete life-cycle analyses and design with the ends in mind. Otherwise, our work will merely be a patchwork or worse yet, end up being a toxic gumbo.

Bioaccumulation

Some substances are eliminated easily, while others build up in an organism's tissues. The likelihood that a substance will find its way into the food web is another important aspect of its hazard. Toxicokinetic models predict the dynamics of uptake, distribution, depuration, and elimination of contaminants within organisms. Persistence and bioaccumulation are interdependent. If the substance is likely to be sorbed to organic matter (i.e., high K_{oc} value), it will have an affinity for tissues. A substance that partitions from the aqueous to the organic phase (i.e., high K_{ow} value) is likely to be stored in fats of higher-trophic-level organisms (e.g., carnivores and omnivores). The *bioconcentration factor* (BCF) is the ratio of the concentration of the substance in a specific genus to the exposure concentration, at equilibrium. The exposure concentration is the concentration in the environmental compartment (almost always surface water). The BCF is similar to the *bioaccumulation factor* (BAF), but the BAF is based on the uptake of the organism from both water and food. The BCF is based on direct uptake from the water only. A BCF of 500 means that an organism takes up and sequesters a contaminant to concentrations 500 times greater than the exposure concentration. Generally, any substance that has a BAF or BCF $>$ 5000 is considered to be highly bioaccumulative, although the cutoff point can differ depending on the chemicals of concern, the regulatory requirements, and the type of ecosystem in need of protection.

It is important to note that genera will vary considerably in reported BCF values and that the same species will bioaccumulate different compounds at various rates. The amount of bioaccumulated contaminant increases generally with the size, age, and fat content of the organism and decreases with increasing growth rate and efficiency. Bioaccumulation also is often higher for males than for females and in organisms that are proficient in storing water. Top predators often have elevated concentrations of persistent, bioaccumulating toxic substances (known as PBTs).

The propensity of a substance to bioaccumulate is usually inversely proportional to its aqueous solubility, since hydrophilic compounds are usually more easily eliminated by metabolic processes. In fact, the first stages of metabolism often involve adding or removing functional groups to make it more water soluble. Generally, compounds with log $K_{ow} > 4$ can be expected to bioaccumulate. However, this is not always the case. For example, very large molecules [e.g., cross-sectional dimensions $>$ 9.5 angstroms (Å) and molecular weights $>$ 600] are often too large to pass through organic membranes (i.e., known as *steric hindrance*). Since, in general, the larger the molecule, the more lipophilic it becomes, some very lipophilic compounds (i.e. log $K_{ow} > 7$) will actually have surprisingly low rates of bioaccumulation, due to steric hindrance.

Bioaccumulation not only makes it difficult to find and measure toxic compounds but complicates how people and ecosystems can become exposed. For example, a release of a persistent, bioaccumulating substance can interfere with treatment plant efficiencies and greatly increase human exposures to pollutants that would otherwise have to be removed and treated. (This is another example of risk shifting, discussed at length in Chapter 3.)

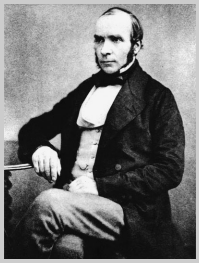
Biological Response

Various organisms respond to environmental insults in different ways, so even if a substance persists and is taken up by an organism, its hazards are still dependent on the

response of the organism after it comes into contact with the substance. This is the essence of the hazard; that is, does the chemical, physical, or biological agent elicit an adverse response?

This response is measurable. When a contaminant interacts with an organism, substances such as enzymes are generated as a response. Thus, measuring such substances in fluids and tissues can provide an indication or *marker* of contaminant exposure and biological effects resulting from the exposure. The term *biomarker* includes any such measurement that indicates an interaction between an environmental hazard and a biological system.[43] In fact, biomarkers may indicate any type of hazard: chemical, physical, and biological. An exposure biomarker is often an actual measurement of the contaminant itself or any chemical substance resulting from the metabolism and detoxification processes that take place in an organism. For example, measuring total lead (Pb) concentration in the blood, urine, or hair may be an acceptable exposure biomarker for people's exposures to Pb. However, other contaminants are better reflected by measuring chemical by-products, such as compounds that are rapidly metabolized upon entering an organism. Nicotine, for example, is not a very good indicator of smoking, but the metabolite, cotinine, can be a reliable indicator of nicotine exposure. Similarly, when breath is analyzed to see if someone has been drinking alcohol, the alcohol itself (i.e., ethanol) is

Biographical Sketch: John Snow



John Snow, the eldest son of a farmer, was educated at a private school and then apprenticed to a surgeon at the age of 14 in preparation for his career in medicine. After attending the Hunterian School of Medicine in London he became a public health physician, and in 1838 he became a member of the Royal College of Surgeons and, in 1850, the Royal College of Physicians.

In those days, London was plagued almost annually by cholera epidemics, and the medical field was trying desperately to understand what caused this dreaded disease. Some public health officials such as Edwin Chadwick thought that it was the bad odors that caused disease, whereas others thought it was ''miasma,'' vapor that rises out of rotting stuff and seeps into rooms and infects people while they are asleep (hence, nightcaps). The possibility that it was contaminated water was not widely believed since the microorganisms that cause cholera and other infectious disease had not been identified. But John Snow thought he knew and set out to prove that cholera was waterborne.

In 1853 when a particularly vicious epidemic struck London, he decided to do a special study by noting on a map the locations of the people who had died. This became the very first ''spot map'' in the history of epidemiology and allowed Snow to pinpoint the water pump on Broad Street as the most likely source of the infection. He convinced the city fathers to remove the handle on the pump and the epidemic subsided. It was more than 30 years later that Koch identified the pathogen causing cholera, but by that time the germ theory had become widely believed and embraced as the etiology of contagious diseases.

not usually a good indicator, but various metabolites, such as acetaldehyde, that have been formed as the body metabolizes the ethanol are excellent markers.

Exposure to ethanol by the oral pathway (i.e., drinking alcoholic beverages) illustrates the continuum of steps between exposure and response (see Figure 4.14). Table 4.4 gives examples of the types of biomarkers for a specific type of exposure (i.e., maternal alcohol consumption). Interestingly, the response and biomarkers for alcohol consumption are similar to those for some environmental contaminants, such as Pb, mercury (Hg), and PCBs.

Exposure biomarkers are also useful as an indication of the contamination of fish and wildlife in ecosystems. For example, measuring the activity of certain enzymes, such as ethoxyresorufin-*O*-deethylase (EROD), in aquatic fauna *in vivo* indicates that the organism has been exposed to planar halogenated hydrocarbons (e.g., certain dioxins and PCBs), PAHs, or similar contaminants. The mechanism for EROD activity in the aquatic fauna is the receptor-mediated induction of cytochrome P450–dependent monooxygenases when exposed to these contaminants.[44] The biological response does not necessarily have to respond to chemical stress. Stresses to environmental quality can also come about from ecosystem stress (e.g., loss of important habitats and decreases in the size of the population of sensitive species).

A substance may also be a *public welfare hazard* that damages property values or physical materials, expressed for example as its corrosiveness or acidity. The hazard may be inherent to the substance, but like toxicity, a welfare hazard usually depends on the situation and conditions where the exposure may occur.

Situations are most hazardous when a number of conditions exist simultaneously; witness the hazard to firefighters using water in the presence of oxidizers. The challenge to the engineer is how to remove or modify the characteristics of a substance that renders it hazardous, or to relocate the substance to a situation where it has value.

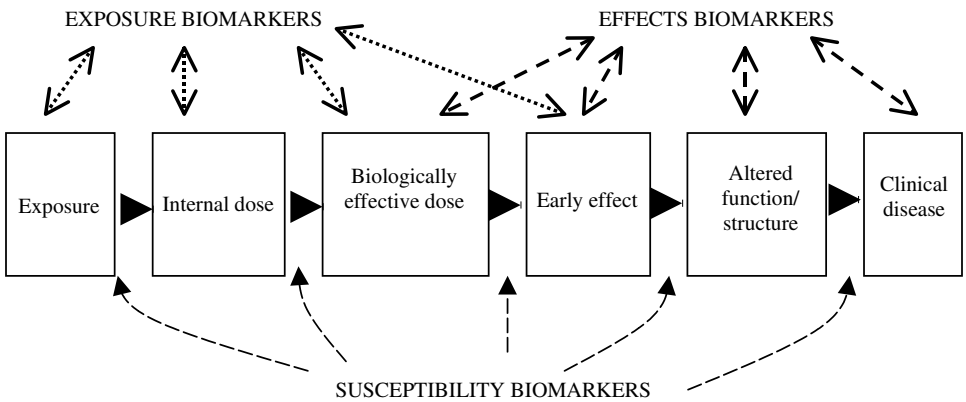


Figure 4.14 Continuum from exposure to a toxic substance to clinically diagnosed disease. The continuum is a time sequence, but the chemical to which the organism is exposed is not necessarily the same chemical in subsequent stages (i.e., metabolites are formed, which can serve as the biomarker). Enzymes produced to enhance metabolism or detoxification can also serve as biomarkers. Susceptibility biomarkers indicate increased vulnerability between the steps. [Adapted from C. F. Bearer, Markers to Detect Drinking during Pregnancy, *Alcohol Research and Health,* 25(3):210–218, 2001.]

Table 4.4 Examples of Biomarkers Following Oral Exposure to Ethanol in Pregnant Women

Exposure/effect step	Biomarker type	Example biomarkers
Internal dose	Alcohol ingestion	Blood ethanol concentration
Biologically effective dose	Ethanol metabolites	Acetaldehyde
		Ethyl glucuronide
		Fatty acid ethyl esters (FAEEs)
		Cocaethylene
Early effects	Enzymes in ethanol metabolic reactions	Cytochrome P450 2E1
		Catalase
		FAEE synthase
Alter function or structure	Target protein alteration	Carbohydrate-deficient transferring
		Serum proteins
		Urinary dolichols
		Sialic acid
	Early target organ damage	γ-Glutamyltransferase
		Aspartate aminotransferase/ alanine aminotransferase
		Mean corpuscular volume
		B-hexosaminidase
Clinical disease	Physiological response, including neurological damage and low birth weight, in newborn baby	Fetal alcohol syndrome

Source: Adapted from C. F. Bearer, Markers to Detect Drinking during Pregnancy, *Alcohol Research and Health,* 25(3):210–218, 2001.

Organic *versus* Inorganic Toxicants

Environmental contaminants fall into two major categories, organic and inorganic. *Organic compounds* are those that have at least one covalent bond between two carbon atoms or between a carbon and a hydrogen atom. Thus, the simplest hydrocarbon, methane (CH_4), has a bond between carbon and each of four hydrogen atoms. Organic compounds are subdivided between aliphatic (chains) and aromatic (rings) compounds. A common group of aliphatic compounds are the chain structures known as alkanes, which are hydrocarbons with the generic formula C_nH_{2n+2}. If these compounds have all of the carbon atoms in a straight line, they are considered ''normal'' and are known as *n*-alkanes. The simplest aromatic, benzene (C_6H_6), has bonds between carbon atoms and between carbon and hydrogen atoms (see Figure 4.15).

The structure of the compound determines its persistence, toxicity, and ability to accumulate in living tissue. Subtle structural differences can lead to very different environmental behaviors. Even various arrangements with identical chemical formulas (i.e., isomers) can exhibit very different chemical characteristics. For example, the boiling points at 1 atm for *n*-pentane, isopentane, and neopentane (all C_5H_{12}) are 36.1°C, 27.8°C, and 9.5°C, respectively. Among the most important factors are the length of the chains in aliphatic compounds and the number and configurations of the rings in aromatics.

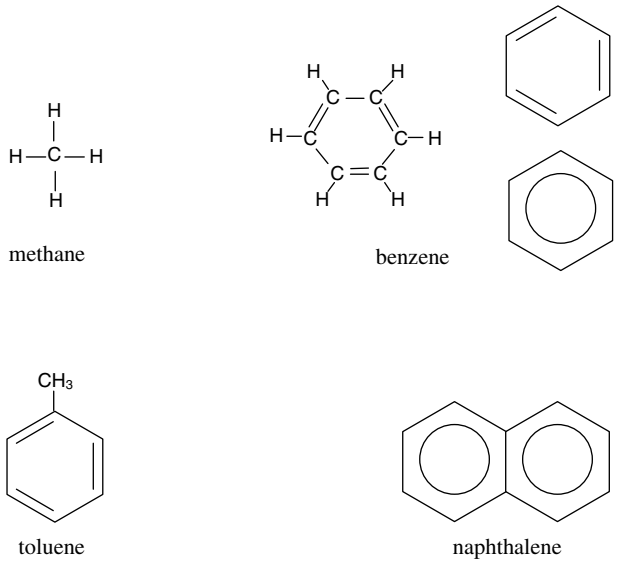
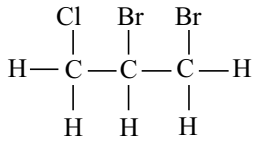


Figure 4.15 Organic compound structures. Methane is the simplest aliphatic structure, and benzene is the simplest aromatic structure. Note that the benzene molecule has alternating double and single bonds between the carbon atoms. The double and single bonds flip (i.e., resonate). This is why the benzene ring is also shown as the two structures on the right, which are the commonly used condensed form in aromatic compounds, such as the solvent toluene and the polycyclic aromatic hydrocarbon, naphthalene.

Arguably, substitutions are even more critical. For example, methane is a gas under environmental conditions, but it becomes a very toxic and bioaccumulating liquid (carbon tetrachloride or tetrachloromethane) when chlorine atoms (CCl_4) are substituted for the hydrogen atoms. Naphthalene, the simplest polycyclic aromatic hydrocarbon ($C_{10}H_8$), is considered to be a possible human carcinogen, but the data are not sufficient to calculate a slope factor. However, when an amine group (NH_2) substitutes for a hydrogen atom to form 2-naphthylamine ($C_{10}H_9N$), the inhalation cancer slope factor is 1.8 kg · day mg^{-1}. The formulation of pesticides takes advantage of the dramatic increases in toxicity by substitution reactions.

CASE STUDY: PESTICIDES AND STERILITY

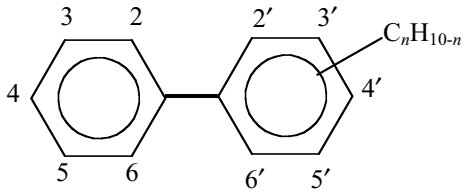
For many years both Shell Oil and Dow Chemical supplied a pesticide containing dibromochloropropane (DBCP) to Standard Fruit Company for use on its banana plantations, even though Shell Oil was aware since the 1950s that DBCP exposure is linked to sterility in laboratory animals. In spite of evidence that DBCP also causes sterility in humans and was banned in the United States, Shell continued to market the pesticide in Central America.



dibromochloropropane (DBCP)

In 1984, banana plantation workers from several Central American countries filed a class action suit against Shell, claiming that they became sterile and faced a high risk of cancer. In response, Shell claimed that it was inconvenient to continue the case because the workers were in Costa Rica, a claim that was quickly thrown out of court. Shell finally settled out of court with the Costa Rican workers and paid $20 million in damages to the 16,000 claimants. A scientist from Shell is quoted as saying: "Anyway, from what I hear they could use a little birth control down there" (quote from David Weir and Constance Matthiessen, Will the Circle Be Unbroken? *Mother Jones,* June 1989).

Congeners are configurations of a common chemical structure. For example, all polychlorinated biphenyls (PCBs) have two benzene rings bonded together at two carbon atoms. They also have at least one chlorine substitution around the rings, so that there are 209 possible configurations (i.e., 209 PCB congeners). Since the two benzene rings can rotate freely on the connecting bond, for any PCB congener (except decachlorobiphenyl, in which every hydrogen has been substituted by a chlorine), the location of chlorines can differ (e.g., 2,3,4-trichlorobiphenyl is the same as 2′,3,4-trichlorobiphenyl and the same as 2,4′,6′- trichlorobiphenyl). The location of the chlorine atoms can lead to different physical, chemical, and biological characteristics of molecules, including their toxicity, persistence, and bioaccumulation potential.



polychlorinated biphenyl structure

Numerous acids are organic, because they contain the C—C and C—H bonds. For example, acetic acid ($HC_2H_3O_2$), benzoic acid ($HC_7H_5O_2$), and cyanoacetic acid ($C_3H_3NO_2$) are organic acids. Like other compounds, organic acids can have substitutions that change their hazard, such as when acetic acid's hydrogen atoms are substituted with chlorines to form trichloroacetic acid ($C_2HCl_3O_2$).

Inorganic compounds are those that do not contain carbon-to-carbon or carbon-to-hydrogen covalent bonds. Thus, even carbon-containing compounds can be inorganic.

For example, the pesticides sodium cyanide (NaCN) and potassium cyanide (KCN) are inorganic compounds, as are the gases carbon monoxide (CO) and carbon dioxide (CO_2), compounds that contain the anions carbonate (CO_3^{2-}) and bicarbonate (HCO_3^-), and inorganic acids, such as carbonic acid (H_2CO_3) and cyanic acid (HCNO).

Metals are particularly important in environmental situations. Like other elements, the compounds formed by metals vary in their toxicity and how rapidly they move and change in the environment. However, certain metals, no matter what their form are hazardous. Unlike carbon, hydrogen, oxygen, and many other elements, which in certain configurations are essential and in others are toxic, heavy metals and metalloids are considered hazardous no matter what the chemical species. For example, any amount of lead or mercury in any form is considered toxic, although some forms are much more toxic than others. And since metals and metalloids are elements, we are not going to be able to ''destroy'' them as we do organic compounds by using chemical, thermal, and biological processes. Destruction simply means that we are changing compounds into simpler compounds (e.g., hydrocarbons are broken down to CO_2 and H_2O). But metals are already in elemental form. So the engineer must attempt to change the metal or metalloid to make it less toxic and less mobile, and once that is done, to take measures to keep the metal wastes away from people, wildlife, and other receptors.

The oxidation state or *valence* of metals and metalloids is the most important factor in their toxicity and mobility. The outermost electrons determine how readily an element will enter into a chemical reaction and what type of reaction will occur. This is the oxidation number of the element. Most metals contain more than one oxidation state, each with its own toxicity and mobility characteristics. However, in most cleanup situations, all forms of the metal, even those with low toxicity and mobility, must be removed since when environmental conditions change the metals may change to more toxic and mobile forms.

CASE STUDY: JERSEY CITY CHROMIUM

One of the biggest selling points for automobiles in the 1950s and 1960s was the amount of "chrome" displayed. To this day, the metal chromium (Cr) is in high demand since it strongly resists corrosion and oxidation. As such, it is an ingredient of stainless steel and is used to plate other metals.

Jersey City, in Hudson County, New Jersey, was once the chromium-processing capital of the United States, and over the years, 20 million tons of chromate ore processing residue was sold or given away as fill. The city has had at least 120 contaminated sites, including ball fields and basements underlying homes and businesses. It has not been uncommon for brightly colored chromium compounds to crystallize on damp basement walls and to "bloom" on soil surfaces where soil moisture evaporates, creating something like an orange hoar frost of hexavalent chromium, Cr^{6+}. A broken water main in the wintertime resulted in the formation of bright green ice due to the presence of trivalent chromium, Cr^{3+}.

The companies that created the chromium waste problem no longer exist, but liability was inherited by three conglomerates through a series of takeovers. In 1991, Florence Trum, a local resident, successfully sued Maxus Energy, a subsidiary of one of the conglomerates, for the death of her husband, who loaded trucks in a warehouse built directly over a chromium waste

disposal site. He developed a hole in the roof of his mouth and cancer of the thorax, and it was determined by autopsy that his death was caused by chromium poisoning. Although the subsidiary company did not produce the chromium contamination, the judge ruled that company managers knew about the hazards of chromium. Such assumed liability is part of the regulations for *potentially responsible parties* (PRPs) under the Superfund law.

The state of New Jersey initially spent $30 million to locate, excavate, and remove some of the contaminated soil. But the extent of the problem was overwhelming and they stopped these efforts. The director of toxic waste cleanup for New Jersey admitted that even if the risks of living or working near chromium were known, the state does not have the money to remove it. Initial estimates for site remediation were well over $1 billion.[45] Citizens of Hudson County were angry and afraid. Those sick with cancer wondered if it could have been prevented. Mrs. Trum perceived the perpetrators as well-dressed business people who were willing to take chances with other peoples' lives. "Big business can do this to the little man . . . ," she said.

The contamination in Jersey City is from industries that used chromium in their processes, including metal plating, leather tanning, and textile manufacturing. The deposition of this chromium residue in dumps has resulted in chromium-contaminated water, soils, and sludge. Chromium is particularly difficult to regulate because of the complexity of its chemical behavior and toxicity, which translates into scientific uncertainty. Uncertainty exacerbates the tendency of regulatory agencies to make conservative and protective assumptions, the tendency of the regulated to question the scientific basis for regulations, and the tendency of potentially exposed citizens to fear potential risk.

Chromium exists in nature primarily in one of two oxidation states: Cr^{3+} and Cr^{6+}. In the reduced form of chromium, Cr^{3+}, there is a tendency to form hydroxides which are relatively insoluble in water at neutral pH values. Cr^{3+} does not appear to be carcinogenic in animal and bioassays. In fact, organically complexed Cr^{3+} has recently become one of the more popular dietary supplements in the United States and can be purchased commercially as chromium picolinate ($C_{18}H_{12}CrN_3O_6$) or with trade names such as Chromalene to help with proper glucose metabolism, to control blood fat concentrations, to aid weight loss and muscle tone, and as essential to gene expression.

When Cr^{3+} oxidized as Cr^{6+}, however, chromium is highly toxic. It is implicated in the development of lung cancer and skin lesions in industrial workers. In contrast to Cr^{3+}, nearly all Cr^{6+} compounds have been shown to be potent mutagens. The U.S. EPA has classified chromium as a human carcinogen by inhalation based on evidence that Cr^{6+} causes lung cancer. However, by ingestion, chromium has not been shown to be carcinogenic.

What confounds the understanding of chromium chemistry is that under certain environmental conditions, Cr^{3+} and Cr^{6+} can interconvert. In soils containing manganese, Cr^{3+} can be oxidized to Cr^{6+}. Given the heterogeneous nature of soils, these redox reactions can occur simultaneously. Al-

though organic matter may serve to reduce Cr^{6+}, it may also complex Cr^{3+} and may make it more soluble—facilitating its transport in groundwater and increasing the likelihood of encountering oxidized manganese present in the soil.

Cleanup limits for chromium are still undecided, but through the controversy there have evolved some useful technologies to aid in resolution of the disputes. For example, analytical tests to measure and distinguish between Cr^{3+} and Cr^{6+} in soils have been developed. Earlier in the history of New Jersey's chromium problem, these assays were not reliable and would have necessitated remediating to soil concentrations based on total chromium. Other technical/scientific advances include *in situ* remediation strategies designed to reduce chemically Cr^{6+} to Cr^{3+} in order to reduce risk without excavation and removal of soil designated as hazardous waste. The establishment of cleanup standards is anticipated, but the proposed endpoint based on contact dermatitis is controversial. Although some perceive contact dermatitis as a legitimate claim to harm, others have jokingly suggested regulatory limits for poison ivy, which also causes contact dermatitis. The methodology by which dermatitis-based soil limits were determined has come under attack by those who question the validity of skin patch tests and the inferences by which patch test results translate into soil Cr^{6+} levels.

The Jersey City community's frustration with slow cleanup and what citizens perceive as double-talk by scientists finally culminated in the unusual step of amending the state constitution to provide funds for hazardous waste cleanups. State environmentalists depicted the constitutional amendment as a referendum on Governor Christine Todd Whitman's (R) environmental record, which they perceived as relaxed enforcement and reduced cleanups. (Whitman was the first administrator of the U.S. Environmental Protection Agency to be named by President George W. Bush.)

Radioisotopes

Different atomic weights of a same element are the result of different numbers of neutrons. The number of electrons and protons of stable atoms must be the same. Elements with differing atomic weights are known as *isotopes*. An element may have numerous isotopes. Stable isotopes do not undergo natural radioactive decay, whereas radioactive isotopes involve spontaneous radioactive decay as their nuclei disintegrate, thus are known as *radioisotopes.* This decay leads to the formation of new isotopes or new elements. The stable product of an element's radioactive decay is known as a *radiogenic isotope.* For example, lead (Pb; atomic number = 82) has four naturally occurring isotopes of different masses (^{204}Pb, ^{206}Pb, ^{207}Pb, ^{208}Pb). Only ^{204}Pb is stable. The isotopes ^{206}Pb and ^{207}Pb are daughter (or progeny) products of the radioactive decay of uranium (U), while ^{208}Pb is a product of thorium (Th) decay. Owing to the radioactive decay, the heavier isotopes of lead will increase in abundance compared to ^{204}Pb. The toxicity of a radioisotope can be twofold (i.e., chemical toxicity and radioactive toxicity). For example, Pb is neurotoxic no matter the atomic weight, but if people are exposed to its unstable

isotopes they are also threatened by radiation emitted from decay of the nucleus. The energy of the radioactive decay can alter genetic material and lead to mutations, including cancer.

CASE STUDY: RADIATION POISONING IN GOIANIA, BRAZIL[46]

Sometimes good intentions lead to unfortunate consequences. In the early 1980s a small cancer clinic was opened in Goiana, Brazil, but business was not good and the clinic closed five years later. Left behind in the abandoned building were a radiation therapy machine and some canisters containing waste radioactive material—1400 curies of cesium 137, which has a half-life of 30 years. In 1987 the container of cesium 137 was discovered by local residents and was opened, revealing a luminous blue powder. The material was a local curiosity and children even used it to paint their bodies, which caused them to sparkle. One of the little girls went home for lunch and ate a sandwich without first washing her hands. Six days later she was diagnosed with radiation illness, having received an estimated five to six times the lethal radiation exposure for adults. The ensuing investigation identified the true content of the curious barrel. In all, over 200 persons had been contaminated and 54 were serious enough to be hospitalized, with four people dying from the exposure (including the little girl with the sandwich). Treatment of radiation disease is challenging. The International Atomic Energy Commission characterized the treatment of the Goianian patients as follows:

> . . . the first task was to attempt to rid their bodies of cesium. For this, they administered Prussian blue, an iron compound that bonds with cesium, aiding its excretion. The problem in this case was the substantial delay—at least a week—from initial exposure to treatment. By that time much of the cesium had moved from the bloodstream into the tissues, where it is far more difficult to remove . . . the patients were also treated with antibiotics as needed to combat infections and with cell infusions to prevent bleeding. . . .[47]

By the time the government mobilized the response, the tragic damage was done. A large fraction of the local population had received excessive radiation exposures, and the export of produce from Goiania dropped to zero, creating a severe economic crisis. The incident is now recognized as the second-worst radiation accident in the world, second only to the explosion of the nuclear power plant in Chernobyl.

Factors of Safety

Of the myriad of chemicals in the environment, workplace, and home, relatively few have been associated with chronic diseases such as cancer. However, for those that do, risk seldom is zero. Simple mathematics tells us that if the hazard is zero, the risk must be zero. So only a carcinogen can cause cancer. No matter what the dose, the cancer risk from a noncarcinogen is zero. A prominent hypothesis in carcinogenesis is the *two-hit theory,* suggested by A. G. Knudson[48] in 1971. The theory argues that cancer develops

after genetic material [i.e., usually deoxyribonucleic acid (DNA)] is damaged. The first damage is known as *initiation.* This step may, but does not necessarily, lead to cancer. The next step, *promotion,* changes the cell's makeup and nature, such as the loss of normal homeostasis (cellular self-regulation) and the rapid division of clonal tumor cells. Promoters may or may not be carcinogens. So when we say that a noncarcinogen dose cannot lead to cancer, we are talking specifically of compounds that initiate cancer, since exposure to noncarcinogenic promoters, such as excessive dietary fats, can hasten the onset of cancer cells.

The RfD is the principal factor of safety used in assigning hazard to noncarcinogens. The slope factor (SF) is the principal hazard characteristic for carcinogens. Both factors are developed from a mix of mutagenicity studies, animal testing, and epidemiology. Unlike the RfD, which provides a ''safe'' level of exposure, cancer risk assessments generally assume that there is no threshold. Thus, the thresholds NOAEL and LOAEL are meaningless for cancer risk. Instead, cancer slope factors are used to calculate the estimated probability of increased cancer incidence over a person's lifetime [called the *excess lifetime cancer risk* (ELCR)]. Slope factors are expressed in inverse exposure units since the slope of the dose–response curve is an indication of risk per exposure. Thus, the units are the inverse of mass per mass per time, usually $(\text{mg kg}^{-1}\ \text{day}^{-1})^{-1} = \text{kg} \cdot \text{day mg}^{-1}$. This means that the product of the cancer slope factor and exposure (i.e., risk) is dimensionless. This should make sense because risk is a unitless probability of adverse outcomes. The SF values are contaminant- and route-specific. Thus, one must not only know the contaminant, but how a person is exposed (e.g., via inhalation, via ingestion, or through the skin). Inhalation, oral, and dermal cancer slope factors are shown in Table 4.5.

The more potent the carcinogen, the larger the slope factor will be (i.e., the steeper the slope of the dose–response curve). Note, for example, that when inhaled, ingested, or dermally exposed, the slope for the most carcinogenic dioxin tetrachlorodibenzo-*p*-dioxin, is eight orders of magnitude steeper than the slope for aniline. Keep in mind that this is the linear part of the curve. The curve is actually sigmoidal because at higher doses the effect is dampened (i.e., the response is increasing at a decreasing dosage rate). This process is sometimes called the saturation effect. One way to think about this is to consider that if the dose–response curve comes from animal tests of various doses there is a point at which increasing the dose of a chemical adds little to the onset of tumors. The dosage approaches an effective limit and becomes asymptotic. So if chemical A is given to 1000 rats, at increasing dosages an incremental increase in rats with tumors is seen. This is the linear range. Doubling the dose doubles the effect. But at some inflection point, say after 50 rats with tumors, if the dose is doubled, half as many additional rats with tumors are seen. The rate continues to decrease up to a point where even very large doses do not produce many additional tumors. This is one of the challenges of animal experiments and models. Dose is substituted for time; the assumed lifetime of humans is about 70 years, and the doses to carcinogens are usually very small (e.g., parts per billion or trillion). Animal doses may last only a few months and use relatively high doses. We have to extrapolate long-term effects from limited data from short-term studies. The same is somewhat true for human studies, where we try to extrapolate effects from a small number of cases to a much larger population (e.g., a small study comparing cases to controls in one hospital, or a retrospective view of risk factors that may have led to a cluster of cases of cancer).

Table 4.5 Cancer Slope Factors for Some Environmental Contaminants[a]

Contaminant	Inhalation slope factor ($kg \cdot day\ mg^{-1}$)	Oral slope factor ($kg \cdot day\ mg^{-1}$)	Dermal slope factor ($kg \cdot day\ mg^{-1}$)
Acrylonitrile	2.38×10^{-1}	5.40×10^{-1}	6.75×10^{-1}
Aniline	5.70×10^{-3}	5.70×10^{-3}	1.14×10^{-3}
Arsenic	1.51×10^{1}	1.50	1.58×10^{1}
Atrazine	4.44×10^{-1}	2.22×10^{-1}	4.44×10^{-1}
Benzene	2.90×10^{-2}	2.90×10^{-2}	3.22×10^{-2}
Benz[*a*]anthracene	3.10×10^{-1}	7.30×10^{-1}	1.46
Benzo[*a*]pyrene	3.10	7.30	1.46×10^{1}
Benzo[*b*]fluoranthene	3.10×10^{-1}	7.30×10^{-1}	1.46
Bis(2-chloroethyl)ether	1.16	1.16	1.13
Bis(2-chloroisopropyl)ether (DEHP)	3.50×10^{-2}	1.10×10^{-2}	8.75×10^{-2}
Bis(2-ethylhexyl)phthalate	1.40×10^{-2}	7.00×10^{-2}	2.80×10^{-2}
Bromodichloromethane	6.20×10^{-2}	6.20×10^{-2}	6.37×10^{-2}
Bromoform	3.85×10^{-3}	7.90×10^{-3}	1.05×10^{-2}
Cadmium	Not given	6.30	Not given
Chlordane	3.50×10^{-1}	3.50×10^{-1}	4.38×10^{-1}
Chloroethane (ethyl chloride)	2.90×10^{-3}	2.90×10^{-3}	1.28
Chloroform	8.05×10^{-2}	6.10×10^{-3}	6.10×10^{-3}
Chloromethane	3.50×10^{-3}	1.30×10^{-2}	1.63×10^{-2}
Chromium(VI)	3.50×10^{-3}	Not given	Not given
DDD	2.40×10^{-1}	2.40×10^{-1}	3.00×10^{-1}
Dichlorobenzene,1,4-	2.20×10^{-2}	2.40×10^{-2}	2.40×10^{-2}
Dieldrin	1.61×10^{1}	1.61×10^{1}	1.60×10^{1}
Dinitrotoluene, 2,4-	6.80×10^{-1}	6.80×10^{-1}	6.80×10^{-1}
Dioxane, 1,4-	2.20×10^{-2}	1.11×10^{-2}	2.20×10^{-2}
Diphenylhydrazine, 1,2-	7.70×10^{-1}	8.00×10^{-1}	1.60
Ethylene oxide	3.50×10^{-1}	1.02	1.28
Formaldehyde	4.55×10^{-2}	Not given	Not given
Heptachlor epoxide	9.10	9.10	2.28×10^{1}
Hexachlorobenzene	1.61	1.60	2.00
Hexachlorocyclohexane, α	6.30	6.30	6.47
Hexachlorocyclohexane, β	1.80	1.80	1.99
Hexachlorocyclohexane, γ (lindane)	1.30	1.30	1.31
Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)	2.22×10^{-1}	1.11×10^{-1}	2.22×10^{-1}
Nitrosodi-*n*-propylamine, *n*-	7.00	7.00	1.47×10^{1}
Pentachlorophenol	1.20×10^{-1}	1.20×10^{-1}	2.40×10^{-1}
Polychlorinated biphenyls (Arochlor mixture)	3.50×10^{-1}	2.00	2.35
Tetrachlorodibenzo-*p*-dioxin, 2,3,7,8-	1.16×10^{5}	1.50×10^{5}	1.68×10^{5}
Tetrachloroethane,1,1,1,2-	2.59×10^{-2}	2.60×10^{-2}	3.25×10^{-2}
Tetrachloroethane,1,1,2,2-	2.03×10^{-1}	2.03×10^{-1}	2.86×10^{-1}
Tetrachloroethene (PCE)	2.00×10^{-3}		5.20×10^{-2}
Tetrachloromethane (carbon tetrachloride)	5.25×10^{-2}	1.30×10^{-1}	1.53×10^{-1}

Table 4.5 (*Continued*)

Contaminant	Inhalation slope factor ($kg \cdot day\ mg^{-1}$)	Oral slope factor ($kg \cdot day\ mg^{-1}$)	Dermal slope factor ($kg \cdot day\ mg^{-1}$)
Toxaphene	1.12	1.10	1.75
Trichloroethane,1,1,2-	5.60×10^{-2}	5.70×10^{-2}	7.04×10^{-2}
Trichloroethene (TCE)	6.00×10^{-3}	1.10×10^{-2}	1.16×10^{-2}
Trichlorophenol, 2,4,6-	1.10×10^{-2}	1.10×10^{-2}	2.20×10^{-2}
Trichloropropane, 1,2,3-	8.75	7.00	8.75
Trinitrotoluene, 2,4.6- (TNT)	6.00×10^{-2}	3.00×10^{-2}	6.00×10^{-2}
Vinyl chloride	3.00×10^{-1}	1.90	2.17

Source: U.S. Environmental Protection Agency, *Integrated Risk Information System,* U.S. EPA, Washington, DC, 2002; U.S. Environmental Protection Agency, *Health Effects Summary Tables,* U.S. EPA, Washington, DC, 1994.

[a]These values are updated periodically. If a carcinogen is not listed in the table, visit http://risk.lsd.ornl.gov/tox/rap_toxp.shtml.

It can be argued that addressing rare and chronic diseases such as cancer, endocrine dysfunction, reproductive disorders, and neurological diseases is an effort in the control of variables to reduce the possibility of an improbable (thankfully!) event. New statistical techniques are being developed to help engineers deal with rare events.

Discussion: Small Changes

Small changes can be very profound in rare events. If you think about it, when you start with very small numbers, a slight change can make a difference. Stockbrokers and retailers use this phenomenon often. For example, a company may be the fastest-growing company in its field this year. Upon investigation, its sales may have been only \$5 last year but grew to \$5000 this year, a 1000-fold increase. Real estate investors might say that sales grew 100,000% this year, whereas engineers and scientists generally prefer absolute terms and might say that the growth rate was $\$4.995 \times 10^3\ yr^{-1}$. Both of these are correct statements. But would you rather invest in a company that had \$10 million in sales last year and grew to \$20 million this year? That is only a doubling of the income, or only 100% growth. But the absolute growth is $\$1 \times 10^6\ yr^{-1}$, or three orders of magnitude greater than that for the small firm. What does this tell us about rare outcomes such as cancer?

In reviewing epidemiological information, are the data given an incidence of disease or prevalence? Disease *incidence* is the number of new cases diagnosed each year, whereas *prevalence* is the number of cases at any given time. We must also be careful to ascertain whether the values are absolute or relative. For example, are the values given a year-over-year change, or are they simply a one-time event? In environmental and public health reports, especially risk assessments, the values are often presented as probabilities in engineering

notation; for example, a common target of cleanup of hazardous waste sites is that no more than one additional case of cancer per million population should result from the clean site (i.e., the added risk is less than or equal to 10^{-6}). Like all probabilities, this is simply a fraction and a decimal. However, if the engineer uses it in a public forum, it can be very disarming and not clearly understood. In fact, the entire concept of population risk is foreign to most people. The point is that when the engineer goes about explaining rare events such as cancer, great care must be taken.

The science of toxicology deals with even smaller values and often very limited data. In fact, one of the raging toxicological debates is that of cancer dose–response and where to literally "draw the line." As a matter of scientific policy, in what is known as the *precautionary principle,* many health agencies around the world assume that a single molecule of a carcinogen *can* cause cancer. In other words, there is no threshold under which a dose, no matter how small, would be safe; "one hit potentially leads to a tumor." This approach is commonly known as the *one-hit model.* Most other diseases have such a threshold dose, known as the *no observed adverse effect level* (NOAEL; shown in Figure 4.7). The precautionary principle is in large part due to our lack of understanding of how things work at the molecular level. Toxicological models

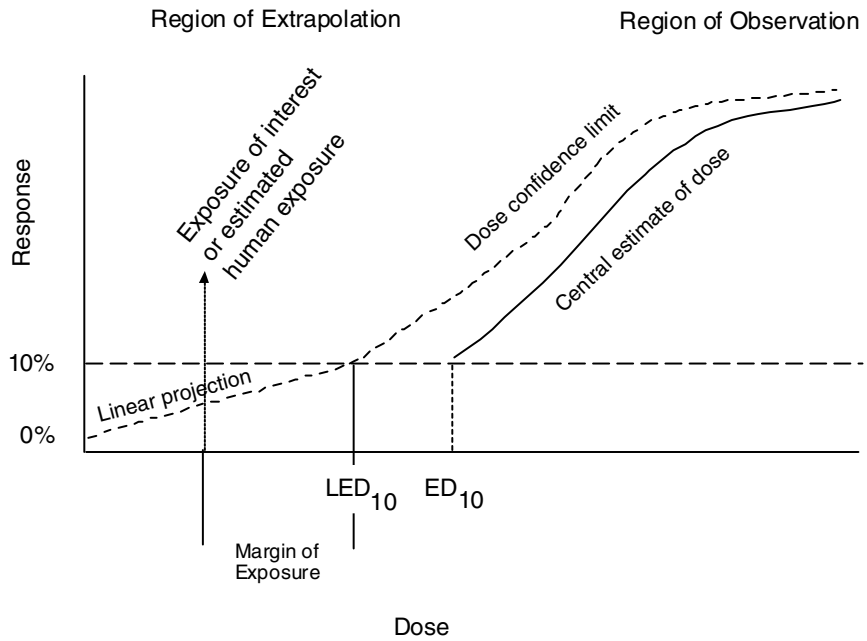


Figure 4.16 Linearized multistage dose–response curve showing the two major regions of data availability. LED_{10} = lower 95% confidence limit on a dose associated with 10% extra risk; ED_{10} = estimate of the dose that would lead to 10% increase in the response (in this case, cancer). (From D. A. Vallero, *Environmental Contaminants: Assessment and Control,* Elsevier Academic Press, Burlington, MA, 2004.)

work better when they use observed data, but at level below this, we are guessing (albeit a very educated guess) as to what is happening (see Figure 4.16). Since risk at very low doses is not directly measurable using animal experiments or from epidemiology, mathematical models are used to extrapolate from high to low doses.

Numerous extrapolation models or procedures may reasonably fit the observed data; however, extremely large differences of risk at low doses can be calculated. Scientists must use different models, depending on the particular chemical compound, as well as use information about how cancer seems to be occurring (i.e., the biological *mechanism of action* at work in the cell).[49] When such biological information is limited, the default is to assume linearity, and since there is no threshold, the curve intersects the *x*-axis and the *y*-axis at 0. For example, the U.S. Environmental Protection Agency usually recommends a linearized multistage procedure as the default model unless sufficient information to the contrary exists. The linearized multistage procedure calls for the fitting of a multistage model to the data. *Multistage models* are exponential models approaching 100% risk at high doses, with a shape at low doses given by a polynomial function. If this is first degree, the model is equivalent to a one-hit model, yielding almost a linear relationship between low dose and cancer risk. An upper bound risk is estimated by applying an appropriate linear term to the statistical bound for the polynomial. At sufficiently small exposures, any higher-order terms in the polynomial are assumed to be negligible, and the graph of the upper bound will appear to be a straight line. The slope of this line is called the *slope factor,* which is a measure of the cancer potency of the compound (i.e., the steeper the slope, the more potent the carcinogen).[50]

The units that we use in engineering can make risk communication unclear. For example, when we treat pollution, we often use a measure of pollutant removal efficiency, such as ''percent removal.'' To see how well an incinerator is destroying a hazardous substance, engineers report the removal efficiency for that compound. In fact, the environmental engineering community uses the *rule of six nines* for extremely hazardous compounds. For example, if the most toxic form of dioxin, tetrachlorodibenzo-*p*-dioxin (TCDD) is in a waste stream, the incinerator must destroy 99.9999% (six nines) of the TCDD. If the incinerator is destroying 99.9998%, theoretically it is out of compliance (of course, this begs the question about the worthiness of our measurement techniques and significant figures, but that is another matter!). Often, however, the removal is reported in units of mass or concentration. If a waste contains a total of 100 mg (mass), or 100 mg L^{-1} (concentration), of TCDD, after treatment in a properly operating incinerator, we are left with 0.0001 mg if we started with 100 mg (100 mg $-$ 0.999999 $\times$ 100 mg). If the incinerator increases its efficiency by seven nines (99.99999% removal), we would have 0.00001 mg of TCDD left; that is, the improvement allowed us to remove only 0.00009 mg of TCDD. If you want to make this incinerator improvement look better, you report it as nanograms (ng) removed (10 ng better). If you want to make the difference look insignificant, you report it as grams removed (only 0.00001 g removed). But both removal efficiencies are the same; only the units differ.

A further problem is that removal efficiency is a relative measure of success. If a waste has a large amount of a contaminant, even relatively inefficient operations look

good. Taking the TCDD example; if waste A has 100 g of TCDD (scary thought!) and waste B has 100 ng of TCDD and they both comply with the rules of six nines, the waste A incinerator is releasing 0.0001 g or 100 ng of the contaminant to the atmosphere, whereas the waste B incinerator is emitting only 0.0001 ng. That is why environmental laws also set limits on the maximum mass or concentration of a contaminant leaving the stack (or pipe, for water discharges). In addition, the laws require that for some pollutants the ambient concentration not be exceeded. However, for many very toxic compounds that require elaborate and expensive monitoring devices, such ambient monitoring is infrequent and highly localized (e.g., near a known polluter). Regulators often depend on self-reporting by the facilities, with occasional audit (analogous to the IRS accepting a taxpayer's self-reporting, which is verified to some extend by audits of a certain sample of taxpayers).

Statistics and probabilities for extreme and rare events can be perplexing. People want to know about trends and differences in exposures and diseases between their town or neighborhood and those of others. Normal statistical information about central tendencies such as the mean, median, and mode, or ranges and deviations, fail us when we analyze rare events. Normal statistics allows us to characterize the typical behaviors in our data in terms of differences between groups and trends, focusing on the center of the data. *Extreme value theory* (EVT), conversely, lets us focus on the points far out on the tail of our data, with the intent of characterizing a rare event. For example, perhaps we have been collecting health data for 10 years for thousands of workers exposed to a contaminant. What is special about those who have been most highly exposed (e.g., those at the 99th percentile)? What can we expect as the highest exposures over the next 50 years? EVT is one means of answering these questions. The first question can be handled with traditional statistics, but the second is an extrapolation (50 years hence) beyond our data set.

Such extrapolations in EVT are justified by a combination of mathematics and statistics (i.e., probability theory and inference and prediction, respectively). This can be a very powerful analytical tool. However, the challenge may come after the engineer has completed the analysis. The engineer may be confident that the neighborhood does not involve much additional risk based on EVT and traditional methods. But how does the engineer explain how such a conclusion was derived? Many in the audience have not taken a formal course in basic statistics, let alone a course that deviates from the foundations of statistics, such as EVT! Senol Utku, a former colleague at Duke, was fond of saying: ''To understand a non-banana, one must first understand a banana.'' This was in the context of discussing the value of linear relationships in engineering. Everyone recognizes that many engineering and scientific processes and relationships are nonlinear in their behavior, but students must first learn to apply linear mathematics. Our advice is to use the best science possible, but be ready to support your approaches in understandable ways, targeted to the specific audience.

Exposure Estimation

Now consider the second part of the risk equation. An *exposure* is any contact with an agent. For chemical and biological agents, contact can come about from a number of

exposure pathways (i.e., routes taken by a substance), from its source to its endpoint (i.e., a target organ such as the liver, or a location short of that, such as in fat tissues). The substances often change to other chemical species as a result of the body's metabolic and detoxification processes. These new substances are known as *degradation products* or *metabolites.*

Physical agents such as electromagnetic radiation, ultraviolet (UV) light, and noise do not follow this pathway exactly. The contact with these sources of energy can elicit a physiological response that may generate endogenous chemical changes that behave somewhat like metabolites. For example, UV light may infiltrate and damage skin cells. The UV light helps to promote skin-tumor promotion by activating the transcription factor complex activator protein-1 (AP-1) and enhancing the expression of the gene that produces the enzyme cyclooxygenase-2 (COX2). Noise (i.e., acoustical energy), can also elicit physiological responses that affect an organism's chemical messaging systems (i.e., endocrine, immune, and neural).

The exposure pathway also includes the manner in which people can come into contact with (i.e., be exposed to) the agent. The pathway has five parts:

1. The source of contamination (e.g., a stack or pipe)
2. An environmental medium and transport mechanism (e.g., the air)
3. A point of exposure (e.g., indoor air)
4. A route of exposure (e.g., inhalation, dietary ingestion, nondietary ingestion, dermal contact, nasal route)
5. A receptor population (those who are actually or potentially exposed)

If all five parts are present, the exposure pathway is known as a *completed exposure pathway.* In addition, the exposure may be short-term, intermediate, or long-term. *Short-term contact* is known as an *acute exposure* [i.e., occurring as a single event or for only a short period of time (up to 14 days)]. An *intermediate exposure* is one that lasts from 14 days to less than one year. *Long-term* or *chronic exposures* are greater than one year in duration.

Determining the exposure for a neighborhood can be complicated. For example, even if we do a good job of identifying all of the contaminants of concern and the possible source of these pollutants (no small task), we may have little idea of the extent to which the receptor population has come into contact with these contaminants (steps 2 through 4). Thus, assessing exposure involves not only the physical sciences but also the social sciences (e.g., psychology and behavioral sciences). People's activities greatly affect the amount and type of exposure. That is why exposure scientists use a number of techniques to establish activity patterns, such as asking potentially exposed individuals to keep diaries, videotaping, using telemetry to monitor vital information (e.g., heart and ventilation rates), and comparing individual records to biomarkers (e.g., cotinine in urine as indication of tobacco smoking).

Ambient measurements, such as air pollution monitoring equipment located throughout cities, are generally not good indicators of actual population exposures. Neither, necessarily, are gross production and release estimates. The metals lead (Pb) and mercury (Hg) and their compounds comprise the greatest mass of toxic substances released into

the U.S. environment. This is due largely to the large volume and surface areas involved in metal extraction and refining operations. However, this does not necessarily mean that more people will be exposed at higher concentrations or more frequently to these compounds than to others. The mere fact that a substance is released or even that it is found in the ambient environment is not tantamount to its coming in contact with people. Conversely, even a small amount of a substance under the right circumstances can lead to very high levels of exposure (e.g., in an occupational setting, in certain indoor environments, and through certain pathways, such as nondietary ingestion of paint chips by children).

The Lawrence Berkley National Laboratory recently demonstrated the importance of not simply assuming that the released or even background concentrations are a good indicator of actual exposure.[51] The researchers were interested in how sorption may affect indoor environments, so they set up a room (chamber) made up of typical building materials and furnished with actual furniture such as that found in most residential settings. A number of air pollutants were released into the room and monitored. Figure 4.17 shows an organic solvent, xylene, exhibiting the effects of sorption. With the room initially sealed, the decay observed in vapor-phase concentrations indicates that the compound is adsorbing onto surfaces (walls, furniture, etc.). The adsorption continues for hours, with xylene concentrations reaching a quasi-steady state. At this point the room is flushed with clean air to free all vapor-phase xylene. Shortly after the flush, the xylene concentrations began to rise again, until reaching a new steady state. This rise must be the result of desorption of the previously sorbed xylene, since the initial source is gone.

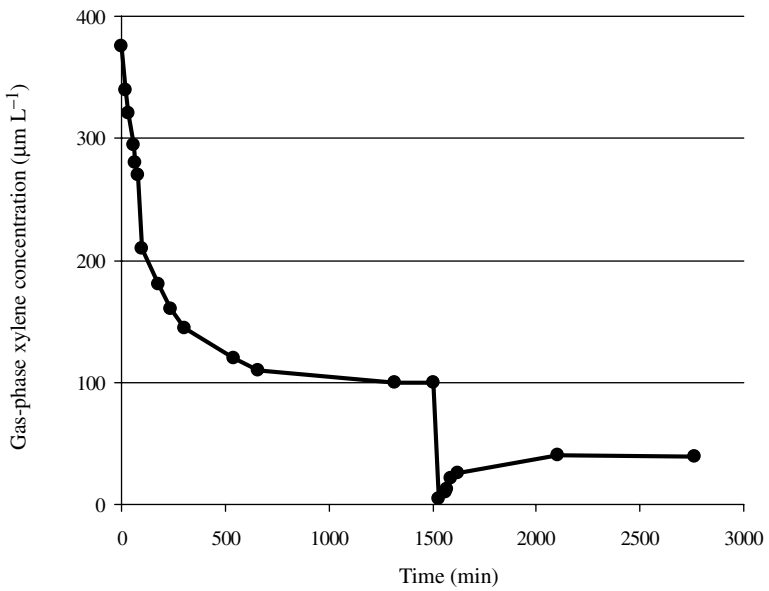


Figure 4.17 Concentrations of xylene measured in its vapor phase in a chamber sealed during adsorption and desorption periods. [Adapted from B. Singer, A Tool to Predict Exposure to Hazardous Air Pollutants, *Environmental Energy Technologies Division News,* 4(4): 5, 2003.]

Sorption is one of the processes that must be considered to account for differences in the temporal pattern of indoor *versus* outdoor concentrations.

Figure 4.18 shows a number of the ways that contaminants can enter and leave an indoor environment. People's activities as they move from one location to another make for unique exposures. For example, people generally spend much more time indoors than outdoors. The simplest quantitative expression of exposure is

$$E = \frac{D}{t} \tag{4.14}$$

where E is the human exposure during the time period t [units of concentration (mass per volume) per time] (mg kg^{-1} day^{-1}), D is the mass of pollutant per body mass (mg kg^{-1}), t is the time (days). Usually, to obtain D, the chemical concentration of a pollutant is measured near the interface of the person and the environment during a specified time period. This measurement is sometimes referred to as the *potential dose* (i.e., the chemical has not yet crossed the boundary into the body, but is present where it may enter the person, such as on the skin, at the mouth, or at the nose).

Exposure is a function of the concentration of the agent and time. It is an expression of the magnitude and duration of the contact. That is, exposure to a contaminant is the concentration of that contact in a medium integrated over the time of contact:

$$E = \int_{t=t_1}^{t=t_2} C(t)\, dt \tag{4.15}$$

where E is the exposure during the time period from t_1 to t_2 and $C(t)$ is the concentration at the interface between the organism and the environment at time t.

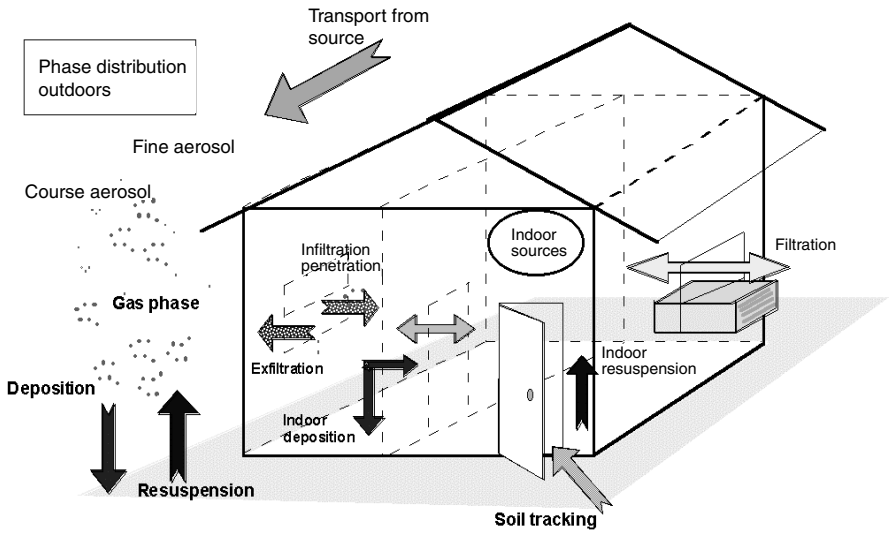


Figure 4.18 The movement and change of a chemical compound (i.e., the mass balance) is a key component of an exposure assessment. (From U.S. Department of Energy, Lawrence Berkeley National Laboratory, http://eetd.lbl.gov/ied/ERA/CalEx/partmatter.html 2003.)

Since the amount of a chemical agent that penetrates from the ambient atmosphere into a building affects the concentration term of the exposure equation, a complete mass balance of the contaminant must be understood and accounted for; otherwise, exposure estimates will be incorrect. The *mass balance* consists of all inputs and outputs as well as chemical changes to the contaminant:

accumulation or loss of contaminant A

$$= \text{mass of A transported in} - \text{mass of A transported out} \pm \text{reactions} \quad (4.16)$$

The reactions may be either those that generate chemical A (i.e., *sources*), or those that destroy chemical A (i.e., *sinks*). Thus, the amount of mass transported in is the *inflow* to the system that includes pollutant discharges, transfer from other control volumes and other media (e.g., if the control volume is soil, the water and air may contribute to the mass of chemical A), and formation of chemical A by abiotic chemistry and biological transformation. Conversely, the *outflow* is the mass transported out of the control volume, which includes uptake by biota, transfer to other compartments (e.g., volatilization to the atmosphere), and abiotic and biological degradation of chemical A. This means that the rate of change of mass in a control volume is equal to the rate of chemical A transported in, minus the rate of chemical A transported out, plus the rate of production from sources, minus the rate of elimination by sinks. Stated as a differential equation, the rate of change for contaminant A is

$$\frac{d[\mathrm{A}]}{dt} = -v\,\frac{d[\mathrm{A}]}{dx} + \frac{d}{dx}\left(\Gamma\,\frac{d[\mathrm{A}]}{dx}\right) + r \quad (4.17)$$

where v is the fluid velocity, Γ is a rate constant specific to the environmental medium, $d[\mathrm{A}]/dx$ is the concentration gradient of chemical A, and r represents the internal sinks and sources within the control volume.

Reactive compounds can be particularly difficult to measure. For example, many volatile organic compounds in the air can be measured by collection in stainless steel canisters and analysis in the lab by chromatography. However, some of these compounds, such as the carbonyls (notably, aldehydes such as formaldehyde and acetaldehyde), are prone to react inside the canister, meaning that by the time the sample is analyzed, a portion of the carbonyls is degraded (underreported). Therefore, other methods are used, such as trapping the compounds with dinitrophenyl hydrazine (DNPH)–treated silica gel tubes that are frozen until being extracted for chromatographic analysis. The purpose of the measurement is to see what is in the air, water, soil, sediment, or biota at the time of sampling, so that any reactions before the analysis gives measurement error.

Remember that the chemical that is released may or may not be what the engineer measures. If the chemical released is reactive, some or all of it may have changed into another form (i.e., *speciated*) by the time it is measured. Even relatively nonreactive compounds may speciate between when the sample is collected (e.g., in a water sample, air canister, soil core, or bag) and when the sample is analyzed. In fact, each contaminant has unique characteristics which vary according to the type of medium in which it exists, and extrinsic conditions such as temperature and pressure. Sample preservation and holding times for anions according to EPA Method 300.1 are shown in Table 4.6. These methods vary according to the contaminant of concern and the environmental

Table 4.6 Preservation and Holding Times for Anion Sampling and Analysis

Analyte	Preservation	Holding time
Part A: Common Anions		
Bromide	None required	28 days
Chloride	None required	28 days
Fluoride	None required	28 days
Nitrate-N	Cool to 4°C	48 hours
Nitrite-N	Cool to 4°C	48 hours
o-Phosphate-P	Cool to 4°C	48 hours
Sulfate	Cool to 4°C	28 days
Part B: Inorganic Disinfection By-products		
Bromate	50 mg L^{-1} EDA	28 days
Bromide	None required	28 days
Chlorate	50 mg L^{-1} EDA	28 days
Chlorite	50 mg L^{-1} EDA, cool to 4°C	14 days

Source: U.S. Environmental Protection Agency, *EPA Method 300.1: Determination of Inorganic Anions in Drinking Water by Ion Chromatography,* Revision 1.0., U.S. EPA, Washington, DC, 1997.

medium from which it is collected, so engineers need to find and follow the correct methods.

The general exposure equation (4.15) is rewritten to address each route of exposure, accounting for chemical concentration and the activities that affect the time of contact. The exposure calculated from these equations is actually the chemical intake (I) in units of concentration (mass per volume or mass per mass) per time, such as mg kg^{-1} day^{-1}:

$$I = \frac{C \cdot CR \cdot EF \cdot ED \cdot AF}{BW \cdot AT} \tag{4.18}$$

where C is the chemical concentration of contaminant (mass per volume), CR is the contact rate (mass per time), EF is the exposure frequency (number of events, dimensionless), and ED is the exposure duration (time). These factors are further specified for each route of exposure, such as the lifetime average daily dose (LADD) as shown in Table 4.7. The LADD is obviously based on chronic, long-term exposure.

Acute and subchronic exposures require different equations, since the exposure duration (ED) is much shorter. For example, instead of LADD, acute exposures to noncarcinogens may use the maximum daily dose (MDD) to calculate exposure. However, even these exposures follow the general model given in equation (4.18).

Table 4.7 Equations for Calculating Lifetime Average Daily Dose (LADD) for Various Routes of Exposure

Route of exposure	Equation: LADD (in mg kg^{-1} day^{-1}) =	Definition
Inhaling aerosols (particulate matter)	$\frac{C \cdot PC \cdot IR \cdot RF \cdot EL \cdot AF \cdot ED \cdot 10^{-6}}{BW \cdot TL}$	C = concentration of the contaminant on the aerosol/particle (mg kg^{-1}) PC = particle concentration in air (g m^{-3}) IR = inhalation rate (m^{-3} h^{-1}) RF = respirable fraction of total particulates (dimensionless, usually determined by aerodynamic diameters, e.g., 2.5 μm) EL = exposure length (h day^{-1}) ED = duration of exposure (days) AF = absorption factor (dimensionless) BW = body weight (kg) TL = typical lifetime (days) 10^{-6} is a conversion factor (kg to mg)
Inhaling vapor-phase contaminants	$\frac{C \cdot IR \cdot EL \cdot AF \cdot ED}{BW \cdot TL}$	C = concentration of the contaminant in the gas phase (mg m^{-3}) Other variables the same as above
Drinking water	$\frac{C \cdot CR \cdot ED \cdot AF}{BW \cdot TL}$	C = concentration of the contaminant in the drinking water (mg L^{-1}) CR= rate of water consumption (L day^{-1}) ED = duration of exposure (days) AF = portion (fraction) of the ingested contaminant that is physiologically absorbed (dimensionless) Other variables the same as above
Contact with soil-borne contaminants	$\frac{C \cdot SA \cdot BF \cdot FC \cdot SDF \cdot ED \cdot 10^{-6}}{BW \cdot TL}$	C = concentration of the contaminant in the soil (mg kg^{-1}) SA = skin surface area exposed (cm^{-2}) BF = bioavailability (percent of contaminant absorbed per day) FC = fraction of total soil from contaminated source (dimensionless) SDF = soil deposition, the mass of soil deposited per unit area of skin surface (mg cm^{-1} day^{-1}) Other variables the same as above

Source: M. Derelanko, Risk Assessment, in *CRC Handbook of Toxicology,* M. J. Derelanko and M. A. Hollinger, (Eds.), CRC Press, Boca Raton, FL, 1999.

Example:

Exposure Calculation

Over an 18-year period, a polymer manufacturer has contaminated the soil on its property with vinyl chloride. The plant closed two years ago but vinyl chloride vapors continue to reach the neighborhood surrounding the plant at an average concentration of 1 mg m^{-3}. Assume that people are breathing at a ventilation rate of 0.5 m^3 h^{-1} (about the average of adult males and females over 18 years of age[52]). The legal settlement allows neighboring residents to evacuate and sell their homes to the company. However, they may also stay. The neighbors have asked for advice on whether to stay or leave, since they have already been exposed for 20 years.

Vinyl chloride is highly volatile, so its phase distribution will be mainly in the gas phase rather than the aerosol phase. Although some of the vinyl chloride may be sorbed to particles, we will use only the vapor-phase LADD equation, since the particle phase is likely to be relatively small. Also, we will assume that outdoor concentrations are the exposure concentrations. This is unlikely, however, since people spend very little time outdoors, so this may provide an additional factor of safety. To determine how much vinyl chloride penetrates living quarters, indoor air studies would have to be conducted. For a scientist to compare exposures, indoor air measurements should be taken.

Find the appropriate equation in Table 4.7 and insert values for each variable. Absorption rates are published by the EPA and the Oak Ridge National Laboratory (http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad). Vinyl chloride is well absorbed, so we can assume that $AF = 1$. We will also assume that a person who stays in the neighborhood is exposed to the average concentration 24 hours a day ($EL = 24$) and that a person lives the remainder of an entire typical lifetime exposed at the measured concentration.

Although the ambient concentrations of vinyl chloride may have been higher when the plant was operating, the only measurements we have are those taken recently. Thus, this is an area of uncertainty that must be discussed with clients. The common default value for a lifetime is 70 years, so we can assume that the longest exposure would be 70 years (25,550 days). Table 4.8 gives some of the commonly used default values in exposure assessments. If the person is now 20 years of age, has already been exposed for that time, and lives a remaining 50 years exposed at 1 mg m^{-3}:

$$\text{LADD} = \frac{C \cdot IR \cdot EL \cdot AF \cdot ED}{BW \cdot TL}$$

$$= \frac{1 \cdot 0.5 \cdot 24 \cdot 1 \cdot 25{,}550}{70 \cdot 25{,}550}$$

$$= 0.2 \text{ mg kg}^{-1} \text{ day}^{-1}$$

If the 20-year-old leaves today, the exposure duration would be for the 20 years that the person lived in the neighborhood. Thus, only the ED term would change: from 25,550 days to 7300 days (i.e., 20 years).

Table 4.8 Commonly Used Human Exposure Factors[a]

Exposure factor	Adult male	Adult female	Child (3–12 years of age)[b]
Body weight (kg)	70	60	15–40
Total fluids ingested (L day^{-1})	2	1.4	1.0
Surface area of skin, without clothing (m^2)	1.8	1.6	0.9
Surface area of skin, wearing clothes (m^2)	0.1–0.3	0.1–0.3	0.05–0.15
Respiration/ventilation rate (L min^{-1})	7.5	6.0	5.0
Resting			
Light activity	20	19	13
Volume of air breathed (m^3 day^{-1})	23	21	15
Typical lifetime (yr)	70	70	N.A.[c]
National upper-bound time (90th percentile) at one residence (yr)	30	30	N.A.
National median time (50th percentile) at one residence (yr)	9	9	N.A.

Source: U.S. Environmental Protection Agency, *Exposure Factor Handbook,* U.S. EPA, Washington, DC, 2003; and Agency for Toxic Substances and Disease Registry, *ATSDR Public Health Assessment Guidance Manual,* ATSDR, Washington, DC, 2003.

[a]These factors are updated periodically by the U.S. EPA in the *Exposure Factors Handbook* at www.epa.gov/ncea/exposfac.htm.

[b]The definition of *child* is highly variable in risk assessment. The *Exposure Factors Handbook* uses these values for children between the ages of 3 and 12 years.

N.A., not applicable.

Therefore, the LADD falls to $\frac{2}{7}$ of its value:

$$\text{LADD} = 0.05 \text{ mg kg}^{-1} \text{ day}^{-1}$$

Once the hazard and exposure calculations are done, we are able to characterize the risk quantitatively. There are two general ways that such risk characterizations are used in environmental problem solving: direct risk assessments and risk-based cleanup standards.

Direct Risk Calculations

In its simplest form, risk is the product of the hazard and the probability of exposure to that hazard, but assumptions can greatly affect risk estimates. For example, cancer risk can be defined as the theoretical probability of contracting cancer when exposed continually for a lifetime (e.g., 70 years) to a given concentration of a substance (carcinogen). The probability is usually calculated as an upper confidence limit. The maximum estimated risk may be presented as the number of chances in a million of contracting cancer.

Two measures of risk are commonly reported. One is the *individual risk,* the probability of a person developing an adverse effect (e.g., cancer) due to the exposure. This is often reported as a *residual* or increased probability above background. For example, if we want to characterize the contribution of all U.S. power plants to increased cancer incidence, the risk above background would be reported. The second way that risk is reported is *population risk,* the annual excess number of cancers in an exposed population. The maximum individual risk might be calculated from exposure estimates based on a *maximum exposed individual* (MEI). The hypothetical MEI lives an entire lifetime outdoors at the point where pollutant concentrations are highest. Assumptions about exposure will greatly affect the risk estimates. For example, the cancer risk from U.S. power plants has been estimated to be 100- to 1000-fold lower for an average exposed person than that calculated for the MEI.[53]

For cancer risk assessments, the hazard is generally assumed to be the slope factor, and the long-term exposure is the lifetime average daily dose:

$$\text{cancer risk} = SF \times \text{LADD} \tag{4.19}$$

Example:

Cancer Risk Calculation

Applying the lifetime average daily dose value from the vinyl chloride exposure calculation earlier, estimate the direct risk to the people living near the abandoned polymer plant. What advice would you give the neighbors?

Insert the calculated LADD values and the vinyl chloride inhalation slope factor of 3.00×10^{-1} from Table 3.5. For the two LADD values under consideration, the cancer risk to the neighborhood exposed for an entire lifetime (exposure duration = 70 years) gives us 0.2 mg kg^{-1} day^{-1} × 0.3 (mg kg^{-1} day^{-1})$^{-1}$ = 0.06. This is an incredibly high risk! The threshold for concern is often 1 in a million (0.000001), but this is a probability of 6%.

Even at the shorter-duration period (20 years of exposure instead of 70 years), the risk is calculated as 0.05 × 0.3 = 0.017, nearly a 2% risk. The combination of a very steep slope factor and very high lifetime exposures leads to a very high risk. Vinyl chloride is a liver carcinogen, so unless corrective actions significantly lower the ambient concentrations of vinyl chloride, the prudent course of action is that the neighbors accept the buyout and leave the area.

Incidentally, vinyl chloride has a relatively high water solubility and can be absorbed to soil particles, so ingestion of drinking water (e.g., people on private wells drawing water from groundwater that has been contaminated) and dermal exposures (e.g., children playing in the soil) are also conceivable. The total risk from a single contaminant such as vinyl chloride is equal to the sum of risks from all pathways (e.g., vinyl chloride in the air, water, and soil):

$$\text{total risk} = \sum \text{risks from all exposure pathways} \tag{4.20}$$

Requirements and measures of success are seldom, if ever, as straightforward as the vinyl chloride example. In fact, the engineer would be ethically remiss if the only advice given is to the local community (i.e., whether or not to accept the buyout). Of course, one of the canons is to be a ''faithful agent'' to the clientele. However, the first engineering canon is to hold paramount the health and safety of the public. Thus, the engineer must balance any proprietary information that the client wants to protect with the need to protect public health. In this case, the engineer must tell the client and prime contractors, for example, that the regulatory agencies need to know that even though the neighbors are moving, a threat continues for others, including future populations. In other words, just because one's clients are taken out of harm's way does not obviate the need for remediation to reduce the vinyl chloride concentrations to acceptable levels.

The risk of adverse outcome other than cancer (''noncancer risk'') is generally called the *hazard quotient* (HQ). It is calculated by dividing the maximum daily dose (MDD) by the acceptable daily intake (ADI):

$$\text{noncancer risk} = \text{HQ} = \frac{\text{MDD}}{\text{ADI}} = \frac{\text{exposure}}{\text{RfD}} \tag{4.21}$$

Note that this is an index, not a probability, so it is really an indication of relative risk. If the noncancer risk is greater than 1, the potential risk may be significant, and if the noncancer risk is less than 1, the noncancer risk may be considered to be insignificant. As shown in equation (4.21), the reference dose, RfD, is one type of ADI.

Example:

Noncancer Risk Calculation

Chromic acid (Cr^{6+}) mist has a dermal chronic RfD of 6.00×10^{-3} mg kg^{-1} day^{-1}. If the actual dermal exposure of people living near a metal processing plant is calculated (e.g., by intake or LADD) to be 4.00×10^{-3} mg kg^{-1} day^{-1}, calculate the hazard quotient for the noncancer risk of chromic acid mist to the neighborhood near the plant and interpret the meanings.

From equation (4.21),

$$\frac{\text{exposure}}{\text{RfD}} = \frac{4.00 \times 10^{-3}}{6.00 \times 10^{-3}} = 0.67$$

Since this is less than 1, one would not expect people chronically exposed at this level to show adverse effects from skin contact. However, at this same chronic exposure (i.e., 4.00×10^{-3} mg kg^{-1} day^{-1}) to hexavalent chromic acid mists via oral route, the RfD is 3.00×10^{-3} mg kg^{-1} day^{-1}, meaning the HQ = 4/3 or 1.3. The value is greater than 1, so we cannot rule out adverse noncancer effects.

If a population is exposed to more than one contaminant, the hazard index (HI) can be used to express the level of cumulative noncancer risk from pollutants 1 through n:

$$\mathrm{HI} = \sum_{1}^{n} \mathrm{HQ} \tag{4.22}$$

The HI is useful in comparing risks at various locations (e.g., benzene risks in St. Louis, Cleveland, and Los Angeles). It can also give the cumulative (additive risk) in a single population exposed to more than one contaminant. For example, if the HQ for benzene is 0.2 (not significant), toluene is 0.5 (not significant), and tetrachloromethane is 0.4 (not significant), the cumulative risk of the three contaminants is 1.1 (potentially significant).

It is desirable to have realistic estimates of the hazard and exposures in such calculations. However, precaution is the watchword for risk. Estimations of both hazard (toxicity) and exposure are often worst-case scenarios, because the risk calculations can have large uncertainties. Models usually assume that effects occur even at very low doses. Human data are usually gathered from epidemiological studies, which no matter how well they are designed, are fraught with error and variability (science must be balanced with the rights and respect of subjects, populations change, activities may be missed, and confounding variables are ever present). Uncertainties exist in every phase of risk assessment, from the quality of data, to limitations and assumptions in models, to natural variability in environments and populations.

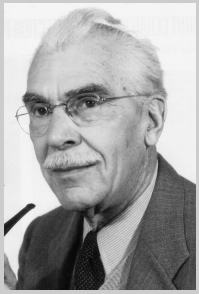
Risk-Based Cleanup Standards

Environmental protection for most of the second half of the twentieth century was based on two types of controls: technology-based and quality-based. Technology-based controls are set according to what is ''achievable'' from the current state of the science and engineering. These are feasibility-based standards. The Clean Air Act has called for best achievable control technologies (BACT), and more recently, for maximally achievable control technologies (MACT). Both standards reflect the reality that even though from an air quality standpoint it would be best to have extremely low levels of pollutants, technologies are not available or are not sufficiently reliable to reach these levels. Requiring unproven or unreliable technologies can even exacerbate the pollution, such as in the early days of wet scrubbers on coal-fired power plants. Theoretically, the removal of sulfur dioxide could be accomplished by venting the power plant flue through a slurry of carbonate, but technology at the time was unproven and unreliable, allowing all-too-frequent releases of untreated emissions while the slurry systems were being repaired. Selecting a new technology over older proven techniques is unwise if the trade-off of the benefit of improved treatment over older methods is outweighed by numerous failures (i.e., no treatment).

Wastewater treatment, groundwater remediation, soil cleaning, sediment reclamation, drinking water supply, air emission controls, and hazardous waste site cleanup all are in part determined by availability and feasibility of control technologies.

Quality-based controls are those that are required to ensure that an environmental resource is in good enough condition to support a particular use. For example, a stream may need to be improved so that people can swim in it and so that it can be a source of water supply. Certain streams may need higher levels of protection than others, such as the so-called ''wild and scenic rivers.'' The parameters will vary but usually include

Biographical Sketch: Earle Phelps



Earle Phelps (1876-1953) graduated from MIT with a degree in chemistry. He was a student of William Sedgwick, who also mentored other notable early sanitary engineers and public health scientists. After graduation, Phelps worked for a while with the Massachusetts Board of Health at the Lawrence Experiment Station and was closely involved in the development of new treatment technology. He moved on first to be a faculty member at MIT and then to the U.S. Public Health Service, where he did his most influential work on stream pollution, authoring the classic text *Stream Pollution,* which for decades was considered the definitive text on the subject.

Phelps was immensely practical. He recognized that pollution would always exist, but the objective would be to reduce the effect to some reasonable level that can be attained economically using available technology. As he stated: ''It is wasteful and therefore inexpedient to require a nearer approach to [the optimal] than is readily obtainable under current engineering practices and at justifiable costs.'' From this reasoned approach to pollution was born the *Principle of Expediency.*

Phelps argued that the objective of regulatory science is to couple the ethics of societal protection with the science of regulation. He defined public health practice as ''the application of the science of preventive medicine, through government, for social ends.''

minimum levels of dissolved oxygen and maximum levels of contaminants. The same goes for air quality, where ambient air quality must be achieved, with the goal that concentrations of contaminants listed as National Ambient Air Quality Standards, as well as certain toxic pollutants, are below levels established to protect health and welfare.

A third type of standard has recently emerged, one based on risk. Although numerous federal agencies were involved, environmental protection in the United States was spearheaded by the U.S. Environmental Protection Agency, created in 1970 and led during its formative years by William Ruckelshaus. After returning for his second term, Ruckelshaus saw the need for ''risk-based'' environmental standards and recognized that such standards would receive public support. Risk-based approaches to environmental protection, especially contaminant target concentrations, are designed to require engineering controls and preventive measures to ensure that risks are not exceeded. The risk-based approach actually embodies elements of both technology-based and quality-based standards. The technology assessment helps determine how realistic it will be to meet certain contaminant concentrations, while the quality of the environment sets the goals and means to achieve cleanup. Engineers are often asked: How clean is clean? When do we know that we have done a sufficient job of cleaning up a spill or hazardous waste site? It is often not possible to have nondetectable concentrations of a pollutant. Commonly, the threshold for cancer risk to a population is 1 in a million excess cancers. However, one

may find that the contaminant is so difficult to remove that we almost give up on dealing with the contamination and put in measures to prevent exposures (i.e., fencing an area in and prohibiting access). This is often done as a first step in remediation but is unsatisfying and controversial (and usually, politically and legally unacceptable). Thus, even if costs are high and technology unreliable, the engineer must find suitable and creative ways to clean up the mess and meet risk-based standards.

Biographical Sketch: William Ruckelshaus

The strength and legitimacy of the U.S. Environmental Protection Agency owes much to the leadership of its first administrator, William D. Ruckelshaus (born 1932). Ruckelshaus is a graduate of Princeton University with a law degree from Harvard. After graduation he was a deputy attorney general in Indiana and was then elected to the Indiana House of Representatives. In 1970 he was asked by President Nixon to head the nascent U.S. EPA. During the EPA's formative years he was able to blend the various federal agencies that oversaw pollution and environmental health into one cohesive structure, took action against the severely polluted cities and industrial polluters, oversaw the setting of health-based standards for both air and water pollution, and developed the first regulations controlling emissions from automobiles (amid general anguish from the automobile and petroleum industries, which claimed that it could not be done). He worked with the states to develop both water quality standards and ambient air quality plans, and he worked to ban the use of some pesticides, such as DDT. Almost all of the environmental legislation we presently enjoy in the United States was guided through the Congress during the years William Ruckelshaus was head of the U.S. EPA.

In 1973 he stepped down from the directorship to become at first the acting director of the FBI and then briefly as deputy attorney general in the Justice Department. He distinguished himself in this post by refusing to fire the special prosecutor investigating the Watergate break-in: instead, resigning his post.

Following the disastrous tenure of Ann Gorsuch as the administrator of the U.S. EPA during the first Reagan administration, in which she was apparently charged with scuttling the agency (a popular move to numerous politicians at the time), William Ruckelshaus was once again asked to take over. He worked to rekindle both the work and the morale of the agency employees and developed widely accepted principles of risk-based decision-making in environmental controls. In his first all-hands speech, that was piped into the EPA offices around the nation, he received great applause when he ensured the employees that the agency would uphold the law and work toward its mission to protect the environment. His work in restoring and protecting water quality in the Chesapeake Bay, in developing processes for cleaning up hazardous waste sites, and in the banning of many chlorinated pesticides were significant accomplishments during his second tenure as the chief of the U.S. EPA.

Risk-based target concentrations can be calculated by solving for the target contaminant concentration in the exposure and risk equations. Since risk is the hazard (e.g., slope factor) times the exposure (e.g., LADD), a cancer risk–based cleanup standard can be found by enumerating the exposure equation (4.18) within the risk equation (in this instance, the drinking water equation from Table 4.7) gives

$$\text{risk} = \frac{C \cdot CR \cdot EF \cdot ED \cdot AF \cdot SF}{BW \cdot AT} \tag{4.23}$$

and solving for C, we have

$$C = \frac{\text{risk} \cdot BW \cdot AT}{CR \cdot EF \cdot ED \cdot AF \cdot SF} \tag{4.24}$$

This is the target concentration for each contaminant needed to protect the population from the specified risk (e.g., 10^{-6}). In other words, this is the concentration that must not be exceeded to protect a population having an average body weight and over a specified averaging time from an exposure of certain duration and frequency that leads to a risk of 1 in a million. Although 1-in-a-million added risk is a commonly used benchmark, cleanup may not always be required to achieve this level. For example, if a site is considered to be a ''removal'' action (i.e., the principal objective is to get rid of a sufficient amount of contaminated soil to reduce possible exposures), the risk reduction target may be as high as one additional cancer per 10,000 (i.e., 10^{-4}). This is an area of risk management, which is discussed in greater detail in Chapter 5.

Example:

Risk-Based Contaminant Cleanup

A well is the principal water supply for the town of Apple Chill. A study has found that the well contains 80 mg L^{-1} tetrachloromethane (CCl_4). Assuming that the average adult in the town drinks 2 L day^{-1} of water from the well and lives in the town for an entire lifetime, what is the lifetime cancer risk to the population if no treatment is added? What concentration is needed to ensure that the population cancer risk is below 10^{-6}?

The lifetime cancer risk added to Apple Chill's population can be estimated using the LADD and slope factor for CCl_4. In addition to the assumptions given, we will use default values from Table 4.8. We will also assume that people live in the town for their entire lifetimes and that their exposure duration is equal to their typical lifetime. Thus, the *ED* and *TL* terms cancel, leaving the abbreviated

$$\text{LADD} = \frac{C \cdot CR \cdot AF}{BW}$$

Since we have not specified male or female adults, we will use the average body weight, assuming that there are about the same number of males as females. We look up the absorption factor for CCl_4 and find that it is 0.85, so the adult lifetime exposure is

$$\text{LADD} = \frac{80 \cdot 2 \cdot 0.85}{65} = 4.2 \text{ mg kg}^{-1} \text{ day}^{-1}$$

Using the midpoint value between the default values [(15 + 40)/2 = 27.5 kg] for body weight and default CR values (1 L day^{-1}), the lifetime exposure for children is

$$\text{LADD} = \frac{80 \cdot 1 \cdot 0.85}{27.5} = 2.5 \text{ mg kg}^{-1} \text{ day}^{-1}$$

for the first 13 years, and the adult exposure of 4.2 mg kg^{-1} day^{-1} thereafter. The oral SF for CCl_4 is 1.30×10^{-1} kg day^{-1}, so the added adult lifetime risk from drinking the water is

$$4.2 \times (1.30 \times 10^{-1}) = 5.5 \times 10^{-1}$$

and the added risk to children is

$$2.5 \times (1.30 \times 10^{-1}) = 3.3 \times 10^{-1}$$

However, for children, environmental and public health agencies recommend an additional factor of safety beyond what would be used to calculate risks for adults. This is known as the *10× rule*: that is, children need to be protected 10 times more than adults because they are more vulnerable, have longer life expectancies (so latency periods for cancer need to be accounted for), and their tissue is developing prolifically and changing. So in this case, with the added risk, our reported risk would be 3.3. Although this is statistically impossible (i.e., one cannot have a probability greater than 1 because it would mean that the outcome is more than 100% likely, which of course is impossible!) However, what this tells us is that the combination of a very high slope of the dose–response curve and a very high LADD leads to much needed protection, and removal of either the contaminants from the water or the provision of a new water supply. The city engineer or health department should mandate bottled water immediately.

The cleanup of the water supply to achieve risks below 1 in a million can also be calculated from the same information and reordering the risk equation to solve for C:

$$\text{risk} = \text{LADD} \times SF$$

$$\text{risk} = \frac{C \cdot CR \cdot AF \cdot SF}{BW}$$

$$C = \frac{BW}{CR \cdot AF \cdot SF \cdot \text{risk}}$$

Based on adult LADD, the well water must be treated so that the tetrachloromethane concentrations are below

$$C = \frac{65 \cdot 10^{-6}}{2 \cdot 0.85 \cdot 0.13} = 2.9 \times 10^{-4} \text{ mg L}^{-1} = 290 \text{ ng L}^{-1}$$

Based on the children's LADD, and the additional 10×, the well water must be treated so that the tetrachloromethane concentrations are below

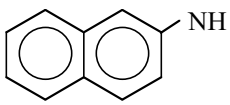
$$C = \frac{27.5 \cdot 10^{-7}}{1 \cdot 0.85 \cdot 0.13} = 2.5 \times 10^{-5} \text{ mg L}^{-1} = 25 \text{ ng L}^{-1}$$

The town will have to remove the contaminant, so that the concentration of CCl_4 in the finished water must be treated to a level six orders of magnitude less than the untreated well water (i.e., lowered from 80 mg L^{-1} to 25 ng L^{-1}).

Cleanup standards are part of the arsenal needed to manage risk. However, other considerations needed to be given to a contaminated site, such as how to monitor progress in lowering pollutant levels and how to ensure that the community stays engaged and is participating in the cleanup actions, where appropriate. Even when the engineering solutions are working well, the engineer must allot sufficient time and effort to these other activities; otherwise, skepticism and distrust can arise.

CASE STUDY: THE DRAKE CHEMICAL COMPANY SUPERFUND SITE[54]

One of the downsides of industrial development is a legacy of harmful chemicals. The Drake Chemical Company of Lock Haven, Pennsylvania, was a major producer of chemicals during World War II and continued to provide employment opportunities to the economically depressed town after the war. Among the chemicals that the company disposed of in an open pit was β-naphthylamine (also known as 2-naphthylamine), a compound used as a dye.



β-naphthylamine

Unfortunately, β-naphthylamine is also a potent carcinogen (inhalation and oral cancer slope factor = 1.8),[55] having been found to be a known human carcinogen based on sufficient evidence of carcinogenicity in humans. Epidemiological studies have shown that occupational exposure to β-naphthylamine alone or when present as an impurity in other compounds is causally associated with bladder cancer in workers.[56]

In 1962, the state of Pennsylvania banned the production of this chemical, but the damage to the groundwater had already been done with the disposal of β-naphthylamine into the uncontrolled pit. An order from the state caused Drake to stop manufacturing β-naphthylamine, but the company continued to produce other chemicals, seemingly without much concern for the environment or the health of the people in Lock Haven. Finally, in 1981, the U.S. EPA closed the company site and took control of the property.

Cleanup crews discovered several unlined lagoons and hundreds of often unmarked barrels of chemicals stored in makeshift buildings. After removing the drums and draining the lagoons, the crews discovered that the β-naphthylamine had seeped into nearby property and into creeks, creating a serious health hazard. The EPA's attempts to clean the soil and the water were, however, met with public opposition. Much of the public blamed the EPA for forcing Drake Chemical, a major local employer, to close the plant. In addition, the best way to treat the contaminated soil was to burn it in an incinerator, and the EPA made plans to bring in a portable combustion unit. The public, not at all happy with EPA being there in the first place, became concerned with the emissions from the incinerator. After many studies and the involvement of the U.S. Army Corps of Engineers, the incinerator was finally allowed to burn the soil, which after treatment was spread out and covered with 3.5 feet of topsoil. The groundwater was pumped and treated, and this continued until the levels of β-naphthylamine reached background concentrations. The project was not completed until 1999. Ironicaly, part of the cleanup cost included the EPA paying the legal fees of the lawyers who argued against the cleanup.

Some general principles have been adopted almost universally by regulatory agencies, especially those concerned with cancer risks from environmental exposures (see Table 4.9).

Zero risk can occur only when either the hazard (e.g., toxicity) does not exist or the exposure to the hazard is zero. A substance found to be associated with cancers based on animal testing or observations of human populations can be further characterized to improve the certainty of linking exposure to cancer. Association of two factors, such as the level of exposure to a compound and the occurrence of a disease, does not necessarily mean that one necessarily ''causes'' the other. Often, after study, a third variable explains the relationship. However, it is important for science to do what it can to link causes with effects. Otherwise, corrective and preventive actions cannot be identified. So strength of association is a beginning step toward cause and effect (see the biographical sketch of Sir Bradford Hill later in the chapter). A major consideration in the strength of association is the application of sound technical judgment of the weight of evidence. For example, characterizing the weight of evidence for carcinogenicity in humans consists of three major steps:[57]

1. Characterization of the evidence from human studies and from animal studies individually
2. Combination of the characterizations of these two types of data to show the overall weight of evidence for human carcinogenicity
3. Evaluation of all supporting information to determine if the overall weight of evidence should be changed

Note that none of these steps is absolutely certain.

Students are rightfully warned in their introductory statistics courses not to confuse association with causality. One can have some very strong statistical associations that are not causal. For example, if one were to observe ice cream eating in Kansas City and

Table 4.9 General Principles Applied to Health and Environmental Risk Assessments in the United States

Principle	Explanation
Human data are preferable to animal data.	For purposes of hazard identification and dose–response evaluation, epidemiological and other human data better predict health effects than do animal models.
Animal data can be used in lieu of sufficient, meaningful human data.	Although epidemiological data are preferred, agencies are allowed to extrapolate hazards and to generate dose–response curves from animal models.
Animal studies can be used as a basis for risk assessment.	Risk assessments can be based on data from the most highly sensitive animal studies.
The route of exposure in animal study should be analogous to human routes.	Animal studies are best if based on the same route of exposure as in humans (e.g., inhalation, dermal, or ingestion routes). For example, if an air pollutant is being studied in rats, inhalation is a better indicator of effect than if the rats are dosed on the skin or if the exposure is dietary.
A threshold is assumed for noncarcinogens.	For noncancer effects (e.g., neurotoxicity, endocrine dysfunction, and immunosuppression), there is assumed to be a safe level under which no effect would occur [e.g., no observed adverse effect level (NOAEL), which is preferred, but also lowest observed adverse effect level (LOAEL)].
The threshold is calculated as a reference dose or reference concentration (air).	Reference dose (RfD) or concentration (RfC) is the quotient of the threshold (NOAEL) divided by factors of safety (uncertainty factors and modifying factors; each usually multiples of 10): $$\text{RfD} = \frac{\text{NOAEL}}{\text{UF} \times \text{MF}}$$
Sources of uncertainty must be identified.	Uncertainty factors (UFs) address: • Interindividual variability in testing • Interspecies extrapolation • LOAEL-to-NOAEL extrapolation • Subchronic-to-chronic extrapolation • Route-to-route extrapolation • Data quality (precision, accuracy, completeness, and representativeness) Modifying factors (MFs) address uncertainties that are less explicit than the UFs.
Factors of safety can be generalized.	The uncertainty and modifying factors should follow certain protocols: e.g., 10 = for extrapolation from a sensitive individual to a population; 10 = rat-to-human extrapolation, 10 = subchronic-to-chronic data extrapolation), and 10 = LOAEL used instead of NOAEL.
No threshold is assumed for carcinogens.	No safe level of exposure is assumed for cancer-causing agents.
Precautionary principle is applied to the cancer model.	A linear, no-threshold dose–response model is used to estimate cancer effects at low doses [i.e., to draw the unknown part of the dose–response curve from the region of observation (where data are available) to the region of extrapolation].
Precautionary principle is applied to cancer exposure assessment.	The most highly exposed person is generally used in the risk assessment (upper-bound exposure assumptions). Agencies are reconsidering this worst-case policy and considering more realistic exposure scenarios.

Source: U.S. Environmental Protection Agency, *General Principles for Performing Aggregate Exposure and Risk Assessment,* Office of Pesticides Programs, U.S. EPA, Washington, DC, 2001.

counted the number of people wearing shorts, one would find a strong association between shorts-wearing and ice cream–eating. Does wearing shorts cause more people to eat more ice cream? In fact, both findings are caused by a third variable, ambient temperature. Hotter temperatures drive more people to wear shorts *and* to eat more ice cream.

People have a keen sense of observation, especially when it has to do with the health and safety of their families and neighborhoods. They can ''put 2 and 2 together.'' Sometimes, it seems to them that we engineers are telling them that 2 + 2 does *not* equal 4. That cluster of cancers in town may have nothing to do with the green gunk that is flowing out of the abandoned building's outfall. But in their minds, the linkage is obvious.

The challenge is to present information in a meaningful way without violating or overextending the interpretation of the data. If we assign causality when none really exists, we may suggest erroneous solutions. But if all we can say is that the variables are associated, the public is going to want to know more about what may be contributing an adverse affect (e.g., learning disabilities and blood lead levels). This was particularly problematic in early cancer research. Possible causes of cancer were being explored and major research efforts were being directed at myriad physical, chemical, and biological agents. So there needed to be some manner of sorting through findings to see what might be causal and what is more likely to be spurious results. Sir Austin Bradford Hill is credited with articulating key criteria that need to be satisfied to attribute cause and effect in medical research.[58] His recommended factors to be considered in determining whether exposure to an agent elicits an effect are as follows:

Criterion 1: Strength of association. For an exposure to an agent to cause an effect, the exposure must be associated with that effect. Strong associations provide more certain evidence of causality than is provided by weak associations. Common epidemiological metrics used in associations include risk ratio, odds ratio, and standardized mortality ratio.

Criterion 2: Consistency. If the exposure is associated with an effect consistently under different studies using diverse methods of study of assorted populations under varying circumstances by different investigators, the link to causality is stronger. For example, if carcinogenic effects of chemical X are found in mutagenicity studies, mouse and Rhesus monkey experiments, and human epidemiological studies, there is greater consistency between chemical X and cancer than if only one of these studies showed the effect. Consistency is one of the important factors in risk models. For example, if the animal and human data do not agree, an increased uncertainty factor is added to the reference dose (RfD).

Criterion 3: Specificity. The specificity criterion holds that the cause should lead to only one disease and that the disease should result from this single cause only. This criterion appears to be based in the germ theory of microbiology, where a specific strain of bacteria and viruses elicits a specific disease. This is rarely the case in studying most chronic environmental diseases, since a chemical can be associated with cancers in numerous organs, and the same chemical may elicit cancer, hormonal, immunological, and neural dysfunctions.

Criterion 4: Temporality. Timing of exposure is critical to causality. This criterion requires that exposure to the chemical must precede the effect. For example, in a retrospective study, the researcher must be certain that the manifestation of a disease was not

already present before exposure to the chemical. If the disease were present prior to the exposure, it may not mean that the chemical in question is not a cause, but it does mean that it is not the sole cause of the disease (see criterion 3 above). This can be challenging, for example, for diseases with extended latency periods and large sub-clinical periods before being diagnosed.

Criterion 5: Biologic gradient. This is another essential criterion for environmental risks. Gradient is a familiar concept to engineers (e.g., it is central to Fick's law concentration change with distance). In risk assessment, the biological gradient is known as the *dose–response step* in risk assessment. If the level, intensity, duration, or total level of chemical exposure is increased a concomitant, progressive increase should occur in the toxic effect.

Criterion 6: Plausibility. Generally, an association needs to follow a well-defined explanation based on a known biological system. However, ''paradigm shifts'' in the understanding of key scientific concepts do occur. A noteworthy example is the change in the latter part of the twentieth century in the understanding of how the endocrine, immune, and neural systems function: from the view that these are exclusive systems, to today's perspective that in many ways they constitute an integrated chemical and electrical set of signals in an organism. For example, Candace Pert, a pioneer in endorphin research, has espoused the concept of mind/body, with all the systems interconnected, rather than separate and independent systems.

Criterion 7: Coherence. The criterion of coherence suggests that all available evidence concerning the natural history and biology of a disease should ''stick together'' (cohere) to form a cohesive whole. By that, the proposed causal relationship should not conflict or contradict information from experimental, laboratory, epidemiologic, theory, or other knowledge sources. For some time, for example, human studies linked arsenic exposure to cancer, but these were not replicated in animal studies. Eventually the animal studies also showed the linkage, but until then, it was a quandary for risk assessors. More often, animal studies first show the link and it is the human data that are more uncertain. See Criterion 2 (Consistency).

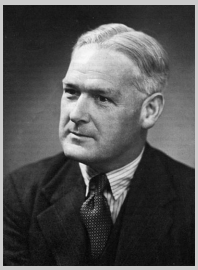
Criterion 8: Experimentation. Experimental evidence in support of a causal hypothesis may come in the form of community and clinical trials, *in vitro* laboratory experiments, animal models, and natural experiments.

Criterion 9: Analogy. The term *analogy* implies a similarity in some respects among things that are otherwise different. It is thus considered one of the weaker forms of evidence.

In assessing and managing environmental risks, some of Hill's criteria are more important than others. Risk assessments rely heavily on strength of association (e.g., to establish dose–response relationships). Coherence is also very important. Animal and human data should be extensions of one another and should not disagree. Biological gradient is crucial, since this is the basis for the dose–response relationship (the more dose, the greater the biological response).

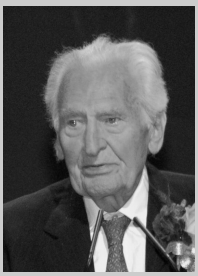
Temporality is crucial to all scientific research (i.e., the cause must precede the effect). However, this is sometimes difficult to see in some instances, such as when the exposures to suspected agents have been continuous for decades and the health data are only recently available.

Biographical Sketch: Sir Bradford Hill



In 1965, Austin Bradford Hill (1897–1991) published his famous paper, ''The Environment and Disease: Association or Causation?'' which included the nine guidelines for establishing the relationship between environmental exposure and effect.[59] Hill meant for the guidelines to be just that—guidelines, not an absolute test for causality. A situation does not have to meet Hill's nine criteria to be shown to be causally related. In the introduction to his paper, Hill acknowledges this by suggesting that there will be circumstances where not all of the nine criteria need to be met before action is taken. He recommended that action may need to be taken when the circumstances warrant. In his opinion, in some cases ''the whole chain may have to be unraveled'' or in other situations ''a few links may suffice.'' The case of the 1853 cholera epidemic in London, concluded by John Snow to be waterborne and controlled by the removal of the pump handle, is a classic example in which only a few links were understood.

Biographical Sketch: Sir William Richard Shoboe Doll



Richard Doll (1912–2005) graduated from medical school and then served as a physician in the Royal Army Medical Corps during World War II. After the war he returned to England to conduct epidemiological research, concentrating on the relationship between radiation and leukemia and the effect of stress on the formation of peptic ulcers.

In 1950, Bradford Hill and Richard Doll initiated a study on the environmental cause of lung cancer, using the then-held hypothesis that automobile exhaust was the causative agent. They soon discovered, through statistical evaluations of large-scale trials, that the only positive correlation existed between cigarette smoking and lung cancer. This was the first time that an unequivocal connection had been made between cigarette smoking and lung cancer. All subsequent studies in this field have been based on this groundbreaking work. Their famous article in the *British Medical Journal* concluded ''The risk of developing the disease increases in proportion to the amount smoked. It may be 50 times as great among those who smoke 25 or more cigarettes a day as among non-smokers.''

Sir Richard Doll was knighted by Queen Elizabeth for his outstanding contribution to the field of epidemiology.

The key is that sound engineering and scientific judgment, based on the best available and most reliable data, should always be used when estimating risk. Linking cause and effect is often difficult in environmental matters. The best we can do is to be upfront and clear about the uncertainties and the approaches we use.

Environmental risk by nature addresses probable impossibilities. From a statistical perspective, it is extremely likely that cancer will not be eliminated during our lifetimes. But the efforts to date have shown great progress toward reducing risks from several forms of cancer. This risk reduction can be attributed to a number of factors, including changes in behavior (smoking cessation, dietary changes, and improved lifestyles), source controls (fewer environmental releases of cancer-causing agents), and the reformulation of products (substitution of chemicals in manufacturing processes).

RISK ASSESSMENT: MERELY THE FIRST STEP

We have covered a wide array of elements needed to access environmental risks. These are crucial because engineers must first understand the science before being able to intervene to make things better. These elements must be pulled together. *Risk characterization* is the stage where the engineer summarizes the necessary assumptions, describes the scientific uncertainties, and determines the strengths and limitations of the analyses. The risks begin to be understood by integrating the analytical results, interpreting adverse outcomes, and describing the uncertainties and weights of evidence. This can be very important for many minority communities, because much of their culture and livelihood

Biographical Sketch: Daniel A. Okun

Dan Okun (born 1917) has viewed his career as a mission—a mission to help others live longer and healthier lives through better sanitation and public health. His efforts have been focused in three directions: research, education, and outreach. In research, he was one of the principal developers of the original oxygen probe, a galvanic device that made measurement of dissolved oxygen in the field practical and convenient. In education, he led the development of an outstanding environmental engineering and science program at the University of North Carolina, recognizing early on the value of interaction among air pollution control, industrial hygiene, epidemiology, radiological health, hazardous waste management, and what was then known as sanitary engineering. But his greatest contribution was in outreach efforts, particularly in Central America and in the Pacific. He spearheaded the development of many environmental engineering programs at such universities as San Paulo in Guatemala, and brought many Central American and South American engineers to UNC to study alongside students from the United States. His legacy as an innovator and mentor will be felt for a very long time in the New World.

is linked directly to ecosystems, such as Native American subsistence agriculture, silviculture, and fishing, African American communities in or near riparian and littoral habitats, and Hispanic American families exposure to agricultural chemicals.

A reliable risk assessment is the groundwork for determining whether risks are disproportionate in a given neighborhood or region; as such, it is a first step in achieving environmental justice. Exposures to hazards can be disproportionate, which leads to disproportionate risk. There are also situations where certain groups of people are more sensitive to the effects of pollutants. Such things are difficult to quantify, but need to be addressed, as we discuss in Chapter 5. Risk assessment is a process distinct from risk management, where actions are taken to address and reduce the risks. But the two are deeply interrelated and require continuous feedback with each other. Engineers are key players in both efforts. In addition, risk communication between the engineer and the client further complicate the implementation of the risk assessment and management processes. What really sets risk assessment apart from the actual management and policy decisions is that the risk assessment must follow the prototypical rigors of scientific investigation and interpretation that we outlined in this chapter. As we see in the next chapters, risk management draws upon the technical risk assessment, but must also factor in other social considerations. The challenge is to maintain the rigors of science and engineering *and* incorporate the societal needs of the community.

REFERENCES AND NOTES

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5. This discussion and examples are based on United Nations Environmental Program, International Labor Organisation, International Programme on Chemical Safety, Environmental Health Criteria 214, *Human Exposure Assessment,* UNEP Geneva, 2000.
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8. K. Smith, Hazards in the Environment, in *Environmental Hazards: Assessing Risk and Reducing Disaster,* Routledge, London, 1992.
9. This calls to mind Jesus's parable of the lost sheep (Matthew 18). In the story, the shepherd leaves (abandons, really) 99 sheep to find a single lost sheep. Some might say that if we as professionals behaved like that shepherd, we would be acting irresponsibly. However, it is actually how most of us act. We must give our full attention to one patient or client at a time.

There are a number of ways to interpret the parable, but one is that there is an individual value to each member of society, and the value of society's members is not mathematically divisible. In other words, a person in a population of 1 million is not one-millionth of the population's value. The individual value is not predicated on the group's value. This can be a difficult concept for those of us who are analytical by nature, but it is important to keep in mind when estimating risk.

10. See, for example, D. Dockery, Epidemiologic Evidence of Cardiovascular Effects of Particulate Air Pollution, *Environmental Health Perspectives,* 109(Supplement), 2001, pp. 483–486.
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25. This goes well beyond mere translation. For example, it is very common in emergency departments of hospitals to hear the physician or nurse conversing in Spanish themselves or via a translator with a patient or family member. Spanish has many dialects and idioms that, if translated incorrectly, can lead to a miscommunication of symptoms and risks. Other tools are used, such as graphical devices (e.g., amount of pain ranging from 0 to 10 is shown as a smiling face and an obviously painful grimace, respectively, or the need for preventing the spread of germs with cartoons of people wearing masks and lathering soap). One cannot help concluding, however, that many of the more subtle and complex medical information is lost on the subjects and their attending medical staffs. For example, does nodding by a person

from Japan mean the same as that of a person from Ecuador or Mozambique (or East LA *versus* Beverly Hills, for that matter)?

26. Typically, researchers withheld treatment to observe the course of the disease (an excruciatingly painful process).
27. J. K. Hammitt, E. S. Belsky, J. I. Levy, and J. D. Graham, Residential Building Codes, Affordability, and Health Protection: A Risk–Tradeoff Approach, *Risk Analysis,* 19(6):1037–1058, 1999.
28. At least on its face, this runs contrary to some of the measures calling for improved environmental risk-based science proposed in the 1980s, especially the separation of risk assessment and risk management. This advice actually was an attempt to make risk science more objective and empirical, so that the science does not become ''contaminated'' by vested interests in the findings (such as political and economic considerations).
29. The phenomenon where children eat such nonfood material as paint chips is known as *pica.* For young children, especially those in poorer homes with older residences, this type of ingestion was (and still is, in some places) a major lead exposure pathway in children. Other pathways include soil ingestion (also pica), inhalation of lead on dust particles (which can be very high when older homes are renovated), and lead in food [such as lead leaching into food from glazes on cooking and dining ware, common in some ethnic groups (e.g., Mexican, Mexican American).]
30. Hydraulics and hydrology provide very interesting case studies in the failure domains and ranges, particularly how absolute and universal measures of success and failure are almost impossible. For example, a levee or dam breach, such as the recent catastrophic failures in New Orleans during and in the wake of Hurricane Katrina, experienced failure when flow rates reached cubic meters per second. Conversely, a hazardous waste landfill failure may be reached when flow across a barrier exceeds a few cubic centimeters per decade.
31. U.S. Environmental Protection Agency, *Guidelines for Carcinogen Risk Assessment,* EPA/630/R-00/004, *Federal Register* 51(185):33992–34003, U.S. EPA, Washington, DC, 1986; and R. I. Larsen, An Air Quality Data Analysis System for Interrelating Effects, Standards, and Needed Source Reductions, Part 13: Applying the EPA *Proposed Guidelines for Carcinogen Risk Assessment* to a Set of Asbestos Lung Cancer Mortality Data, *Journal of the Air and Waste Management Association,* 53:1326–1339, 2003.
32. J. Duffus and H. Worth, Training program: The Science of Chemical Safety: Essential Toxicology, Part 4: Hazard and Risk, *IUPAC Educators' Resource Material,* International Union of Pure and Applied Chemistry, Research Triangle Park, NC, 2001.
33. Actually, another curve could be shown for essential compounds such as vitamins and certain metallic compounds. In such a curve, the left-hand side (low dose or low exposure) of the curve would represent deficiency and the right-hand side (high dose or exposure) would represent toxicity, with an optimal, healthy range between these two adverse responses (see Fig. 4.19). Note that the two responses will differ at the low and high doses. For example, anemia and its related effects may occur at the low end, with neurotoxicity at the high end of exposures. Ideally, the optimal range has neither effect. Like the other curves, the safe levels of both effects would be calculated and appropriate factors of safety applied.
34. For an excellent summary of the theory and practical applications of the Ames test, see K. Mortelmans and E. Zeiger, The Ames *Salmonella*/Microsome Mutagenicity Assay, *Mutation Research,* 455:29–60, 2000.
35. The source for much of the technical information in the rest of the chapter is D. A. Vallero, *Paradigms Lost: Learning from Environmental Mistakes, Mishaps, and Misdeeds,* Butterworth-Heinemann, Burlington, MA, 2006.
36. The source for the physicochemical properties of DDT and its metabolites is United Nations Environmental Programme, Chemicals: North American Regional Report, Regionally Based

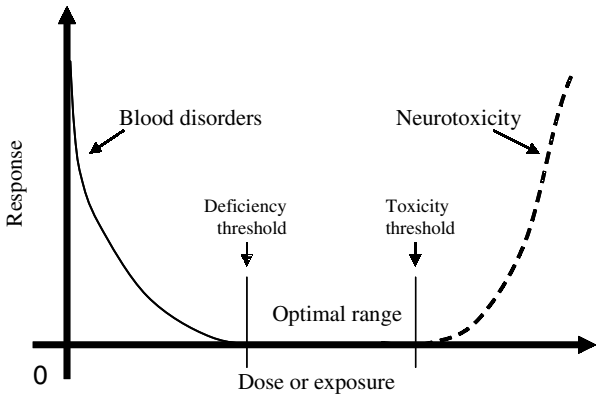

Figure 4.19 Hypothetical dose–response curves for essential substances.

Assessment of Persistent Toxic Substances, Global Environment Facility, UNEP, New York, 2002.

37. The two principal isomers of DDD are *p,p′*-2,2-bis(4-chlorophenyl)-1,1-dichloroethane and *o,p′*-1-(2-chlorophenyl)-1-(4-chlorophenyl)-2,2-dichloroethane. The principal isomer of DDE is *p,p′*-1,1′-(2,2-dichloroethenylidene)-bis[4-chlorobenzene].
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