is generally quite a complex phenomenon; it can be very approximately described by the concept of "biological half-life"—the time it takes for half the material to be eliminated from the body.

When estimating doses from environmental radioactivity, direct measurements are almost never available for the amounts of particular radionuclides in the body. Complex computer models have to be used, often with large numbers of parameters and associated uncertainties. This is especially true of dose estimation for off-site populations where there are no direct measurements for dose or for body-burdens of radioactive materials. However, radionuclides in food, water, and air can be measured. If done carefully, such measurements can provide a basis for estimating doses. If internal burdens are large, techniques such as whole-body counting (called *in vivo* measurements) and urine sampling can also be used.

Health Risks of Ionizing Radiation

Ionizing radiation can cause stochastic (random) and deterministic (or nonstochastic) effects. Deterministic effects appear if a minimum radiation dose is exceeded. Above that threshold, the effects are readily observed in most or all exposed people and the severity increases with dose. The occurrence and severity of a deterministic effect in any one individual are reasonably predictable. A radiation burn is an example of a deterministic effect.

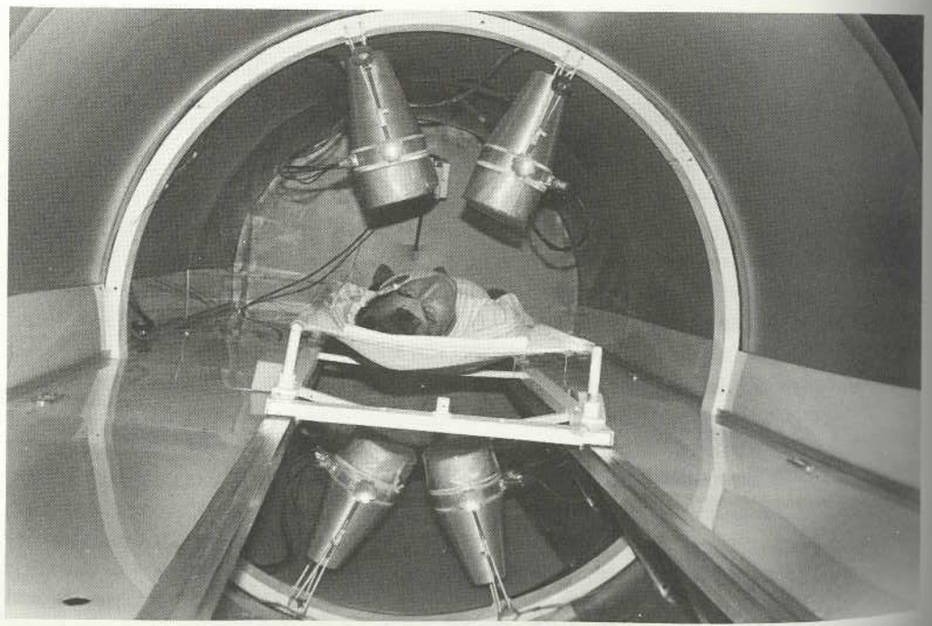

Figure 4.2 The strontium-90 whole-body counter, Chelyabinsk. Photo by Robert del Tredici.

In adults, nonstochastic effects dominate when the dose to the entire body is more than about one sievert. An exception is temporary sterility in the male, which can occur with a single absorbed dose to the testis of about 0.15 grays.[6] With respect to children, the threshold for congenital malformations and other developmental abnormalities has been estimated to be 0.25 grays of radiation exposure up to 28 days of gestation.

Single radiation doses over about 1 gray cause radiation sickness; acute effects include nausea, vomiting, and diarrhea, sometimes accompanied by malaise, fever, and hemorrhage. The victim may die in a few hours, days, or weeks. Other acute effects can include sterility and radiation burns, depending on the absorbed dose and the rate of the exposure. The dose at which half the exposed population would die in sixty days without medical treatment is called the LD50 dose (LD for lethal dose, and 50 for 50 percent). It is about 4 sieverts for adults. The sixty-day period is sometimes explicitly identified, and the dose is then called the LD50/60 dose. In general, a number of different LD50 doses can be specified, depending on the number of days, T, after which the observations of death are cut off.

For radiation doses less than about 1 sievert, stochastic effects have been the greatest concern. The most important stochastic effects, cancer and inheritable genetic damage, may appear many years or decades after exposure. It is thought that there is no minimum threshold for these effects; as dose decreases the effects are still expected to occur, but with lower frequency. However, the uncertainties at low doses (10 millisieverts or less) are very large. Estimates of the magnitude of low-dose radiation effects have tended to rise over the years, but remain the subject of controversy.

Because ionizing radiation can damage the genetic material of virtually any cell, cancer can occur in many sites or tissues of the body. The actual effect depends in part on the route of exposure. For example, external radiation, such as X rays or gamma radiation, can affect DNA in blood-forming cells or in many organs in ways that cause cancers of these organs decades later. It should be noted that tissues vary in their sensitivity to radiation damage. For instance, muscles are less sensitive than bone marrow.

There are many pathways by which the body can be exposed to internal irradiation. Decay products of radon, which are present in an underground uranium mine, may be inhaled by miners and end up in their lungs. Particles of plutonium-239 or other actinides, which emit mostly high-LET alpha particles, may be inhaled and deposited on the epithelial lining of bronchi in the lung. A radiation dose from such

6. ICRP 1991, p. 15.

exposure pathways increases the risk of lung cancer. In addition, soluble particles may be absorbed and distributed through the blood or lymph systems to other parts of the body. Some elements, such as radium, strontium, or iodine, tend to accumulate in certain organs. For example, iodine-131 delivers its principal ionizing radiation dose to the thyroid gland, making that the most likely site of a resultant cancer. Iodine-131 is also used to combat thyroid cancer, since the emitted radiation destroys the cancerous cells along with healthy ones. But when there is no disease in the thyroid, the radiation affects only healthy cells.

Estimating the Risk of Cancer from Ionizing Radiation

Various institutions have estimated the risk of cancer following exposures to ionizing radiation, particularly the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), the U.S. National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation (BEIR), and the International Commission on Radiological Protection (ICRP). These estimates are derived mainly from studies of the survivors of the Hiroshima and Nagasaki bombings, and also from various groups of people given radiation for therapeutic and diagnostic purposes or who have been exposed at work, such as radium dial painters and uranium miners. (The latter are discussed in more detail below.)

Studies of survivors of the atomic bombings of Hiroshima and Nagaski indicate statistically significant excess cancers for doses greater than 0.2 grays. These doses were delivered suddenly, following explosions. A number of problems arise when using such data to estimate cancer risks for lower doses of ionizing radiation or doses delivered in gradual increments.

The first problem is how to extrapolate the dose-response relationship down to low doses. It is usually assumed that a "linear no-threshold" model applies—that is, the risk is directly proportional to dose, with no threshold. Because the main effect of low-dose radiation is the induction of cancer, and cancer is a common disease with many causes, it is not yet possible to verify the linear no-threshold model; nevertheless, there is considerable radiobiological evidence for this theory and it is generally used for public health protection purposes, such as setting standards.

The second problem is that some assumption has to be made about how calculations of cancer risk will change in the future. After all, more than half the Hiroshima and Nagasaki survivors are still alive. At present, the data best fit a relative-risk model—that is, the cancer risk is proportional to the "spontaneous" or "natural" cancer risk. If this is correct, there will be an increasing number of radiation-induced cancers later in life.

A third problem is that the relative biological effectiveness of radiation depends partly on the energy of the radiation. For instance, data indicate that low energy neutrons and alpha particles may be more effective in producing biological damage than high energy particles (per unit of absorbed energy).[7] Thus, assuming a constant quality factor, as is common practice, can sometimes yield an inaccurate estimate of the dose.[8]

Finally, there are uncertainties related to the effect of low doses and low dose rates of low-LET radiation. The conclusion of the BEIR Committee, ICRP, and others is that low doses and dose rates of low-LET radiation are less effective in producing cancer, particularly leukemia, than would be expected based on linear extrapolation of data for low-LET radiation at high doses and high dose rates (i.e., the effect is nonlinear at low doses and dose rates). Unfortunately, the epidemiological database for evaluating the validity of DREF adjustments is sparse.[9]

Despite these potential limitations, most cancer projections continue to utilize the cancer risk factors estimated by established radiological protection committees. Their current estimates are as follows:

- UNSCEAR, 1993:[10] 0.11 fatal cancers per person-sievert for high doses (comparable to those experienced by the survivors of the Hiroshima and Nagasaki bombings). For low doses, UNSCEAR states that "no single figure can be quoted" for the risk reduction factor, "but it is clear that the factor is small. The data from the Japanese studies suggest a factor not exceeding 2."[11] For a population between the ages of 18 and 64 (corresponding to the ages of people in a typical industrial

7. National Research Council 1990, pp. 27–30.

8. There is also some experimental evidence that alpha particles may be more effective than recognized heretofore in producing chromosomal abberations. Kadhim et al. exposed stem cells in the bone marrow of mice to a range of doses of alpha particles from plutonium-238. Many surviving cells were either not traversed by a particle or suffered ionization from only a small part of the track. When the chromosomes of these surviving cells were analyzed, a surprisingly high frequency of aberrations was evident many cell divisions after the original exposure. Comparable aberrations were not induced by similar doses of X rays. The implication of this research, if confirmed, for the biological effectiveness of alpha radiation needs to be evaluated (Kadhim et al. 1992).

9. One study that provides some evidence against application of DREF adjustments for cancer was a recent study of British workers that suggested an excess cancer mortality risk of 1 death per 10 sieverts of low-LET radiation exposure. However, the lower 95 percent confidence bound for the risk was below zero and the finding was not statistically significant. In view of the lack of statistical significance, the authors of the study did not feel that any changes in existing practice of risk estimation were warranted due to their findings (Kendall et al. 1992).

10. UNSCEAR 1993, pp. 16–17.

11. UNSCEAR 1993, p. 17.

work force), a factor of 2 yields a fatal cancer risk at low dose rates of 0.04 per person-sievert.

- BEIR Committee, 1990:[12] 0.08 fatal cancers per person-sievert for a single dose of 0.1 sievert, based on Hiroshima and Nagasaki survivor data. This figure is unadjusted for any reduction of risk at low dose rates.
- ICRP, 1991:[13] 0.05 fatal cancers per person-sievert for the entire population and 0.04 fatal cancers per person-sievert for adult workers, with both estimates being for low doses and incorporating a dose rate reduction factor of 2.
- The U.S. Environmental Protection Agency uses a cancer *incidence* risk factor of 0.06 per person-sievert.[14] Since the cancer incidence rate is about 50 percent greater than the cancer fatality rate, the implicit risk for fatal cancers is about 0.04 per person-sievert.

Estimates of the risk per unit dose may be revised substantially again (upward or downward). As the BEIR committee points out:

> Most of the A-bomb survivors are still alive, and their mortality experience must be followed if reliable estimates of lifetime risk are to be made. This is particularly important for those survivors irradiated as children or in utero who are now entering the years of maximum cancer risk.[15]

Radiation Protection

Radiation protection regulations are based on three basic recommendations originally made in 1977 by the ICRP and reaffirmed later:[16,17]

- *Justification:* No practice involving exposures to radiation should be adopted unless it produces enough benefit to the exposed individuals or to society to offset the radiation detriment it causes.
- *Optimization:* Exposures to radiation should be as low as reasonably achievable.
- *Individual dose and risk limitation:* No individual should receive radiation doses higher than the maximum allowable limits.

The most difficult of these principles, and certainly the one that is rarely adequately addressed, is justification. Assessing the likelihood that any practice will produce a net benefit involves many value judgments that are difficult, if not impossible, to quantify. ICRP recognizes this:

> The Commission recommends that, when practices involving exposure, or potential exposure, to radiation are being considered, the radiation detriment should be explicitly included in the process of choice. The detriment to be considered is not confined to that associated with radiation—it includes other detriments and the costs of the practice. Often, the radiation detriment will be a small part of the total. The justification of a practice thus goes far beyond the scope of radiological protection.... To search for the best of all the available options is usually a task beyond the responsibility of radiological protection agencies.[18]

This point is expanded in a statement by the Committee on Radiation Protection and Public Health of the OECD Nuclear Energy Agency:

> Decisions about the justification of a practice or activity involving radiation exposure usually involve a broad range of social, economical and political issues in addition to those concerning radiological protection.... Justification is essentially a political decision-making process, in which the technical and purely radiation-related advantages or detriments play an important, but relatively limited role.[19]

In the early years of nuclear weapons development, the scientists and administrators involved implicitly assumed that national security justified the risks of the enterprise. According to J. Newell Stannard, "In 1947, the data for plutonium and the other actinides were used at a series of three-nation conferences on radiation exposure limits.... They required careful interpretation, for the most conservative interpretation could have closed Los Alamos."[20]

The principle of justification continues to be a cornerstone of ICRP philosophy, but the application of this principle to a particular situation in the nuclear industry, whether civil or military, is rarely discussed.[21]

Optimization implies that measures will be taken to reduce exposures until the benefits of further reductions do not justify their cost. It is not clear how this principle can be rigorously applied, particularly as it requires some quantitative estimate of the monetary value of a life saved. In practice, optimization is applied in two ways: as an exhortation to use "best available technology" and as a recognition that merely complying with dose limits is not enough. If further dose

12. National Research Council 1990, pp. 5–6.
13. ICRP 1991, pp. 69–70.
14. U.S. EPA 1993, p. 7.
15. National Research Council 1990, p. 8.
16. ICRP 1977, p. 3
17. ICRP 1991, p. 28.
18. ICRP 1991, para. 115.
19. Nuclear Energy Agency 1992, p. 15.
20. Stannard 1988.
21. QUEST Radiation Database (1992) gave just 5 references to "justification" but 91 to the principle of optimization.

reductions are practicable at reasonable cost, they should be made. Optimization generally refers to collective rather than individual radiation doses.

The principal dose limits recommended in ICRP Publication 26 (1977) were 50 millisieverts (5 rem) per year for radiation workers and 5 millisieverts (500 millirem) per year for members of the public. A subsidiary recommendation to keep doses to the public below 1 millisievert per year if possible has slowly become the primary long-term dose limit for the public, with short-term exposures of 5 millisieverts per year allowed.

The ICRP intended these limits to apply to the total exposure from all sources except natural background radiation. It has developed a methodology for combining the doses from different sources—such as combining exposures from inhaling ore dust with those from gamma exposure—and it is this total that should be compared with the appropriate limit.

In 1991, the ICRP revised its radiation protection standards, largely in response to reevaluation of dosimetry and cancer risk among atomic bomb survivors.[22] The most significant change lowered the worker's annual limit to 20 millisieverts. Regulations do not yet widely reflect this change.

UNDERSTANDING NONRADIATION HAZARDS

In addition to radiation hazards, nuclear weapons production requires and generates enormous quantities of a wide variety of toxic substances. Estimating the potential harm of each of these substances requires the application of basic principles of toxicology. Among the major principles are that toxins, or their metabolic products, must come into close contact with the target organ for which they have the potential to cause injury; that the observed toxicity should be quantitatively related to the degree of exposure to the toxin, i.e. there should be a "dose-response relationship;" and that toxicity varies according to a toxin's chemical and physical form, the route of exposure to humans, the level and duration of the exposure, the mechanism of toxicity (i.e., the fundamental chemical and biologic interactions and resultant aberrations that are responsible for the toxic response), and the presence of modifying factors (e.g., species, sex, and environmental conditions).

Since it is far beyond the scope of this book to provide a summary of basic toxicology, readers are referred to recent texts.[23] Nevertheless, it is possible to provide an overview of toxic outcomes that can be expected from exposure to general classes of nonradiation hazards that are commonly experienced in the nuclear weapons industry.

22. ICRP 1991.

23. See for example Sullivan and Krieger 1992.

Mineral Dusts—Silica

Mineral dusts in the nuclear weapons industry are primarily encountered in uranium mines, where dusty conditions prevail if industrial hygiene controls are not applied. Of greatest concern is the inhalation of silica dust generated by drilling hard rock, which can lead to a variety of pulmonary diseases collectively known as silicosis. Most characteristic is the gradual development of nodular fibrosis (a type of scarring) in the lungs. This typically produces in an individual restricted lung function, a pattern of round opacities (multiple spots) on chest X ray, and the main clinical symptom of progressive shortness of breath. Common complications include respiratory insufficiency with diminished oxygenation and an increased risk of tuberculosis. Sufferers also are at increased risk of rheumatoid arthritis, scleroderma and other rheumatic disorders, kidney disease, and possibly lung cancer. Very high exposures to silica dust can lead to an acute form of silicosis in which the lung alveoli (air sacs) fill with a thick proteinaceous material, ultimately causing death by asphyxiation within a few years of exposure.

Heavy Metals

Heavy metals in the nuclear weapons industry include highly toxic substances that have wide commercial applications (such as chromium and mercury), metals that may be encountered in mining (such as molybdenum, vanadium, and arsenic), and other metals that are used for specialized purposes (such as barium, nickel, and chromium). Uranium itself is a heavy metal and has toxic effects in addition to its radiological effects.

Significant exposure to metals occurs mainly through inhalation of dust or fumes (tiny particles with the appearance of smoke, e.g., welding fumes), or by ingestion of contaminated food, water, or by smoking cigarettes. The degree to which a body will absorb a metal (i.e., the internal dose) depends largely on the metal's chemical form. For instance, trivalent chromium (Cr^{+3}) compounds are less toxic than hexavalent chromium (Cr^{+6}) compounds, due to differences in both absorption and oxidation. Once metals gain entry into the body, the circulatory system widely distributes them to hard and soft tissues.

In general, the most toxic metals, particularly lead, mercury, cadmium, and arsenic, are poisons that interfere with metabolism and enzymatic function on a cellular level, thereby leading to damage to

many different organs and the possibility of fatal injury at high enough doses. Lower amounts of persistent exposure can lead to gradual accumulation and increase the risk of chronic disease. The most common targets of metal toxicity are the kidneys and the neurological system. Some also affect the lung, and others increase the risk for cancer.

Lead is by far the most studied metal toxin, due in part to the widespread nature of lead exposure in contemporary society from the inhalation of combusted leaded gasoline and air from polluting lead industries, and the ingestion of lead originating from the unfortunate use of lead historically in domestic products such as paint, ceramics, solder, and plumbing. Decades ago, children who ate lead paint chips were noted to develop constipation, abdominal pain, seizures, mental retardation, anemia, limb weakness, and other manifestations of systemic toxicity. Adults suffer similar effects, although their already developed nervous systems are less vulnerable to lead's neurotoxicity. Most recently, lead has received increased attention because of investigations indicating an adverse effect of lead on neurological development (affecting, for instance, learning and hearing acuity) in children and blood pressure in adults at levels of exposure far lower than previously thought.[24]

Due to its low vapor pressure, mercury exposure can occur readily through the inhalation of elemental mercury vapor. Alternatively, mercury contained in pollutant discharges, as has occurred in lithium-6 production for nuclear weapons, can be converted to organic mercury forms by microorganisms, leading to concentration of organic mercury in the food chain and the threat of human exposure through ingestion (particularly of contaminated fish). Mercury vapor exposure can damage the lungs, leading to toxic pulmonary edema (lung injury followed by fluid accumulation and interference with gas exchange). The central nervous system is the primary target of chronic elemental mercury exposure, with injury producing the classic triad of tremor, gingivitis, and erethism (insomnia, shyness, memory loss, emotional instability, nervousness, and anorexia). Ingested elemental mercury is not absorbed well, although it can produce local gastrointestinal corrosion. Ingestion of organic mercury, particularly methyl mercury, leads to a form of central nervous system toxicity with some similarities to but also important differences from that of elemental mercury. Typical are depression, decreased intellectual ability, clumsiness (from cerebellar damage), sensory numbness, slurred speech, and spasticity of movement leading to paralysis. As with lead, increased attention is now being focused on the risks posed by low-level mercury exposure. Recent epidemiological work found an association between low levels of mercury in urine and indicators of early kidney and neuro-psychological damage.[25]

Arsenic exposure can occur through inhalation, which is an issue in uranium mining, as well as ingestion. Arsenic is an effective acute poison at high doses, leading to severe blood, brain, heart, kidney, and gastrointestinal tract injury. Lower levels of exposure can cause skin eczema and darkening, muscle wasting, and painful peripheral nerve lesions. Of greatest concern with low levels of exposure is cancer, particularly of the skin, lungs, bladder, and liver, as well as other organs.

In addition to the radiological hazard it poses, uranium poses a significant risk of kidney damage. Acute damage can be severe, but also is largely reversible. Low-level exposure may possibly cause kidney damage in the form of tubular dysfunction. Compounds of uranium that are commonly found in the nuclear weapons industry also carry the risk of kidney damage as well as other toxicities. One such example is uranium hexafluoride, which, in the presence of water, hydrolyzes to uranyl fluoride and hydrogen fluoride, a severe irritant (see discussion below).

Exposures to beryllium, a hard metal used extensively in the nuclear weapons industry, occur principally through inhalation of beryllium particles or oxides. Beryllium's main toxic effects take the form of inflammation and immunological sensitization manifesting, for instance, in fibrosis of the lung (with resultant shortness of breath) and enlargement of internal lymph nodes. The mechanisms are poorly understood. The spectrum of health effects is similar to those seen in other chronic granulomatous diseases, particularly sarcoidosis. A mildly elevated risk of lung cancer may also exist.

Acids

The nuclear weapons industry involves the heavy use of acids, such as nitric acid, that can cause intense irritation and destroy cells upon contact. These compounds are highly reactive, and their effects are generally nonselective and limited to the sites of exposure. Acids easily burn the skin and eyes. Inhaling vapors, gases, or dusts of acids in their anhydride form can cause lung injury. Scarring can complicate recovery from acid skin burns, and severe pulmonary inhalation injury can lead to fibrosis, restrictive lung disease, or chronic obstructive airway disease.[26] As a rule, acids are not absorbed, and their action stops at the sites of exposure.

24. ATSDR 1988.

25. Rosenman et al. 1986.

26. Linden 1992.

The uniquely toxic properties of hydrofluoric acid are noteworthy, however, particularly because it is commonly used in nuclear weapons production. In contrast to other acids, even small amounts of hydrofluoric acid can penetrate the skin, underlying lipid barriers, muscles, and even bones. It penetrates tissues rapidly and deeply, but the effects are delayed; it may be several hours before intense pain develops at the site of the burn. The tissue is gradually destroyed, and the affected part may ultimately become gangrenous. Hydrofluoric acid can also cause pulmonary edema (excess body fluids in the lungs) and other respiratory damage. Hydrofluoric acid distributes throughout the body. The fluoride ion can dissociate from the hydrogen ion and combine with calcium, magnesium, and other positively charged ions to increase the risk of cardiac arrhythmias. Depending on the exposure level, burns from hydrofluoric acid can be much more severe than they first appear and can even lead to fatal metabolic disturbances.[27] Some of these properties are also shared to a degree by other fluorine-containing compounds found in the nuclear weapons industry, notably chlorine trifluoride.

Organic Compounds

The making of nuclear weapons uses a wide variety of organic compounds including solvents and chelating agents. Some are or have been used in large quantities, including toluene, carbon tetrachloride, benzene, acetone, methyl ethyl ketone, EDTA, HEDTA, and tributyl phosphate.

Most solvents consist of a single compound, but some are mixtures of compounds. Given their chemical nature, the body readily absorbs solvents through inhalation, ingestion, and skin contact. The body excretes or metabolizes most solvents fairly quickly, so they don't accumulate. Solvents mainly affect the nervous system, liver, kidneys, and skin. Benzene and some other solvents are known human carcinogens; others are suspected carcinogens. Heavy exposure produces, most acutely, an anesthetic effect manifested by dizziness and lightheadedness. Prolonged exposure can produce persistent, potentially irreversible impairment of cognitive (intellectual) function, including impaired memory and concentration, and disturbances of mood and affect such as depression. Some solvents can cause degeneration of peripheral nerves, leading to paralysis or numbness. Liver injury and kidney toxicity occur during excretion and as the body attempts to detoxify the original compound. Skin injury occurs as solvents dissolve the protective fat compounds in skin, leading to dermatitis and increasing the risk of infection.

27. Krenzelok 1992.

An important issue arising from lower levels of exposure to solvents, particularly with respect to people living near nuclear weapons plants, is groundwater contamination and possible links with cancer. Volatile organic compounds (VOCs) can migrate from hazardous waste sites through soil, leading to detectable levels in groundwater. As with many toxic agents, it is not known whether low levels of VOCs cause human cancer. A few epidemiological studies of drinking-water contamination by VOCs have found small but statistically significant increases in the risks for leukemia and cancers of the bladder, colon, and rectum.[28] Public concerns underlie the current conservative assumption for purposes of regulation that exposure to known and suspected carcinogens must be reduced to the lowest level possible.

ENVIRONMENTAL AND OCCUPATIONAL EPIDEMIOLOGY

It is important to understand the process by which scientists actually try to assess and quantify the relationship between toxic exposures and disease using environmental and occupational epidemiology. Epidemiology studies the occurrence and distribution of disease among populations. Environmental and occupational epidemiology focuses specifically on the relationship of environmental and occupational "exposures" to the development of disease. Although infectious agents, nutrition, and other personal lifestyle factors such as smoking and drinking can be considered external exposures, environmental and occupational epidemiology generally refers to exposures to chemicals, dusts, physical factors (such as radiation), jobs (which may entail multiple exposures), and other stressors.

Types of Studies

A key feature of any epidemiological study is the technique of examining the relationship between an exposure and a health outcome by comparing populations. Two main approaches exist. Cohort studies compare the health outcomes of people who were exposed versus those of people who were less exposed or not exposed. Case-control studies compare the exposures of people who have a particular disease versus the exposures of people who do not have that disease. Health outcomes are specified either in terms of mortality, usually in the form of diagnoses derived from death certificates, or morbidity (illness). In the case of cancer, morbidity usually takes the form of diagnoses compiled in cancer registries (centralized bureaus that record cases of cancer).

28. Shy 1985.