Potential Human Health Effects of Uranium Mining, Processing, and Reclamation

Key Points

- Uranium mining and processing are associated with a wide range of potential adverse human health risks. Some of these risks arise out of aspects of uranium mining and processing specific to that enterprise, whereas other risks apply to the mining sector generally and still others are linked more broadly to large-scale industrial or construction activities. These health risks typically are most relevant to individuals occupationally exposed in this industry but certain exposures and their associated risks can extend via environmental pathways to the general population.

- Protracted exposure to radon decay products generally represents the greatest radiation-related health risk from uranium-related mining and processing operations. Radon’s alpha-emitting radioactive decay products are strongly and causally linked to lung cancer in humans. Indeed, the populations in which this has been most clearly established are uranium miners that were occupationally exposed to radon.

- In 1987, the National Institute for Occupational Safety and Health (NIOSH) recognized that current occupational standards for radon exposure in the United States do not provide adequate protection for workers at risk of lung cancer from protracted radon decay exposure, recommending that the occupational exposure limit for radon decay products should be reduced substantially. To date, this recommendation by NIOSH has not been incorporated into an enforceable standard by the Department of Labor’s Mine Safety and Health Administration or the Occupational Safety and Health Administration.

- Radon and its alpha-emitting radioactive decay products are generally the most important, but are not the only radionuclides of health concern associated with uranium mining and processing. Workers are also at risk from exposure to other radionuclides, including uranium itself, which undergo radioactive decay by alpha, beta, or gamma emission. In particular, radium-226 and its decay products (e.g., bismuth-214 and lead-214) present alpha and gamma radiation hazards to uranium miners and processors.

- Radiation exposures to the general population resulting from off-site releases of radionuclides (e.g., airborne radon decay products, airborne thorium-230 ($^{230}$Th) or radium-226 ($^{226}$Ra) particles, $^{226}$Ra in water supplies) present some risk. The potential for adverse health effects increases if there are uncontrolled releases as a result of extreme events (e.g., floods, fires, earthquakes) or human error. The potential for adverse health effects related to releases of radionuclides is directly related to the population density near the mine or processing facility.

- Internal exposure to radioactive materials during uranium mining and processing can take place through inhalation, ingestion, or through a cut in the skin. External radiation exposure (e.g., exposure to beta, gamma, and to a lesser extent, alpha radiation) can also present a health risk.
Because $^{230}\text{Th}$ and $^{226}\text{Ra}$ are present in mine tailings, these radionuclides and their decay products can—if not controlled adequately—contaminate the local environment under certain conditions, in particular by seeping into water sources and thereby increasing radionuclide concentrations. This, in turn, can lead to a risk of cancer from drinking water (e.g., cancer of the bone) that is higher than the risk of cancer that would have existed had there been no radionuclide release from tailings.

A large proportion of the epidemiological studies performed in the United States, exploring adverse health effects from potential off-site radionuclide releases from uranium mining and processing facilities, have lacked the ability to evaluate causal relationships (e.g., to test study hypotheses) because of their ecological study design.

The decay products of uranium (e.g., $^{230}\text{Th}$, $^{226}\text{Ra}$) provide a constant source of radiation in uranium tailings for thousands of years, substantially outlasting the current U.S. regulations for oversight of processing facility tailings.

Radionuclides are not the only uranium mining- and processing-associated occupational exposures with potential adverse human health effects; two other notable inhalation risks are posed by silica dust and diesel exhaust. Neither of these is specific to uranium mining, but both have been prevalent historically in the uranium mining and processing industry. Of particular importance is the body of evidence from occupational studies showing that both silica and diesel exhaust exposure increase the risk of lung cancer, the main risk also associated with radon decay product exposure. To the extent that cigarette smoking poses further risk in absolute terms, there is potential for increased disease, including combined effects that are more than just additive.

Although uranium mining-specific injury data for the United States were not available for review, work-related physical trauma risk (including electrical injury) is particularly high in the mining sector overall and this could be anticipated to also apply to uranium mining. In addition, hearing loss has been a major problem in the mining sector generally, and based on limited data from overseas studies, may also be a problem for uranium mining.

A number of other exposures associated with uranium mining or processing, including waste management, also could carry the potential for adverse human health effects, although in many cases the detailed studies that might better elucidate such risks are not available.

Assessing the potential risks of multiple combined exposures from uranium mining and processing activities is not possible in practical terms, even though the example of multiple potential lung carcinogen exposures in uranium mining and processing underscores that this is more than a theoretical concern.

Many of the findings related to occupational exposures and adverse health outcomes presented in this chapter are based on studies of uranium and hard-rock miners (e.g., worker-based radon studies) for periods of disease risk when the magnitude of the exposures was much greater than the exposures reported at most mines and processing facilities in North America today. Nevertheless, although current exposures are generally much lower, contemporary uranium workers and processors in the United States continue to express work-related health concerns. For example, in 2008 the National Institute of Occupational Safety and Health (NIOSH) organized stakeholder meetings that included uranium miners and processors in Wyoming, Texas, Colorado, and Utah. The stakeholders expressed numerous health-related concerns, including concerns about exposure to alpha radiation via inhalation or ingestion of dust particles containing radon decay products, exposure to both radiation and particulate uranium via inhalation, ingestion and inhalation of ore dust, and exposure to diesel particulate matter (Miller et al., 2008).
This chapter describes some of the major human health effects related to occupational and public (i.e., off-site) health and safety as they pertain to uranium mining, processing, and reclamation in the Commonwealth of Virginia. Specifically, the chapter discusses the well-documented human health effects arising from the radioactive constituents of uranium mining that are of primary health concern, including uranium and its decay products (e.g., radium, radon). In addition, the chapter provides an overview of other, nonradioactive hazards related to mining and processing. This includes both a group of major exposures (i.e., silica, diesel, and physical exposure hazards) as well as a group of miscellaneous potential hazards related to mining in general and to uranium processing in particular. Epidemiological and other human health data derived from previous studies of uranium mining and processing were examined, as well as other relevant biomedical data pertaining to the potential exposures of interest.

It was not the Committee’s charge to develop a quantitative risk assessment, or to characterize uranium mining- and processing-associated risks scaled and ranked against various occupational and nonoccupational hazards (such as risks quantified for activities such as travel, hobby activities, or military service). Although such information might be of interest to various stakeholders in Virginia, and would undoubtedly be required for a site-specific analysis, it is beyond the resources, scope, and capabilities of the Committee as constituted to carry out the extensive research that would be required to undertake such a Virginia-wide analysis.

### RADIONUCLIDE-RELATED HEALTH HAZARDS

For many of its aspects, the potential adverse health effects associated with uranium mining are no different than the risks identified in other types of non-radiation-related mining activities (Laurence, 2011). Uranium mining, however, adds another dimension of risk because of the potential for exposure to elevated concentrations of radionuclides. Internal exposure to radioactive materials during uranium mining and processing can take place through inhalation, ingestion, or absorption through an open cut or wound. External radiation exposure from beta particles or gamma rays can also present a health risk.

Radiation typically encountered in uranium mining or processing facility operations includes alpha (α), beta (β), and gamma (γ) radiation. All three are types of ionizing radiation—energy in the form of particles or waves that has sufficient force to remove electrons from atoms. Alpha particles consist of two neutrons and two protons, travel only a few centimeters in air, and can cause a high density of ionizations along their path. In some cases, alpha particles can penetrate the dead layer of skin. If radionuclides that decay by alpha emission (e.g., polonium-218, polonium-214) are inhaled, they have the potential to impart a significant dose to the pulmonary epithelium. The dose of alpha energy delivered by an alpha particle to the DNA in a cell in the respiratory epithelium is fixed and not dependent on concentration or duration of exposure. Although alpha particles can travel only a short distance, they impart a much greater effective dose than beta particles or gamma rays (NRC, 1988, 2008b). The high effective doses from alpha particles, as compared with beta particles or gamma rays, result from their relatively high energies combined with their very short ranges in tissue. Alpha particles are notable among environmental carcinogens because of their potent ability to produce a high proportion of double-strand DNA breaks per particle. Double-strand DNA breaks are more difficult for the body to repair.

Compared with alpha particles, beta particles are light and fast electrons with a mass of about 1/2000th of a proton. Beta particles have greater penetrating power than alpha particles, but have much less ability than alpha particles to ionize tissues and cause disruptions of the DNA. Beta particles present both an external and an internal radiation hazard. Beta particles can travel over 50 cm in air and, if an individual is externally exposed, beta particles can penetrate the dead layer of the skin and reach the germinal layer of the skin. In most exposure scenarios related to uranium mining and processing, beta radiation presents a greater external than internal radiation hazard. For example, the beta dose rate from uranium decay products is negligible immediately after separation of uranium, but can produce a beta dose rate on contact of about 150 mrem/hr several months after separation because of the buildup of $^{234}$Th (USNRC, 2002).
Gamma rays are not particles, but rather are highly penetrating electromagnetic radiation traveling at the speed of light. Gamma rays do not have a charge or mass; they are highly penetrating radiation that can ionize atoms in the body directly or cause “secondary ionizations” when their energy is transferred to atomic particles such as electrons. In most exposure scenarios related to uranium mining and processing, gamma rays present a greater external than internal radiation hazard.

The energy deposited by alpha, beta, or gamma radiation can damage or kill cells. The impact of radiation on a cell depends on the duration of radiation exposure, the dose rate of the exposure, the total amount of energy absorbed, and the tissue or organ exposed. If radiation damages a cell’s genetic material (DNA) and the cell survives, this damage can initiate cancer. The risk of cell damage increases with increasing dose. Although radiation-induced heritable mutations have not been documented in the children of uranium mine or processing workers, or in the children of Japanese atomic bomb survivors, there is some very limited evidence (lacking consistent findings of exposure-response) suggesting that radiation-induced heritable mutations may occur in humans (NRC, 2006; Kodaira et al., 2010; Bunin et al., 2011; Tawn et al., 2011).

The radionuclides of greatest health-related concern in uranium mining and processing are those present in the uranium-238 ($^{238}\text{U}$) (Figure 5.1), uranium-235 ($^{235}\text{U}$) (Figure 5.2), and thorium-232 ($^{232}\text{Th}$) decay series. The potential for occupational exposure to uranium or thorium and their decay products can vary greatly depending on numerous factors, including the type of ore deposit, uranium grade, mineralogy of deposit, production capacity, uranium mining method, production rate, variation in process methods (e.g., types of crushers or grinders), reagents used in the chemical dissolution of uranium-bearing mineral species, solid-liquid separation method, purification method, precipitation, packaging, transportation, waste treatment (e.g., effluent treatment, or water treatment), storage of tailings, environmental conditions around the plant (e.g., hydrological balance and local geology), and engineering controls and safeguards. Although $^{232}\text{Th}$ sometimes occurs in high concentrations in uranium deposits, limited data suggest that presently known commercially viable uranium occurrences in Virginia (see Chapter 3) are unlikely to contain high $^{232}\text{Th}$ concentrations.

**FIGURE 5.1**


**FIGURE 5.2**


In addition to $^{238}\text{U}$, the radionuclides of greatest health concern in this decay series are uranium-234 ($^{234}\text{U}$) with a 240,000-year half-life, thorium-230 ($^{230}\text{Th}$) with its 77,000-year half-life, radium-226 ($^{226}\text{Ra}$) with a 1,600-year half-life, and the short-lived radon-222 ($^{222}\text{Rn}$) decay products—polonium-218 ($^{218}\text{Po}$), polonium-214 ($^{214}\text{Po}$), and polonium-210 ($^{210}\text{Po}$). In modern uranium processing facilities, over 97 percent of the uranium in the ore can be extracted. However, other radionuclides with potential adverse health effects, including $^{230}\text{Th}$, $^{226}\text{Ra}$, $^{222}\text{Rn}$, and $^{210}\text{Po}$, and their decay products, remain in the tailings and other waste materials generated by the extraction. In fact, about 85 percent of the original radioactivity in the ore remains after the uranium is extracted. Of particular note, the 77,000-year radioactive half-life of $^{230}\text{Th}$ provides a constant source of $^{226}\text{Ra}$. Both radionuclides ($^{230}\text{Th}$ and $^{226}\text{Ra}$) are common components of leached materials and airborne dusts from uranium ore tailings and waste piles.
and $^{230}$Th and $^{226}$Ra can pose a health hazard if inhaled or ingested. Radium-226 and its decay products present both an alpha (e.g., internal exposure hazard) and a gamma (e.g., external exposure hazard from the decay products bismuth-214 and lead-214) radiation hazard to miners as well as to uranium processors.

A summary of the major radon and uranium series occupational exposure standards is presented in Table 5.1; note that this table is not intended to be an exhaustive compilation of all recommendations regarding radon and uranium occupational exposure limits, but rather is intended to highlight the complexity and the differences among the guidelines as context for ensuing descriptions of dose and exposure standards and regulations both in this chapter and in Chapter 7. For additional background, Box 5.1 presents a summary of the rather confusing terms and units used for radiation activity, exposure, and dose. Additional information on current regulations and guidelines applicable to uranium is available in ATSDR (2011).

### TABLE 5.1
Selected Radon and Uranium Decay Series Occupational Exposure Regulations and Standards.

### BOX 5.1
Common Units and Terms Used for Radiation Activity, Exposure, and Dose. The activity, or rate of nuclear transformations, of a radionuclide is expressed in disintegrations (or decays) per unit of time. The two units for radiation activity are the curie (more...)

The type of radiation exposure that may be encountered in uranium mining and processing varies by source material and work process (Table 5.3). For example, uranium miners working in underground mines generally have a much greater potential for exposure to radon and radon decay products during the mining process as compared with miners working in open-pit mines (UNSCEAR, 2000). In addition to radon and its short-lived alpha-emitting decay products (i.e., $^{218}$Po, $^{214}$Po), other important sources of airborne radioactivity in the mine include the longer-lived radioactive decay products of $^{238}$U and $^{235}$U (e.g., $^{234}$U, $^{230}$Th, $^{226}$Ra, $^{210}$Po) (Ahmed, 1981). Work with processed uranium (e.g., yellow-cake) generally only increases the potential for alpha exposure. However, drums containing yellowcake that have been stored for several months can lead to increased exposure to x-rays as a result of the interaction of beta particles from aged yellowcake with the steel drums; the beta surface dose is about 150 mrem/hr after a few months (USNRC, 2002) (this potential beta and x-ray exposure is not included in Table 5.3). Work with materials that have undergone uranium separation (e.g., mine or processing plant tailings) primarily presents an alpha and gamma radiation hazard. Process workers in proximity to materials that are being tipped into comminution equipment (grinder) are often at greater risk from airborne exposure to radioactive materials, while those performing maintenance on such equipment may be at higher risk of gamma radiation exposure.

### TABLE 5.3
Simplified Matrix Showing Potential Exposure Types and Some of the Major Radionuclides Associated with Different Mining and Milling Processes That Have the Potential to Cause Adverse Health Effects (X indicates elevated potential for exposure).

Worker radiation exposures most often occur from inhaling or ingesting radioactive materials or through external radiation exposure. Generally, the highest potential radiation-related health risk for uranium mining or processing facility workers is lung cancer associated with inhaling uranium decay products (more specifically, radon decay products), as well as other non-lung-
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...cancer risks associated with gamma radiation exposure on-site. Nonoccupational radiation exposures to the general population can occur from airborne dispersal of radioactive particulates to off-site locations, including subsequent resuspension, or gases from mining operations, processing facility exhausts, waste rock, wastewater impoundments, or tailings. Exposures may also occur by release of contaminated water or leaching of radioactive materials into surface or groundwater sources where they may eventually end up in potable water supplies. Radon and its decay products can also be transported off-site, especially from tailings or waste areas, in the form of radon gas or radon decay products. The potential for internal radiation exposure from drinking water contaminated with radionuclides (e.g., $^{226}$Ra, $^{228}$Ra, $^{230}$Th, uranium) that have been leached or otherwise released from tailings or other wastes is a common health concern for the public (Landa and Gray, 1995; Baker, 2010). Another health concern for people living near mines and processing facilities is the potential for off-site radiation exposure from atmospheric deposition of “fugitive” ore or tailings dust (e.g., dust containing uranium, $^{226}$Ra, $^{230}$Th, $^{210}$Pb, $^{210}$Po, and other radionuclides). Even though such fugitive dusts are extensively diluted once they leave the plant or mine boundaries (Thomas, 2000), accumulation in the food chain can occur with subsequent human consumption of wild or domestic animal meat, fish, or milk.

Additional information concerning a selection of the major radionuclides of health interest ($^{222}$Rn, $^{238}$U, $^{226}$Ra) is presented below.

### RADON HEALTH HAZARDS

Three radon isotopes are generated in the $^{238}$U, $^{235}$U, and $^{232}$Th decay chains, including radon-222 (radon), radon-219 (actinon), and radon-220 (thoron). These are the immediate decay products of $^{226}$Ra, radium-223 ($^{223}$Ra), and radium 224 ($^{224}$Ra), respectively. Because $^{235}$U has low abundance in natural crustal rock, as compared with $^{238}$U, and because of the relatively short radioactive half-life of its radon decay product, actinon (Figure 5.2), $^{235}$U is generally not considered to be a significant health risk as compared with $^{238}$U in the mining and processing setting. In addition, the majority of uranium deposits in Virginia are thought to contain low concentrations of $^{232}$Th (see Chapter 3). Therefore, thoron, a radioactive decay product of $^{232}$Th, as noted above, is anticipated to present a much lower risk to workers than exposure to radon-222 decay products.

Radon-222, hereafter referred to as radon, is a colorless and odorless gas that possesses no sensory reminders that provide an alert to its presence. It is ubiquitous in soils, rocks, and groundwater supplies. Radon has the longest half-life among the 35 known isotopes of radon, including the other two forms (i.e., actinon and thoron) noted above. Because the relative abundance of radon, its relatively long half-life compared with the other radon isotopes, as well as its alpha-emitting decay products, protracted exposure even at background levels accounts for an adverse human health risk, while exposure exceeding such background levels contributes a further increased incremental adverse health risk.

Radon is formed from the radioactive decay of radium-226 (Figure 5.2). It has a half-life of 3.8 days and decays into a series of radioactive solid decay products, ending with stable lead-206. The radon decay products, particularly $^{218}$Po and $^{214}$Po, deliver the primary radiation dose to the respiratory epithelium, rather than the radon gas itself. After the decay of radon gas, the short-lived solid decay products that remain suspended in air undergo varying degrees of attachment to ambient aerosols. The percentage of decay products that attach is influenced by numerous factors, including air movement and aerosol concentration as well as ambient particle size. Pulmonary deposition of radon decay products depends on particle size (which is affected by the proportion of attached or unattached decay products), volume of air displaced between normal inspiration and expiration, breathing rate (which is affected by mining or processing-related physical activity), nasal versus oral breathing (which is also affected by mining- or processing-related physical activity), and lung volume. The quantity and distribution of deposited radon decay products is influenced by mechanisms that remove the radon decay products from the lung or move them to other areas of the lung and body (NRC, 1991, 1999b; ATSDR, 2008).
Once deposited in the lung, the short-lived radon decay products, $^{218}\text{Po}$ and $^{214}\text{Po}$, rather than the radon gas, deliver the majority of the radiation dose in the form of alpha particles to the respiratory epithelium. Alpha particles impart a high density of ionizations along their short path (i.e., high linear energy transfer), a process that results in DNA damage. Radiation-induced carcinogenesis is thought to arise from DNA damage to a single cell (i.e., cancer is monoclonal in nature). NRC (1999b) concluded not only that there is overwhelming evidence supporting such a monoclonal cancer origin, but also that there is no apparent threshold for radon-induced lung cancer. Radon-caused lung cancer is one of the earliest recognized forms of occupational cancer. An overview of the earlier history of radon-caused cancer of the lung is presented in Box 5.2.

**BOX 5.2**

Early History of Lung Cancer and Uranium Miners. Although it is broadly appreciated by the general public that radioactive exposures—including radon—carry adverse effects, this has not always been the case. In particular, the link between (more...)

### Mining-Based Epidemiological Studies of Radon Health Effects

The highest radon-related exposures to workers generally occur during underground uranium mining operations. However, significant radon exposure can also occur in open-pit mines, for example, as a result of meteorological factors such as air inversions. As noted above (Table 5.3), radon exposures can also occur during several of the steps in uranium ore processing as well as from radon emanation from tailings and from mining and processing wastes. Findings from early studies of radon-exposed underground miners performed in Central Europe (see Box 5.2), as well as more formal epidemiological investigations of underground miners in the United States (e.g., Wagoner et al., 1965), provided very strong evidence by the mid-1960s to causally link protracted radon decay product exposure with lung cancer (UNSCEAR, 2009; Samet, 2011).

Over 20 retrospective epidemiological studies examining the association between radon and cancer mortality have been performed in North America, Europe, and China. In a typical retrospective radon-related cohort mortality study, the investigators identify a cohort of exposed workers (e.g., underground radon-exposed uranium or hard-rock miners) and then determine their disease experience (i.e., cancer occurrence) many years after their initial mining exposures. The assessment of retrospective radon exposure, as well as other important exposures in the same workplace (e.g., diesel, arsenic, and silica co-exposures), presents a key challenge when conducting such studies. In most cases, the retrospective assessment of radon decay product exposure has been based on periodic area measurements (e.g., a particular tunnel) of radon decay products rather than on measurements of radon decay product concentrations in close proximity to where the miners worked as would be done if personal dosimetry data for radon exposure were available. The collection of important lifestyle information, such as cigarette smoking, has also been lacking in many of the retrospective cohort mortality studies of underground radon-exposed miners. Even with these limitations, the overwhelming majority of the epidemiological studies have demonstrated a positive linear dose-response relationship between radon decay product exposure and lung cancer; that is, the greater the exposure, the greater the risk, falling on a straight line (Samet, 1988; NRC, 1999b; ATSDR, 2008).

To develop a more comprehensive assessment of the risk posed by protracted radon exposure that included adjustment for potential concomitant risk factors for lung cancer (e.g., smoking, silica exposure), data have been pooled (i.e., combined) from multiple retrospective mortality studies to increase the sample size available for analyses (NRC, 1988). A pooled epidemiological study is a type of combined study that collects the raw data from the studies and uses these data for a new overall analysis. The most extensive pooling of data from retrospective cohort mortality studies of radon was performed by Lubin and colleagues (1994) and served as the basis for a subsequent pooling by the NRC’s Committee on the Biological Effects of Ionizing Radiation.
The BEIR VI analysis pooled data from 11 radon-exposed retrospective mortality studies of miners with very long follow-up of mortality and included nearly 2,800 lung cancer deaths. The pooled cohort data included radon-exposed miners from the United States, Canada, Australia, France, the Czech Republic (at that time part of Czechoslovakia), Sweden, and China. Each of the 11 studies had independently found increased lung cancer mortality rates associated with increased exposure to radon and its decay products (Lubin, 2010). For comparison, the mean cumulative radon exposure from the pooled miner studies is approximately 10 times higher than the exposure an individual would receive from spending a protracted period (e.g., decades) in a home with radon concentrations similar to the U.S. Environmental Protection Agency’s (USEPA) Radon Action Level of 4 pCi/L.

Every study of miners examined in the BEIR VI report (NRC, 1999b) included the range of exposures that overlap with the cumulative exposures experienced in homes at the USEPA’s Radon Action Level of 4 pCi/L (Lubin, 2010). The BEIR VI estimates of the risks posed by lower level radon decay product exposures are particularly relevant to the general public living near uranium mining and processing operations, because radon decay product exposure has been shown to be an important source of radiation exposure to nearby offsite communities (SC&A, 2011).

Numerous factors affected the excess relative risk related to radon decay product exposure quantified in working level months (WLM). A WLM is used to quantify cumulative exposure to radon decay products (see glossary for more details). The risk estimate was affected by smoking history, dose rate, and age at exposure. For example, the BEIR VI committee observed that exposure to both radon and tobacco usage increases lung cancer risk higher than simply an additive effect, but less than a full multiplicative degree of risk. Thus, the risk of lung cancer among uranium miners who smoke cigarettes is greater, in absolute and relative terms, than the risk for cigarette smokers who do not experience radiation exposure; moreover, the incremental increase in absolute risk (reflected in the rate of lung cancer among those concomitantly exposed) is more than simply the rates added together—thereby indicating a degree of synergism—even though the combined rate may not be as high as the cross-product of the rates multiplied against each other. The International Council of Radiation Protection (ICRP, 2012) indicates, based on the pooled results from radon-exposed miner studies, that a lifetime excess absolute risk of $5 \times 10^{-4}$ per WLM should be used as the nominal probability coefficient for radon progeny-induced lung cancer.

Since the publication of the BEIR VI Report, additional findings from other radon-related miner studies further support the findings of the BEIR VI report (e.g., Villeneuve et al., 2007; Schubauer-Berigan et al., 2009; Kreuzer et al., 2010; Lane et al., 2010; Leuraud et al., 2011). Additional information summarizing the experience of radon-exposed miner cohorts is presented in the report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 2009).

Although the occupational lung carcinogenicity of radon decay product exposure has been clearly established for decades, the causal association between occupational radon exposure and cancer of other types (i.e., nonlung cancer), as well as radon-related non-cancer adverse health outcomes, has been less clear. Such endpoints are of concern because, in addition to the respiratory epithelium, protracted radon decay product exposure can deliver varying degrees of radiation dose to other sites in the body, including the skin, bone marrow, and kidney (Kendall and Smith, 2002). Several researchers have published findings that are suggestive of an association between occupational radon decay product exposure via mining and leukemia, as well as cancers of the stomach, liver, and trachea (Darby et al., 1995; Kreuzer et al., 2008, 2010).

Since retrospective mortality studies generally rely on adverse health outcomes noted on death certificates or mortality registries, cancers with a long survival period—or other non-cancer adverse health conditions that cannot be accurately determined—cannot be assessed with the same reliability as for lung cancer, from which survival is generally not extended. For example, Bedford (2010) found that the ability of death certificates to document cancer occurrence is
directly related to the survival period of the cancer. Cancers with relatively short survival periods (e.g., pancreatic cancer, lung cancer) are more likely to be noted on a death certificate. One of the few studies to examine cancer incidence, rather than mortality, was performed by Řeřicha et al. (2006) in Czech uranium miners and reported a positive association between radon exposure and leukemia, including chronic lymphocytic leukemia. Additional well-designed epidemiological studies are required to assess further the possible association between radon decay product exposure and other adverse health outcomes (Linet et al., 2007; Field, 2010). The need for additional epidemiological studies is particularly crucial for radon-exposed female workers, because there is little information on radon decay product exposure and the occurrence of female-specific cancers, for example, cancer of the breast or ovaries (Field, 2010).

Studies examining possible associations between protracted radon exposure and non-cancer adverse health outcomes are almost nonexistent (NRC, 1999b). Archer and colleagues (1976) noted a linear positive relationship between radon decay product exposure and nonmalignant respiratory disease in nonsmoking uranium miners, that the authors attributed to diffuse parenchymal radiation damage.

**Occupational Exposure Guidelines for Radon**

In many cases, the primary radiation risks associated with uranium mines and processing facilities are exposure to radon decay product exposure (Ahmed, 1981; NIOSH, 1987) and gamma radiation. Although the radon decay product concentrations measured in mines today are expected to be less than those that were routinely observed in the past, there have been efforts by NIOSH to lower (i.e., make more protective) the allowed exposure promulgated in the current U.S. standards (NIOSH, 1987). The current Mine Safety and Health Administration (MSHA) and Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) for cumulative radon decay product exposure is 4 WLM per year (Table 5.1). Using the ICRP risk estimate of $5 \times 10^{-4}$ lifetime risk of lung cancer per WLM as cited above, the 4 WLM/yr limit at 30 years of exposure would result in a 6 percent increase in lifetime risk of lung cancer (i.e., 600 per 10,000 persons thus exposed). The quantitative risk assessment performed by the U.S. National Institute for Occupational Safety and Health (NIOSH) in the 1980s concluded that exposures to 1 WLM per year over a 30-year working lifetime posed substantial health risks (NIOSH, 1987). Despite such risks, in 1987 NIOSH recommended lowering the PEL from 4 WLM/yr to 1 WLM/yr (NIOSH, 1987). In putting forward the NIOSH recommendation, NIOSH Director and Assistant Surgeon General, Dr. J. Donald Millar noted that although NIOSH was recommending lowering of the PEL to 1 WLM/yr for radon decay product exposure, he did not believe the recommendation satisfied NIOSH’s commitment to protect the health of the nation’s miners. He went on to state that, “if new information demonstrates that a lower exposure limit constitutes both prudent public health and a feasible engineering policy, NIOSH will revise its recommended standard” (NIOSH, 1987, p. vi). Subsequent miner-based studies (Lubin et al., 1994) have provided convincing evidence that a PEL of 1 WLM/yr, even if promulgated, would not provide an acceptable health-based limit to protect worker health.

**Environmental Radon Exposure and Health Effects**

Radon gas is ubiquitous in both the outdoor and indoor nonoccupational environment. The average indoor and outdoor radon concentration is 1.3 pCi/L and 0.4 pCi/L, respectively, in the United States (USEPA, 1992). Both indoor and outdoor radon environmental concentrations often undergo significant temporal and spatial variation (Fisher et al., 1998; Steck et al., 1999; Zhang et al., 2007). In some areas of the United States, the average year-long outdoor radon concentration can equal that of the national indoor average radon concentration (i.e., 1.3 pCi/L) (Steck et al., 1999). The USEPA has assigned each county in the United States to one of three radon potential zones based on numerous factors, including short-term indoor radon measurements, aerial measurements of uranium, geology, soil permeability, and building foundation type. Zone 1 counties have a predicted average indoor screening (i.e., short-term test generally performed in the basement) radon measurement greater than 4 pCi/L. Zone 2 counties have predicted indoor average screening measurements $\geq 2$ and $\leq 4$ pCi/L. Zone 3 counties have
a predicted average radon screening measurement of < 2 pCi/L. In the early 1980s, the National Council on Radiation Protection (NCRP) estimated that the average effective dose of radiation per individual in the United States was 3.6 mSv; by 2006, the average dose had increased to 6.2 mSv, primarily as a result of medically related procedures (NCRP, 2009). Radon decay product exposure delivers 37 percent of the total effective dose per individual in the United States (Figure 5.4) (NCRP, 2009).

FIGURE 5.4
Percent contribution of various sources of radiation exposure to the total effective dose per individual in the United States for 2006. Percent values have been rounded to the nearest 1 percent, except for those < 1 percent. SOURCE: Reprinted (more...)

The radon exposure potential within the boundaries of the Commonwealth of Virginia is highest in the eastern Piedmont along the Fall Line, the western Piedmont, and the Valley and Ridge province (USEPA, 1993a; VA DMME, 2006) (Figure 5.5). In a 1991-1992 statewide survey of 1,156 homes performed by the USEPA and the Virginia Department of Health, the average radon concentration was 2.7 pCi/L, with 17.6 percent of homes exhibiting screening radon concentrations above 4 pCi/L. The maximum residential radon screening measurement recorded was 81.5 pCi/L, in a home in Danville, Pittsylvania County, Virginia (USEPA, 1993a). The Virginia Department of Mines, Minerals and Energy (VA DMME) indicated that it is, “reasonable to assume that radon would be a significant problem over the massive uranium deposits in Pittsylvania County” (VA DMME, 2006). Note that the existing elevated residential radon concentrations in Pittsylvania County, Virginia, are not related to mining activities, but rather are attributable to the strong radium-226 source strength in that geographical area.

FIGURE 5.5
Radon zones in Virginia; red zones indicate high radon potential, orange zones indicate moderate radon potential, and yellow zones represent low radon potential. SOURCE: VA DMME Division of Geology and Mineral Resources (; accessed September 26, 2011.) (more...)

Radon Risk Estimates
The NRC’s BEIR VI Committee estimated—based on projections (i.e., interpolations from the radon-exposed underground miner studies they examined)—that 18,600 lung cancer deaths occur each year in the United States from nonoccupational exposures to radon decay products (NRC, 1999b). The USEPA updated the risk estimate in 2003, projecting that of the total 157,400 lung cancer deaths that occurred nationally in 1995, 21,100 (13.4 percent) were radon related (USEPA, 2003). The USEPA also estimated that the risks from lifetime exposure at the radon action level of 4 pCi/L are 2.3 percent for the entire population, 4.1 percent for individuals who smoked cigarettes at some time in their lives, and 0.73 percent for individuals who never smoked. The BEIR VI committee and the USEPA note that, although it is not possible to eliminate radon exposure completely, projections from miner-based studies to the residential setting indicate that approximately one-fourth of the radon-related lung cancers could be avoided by lowering radon concentrations in all U.S. homes to no more than the USEPA’s radon action level of 4 pCi/L (NRC, 1999b; USEPA, 2003).

As noted above, risk estimates for protracted exposure to radon decay products among the general public are based on the indirect evidence from radon-exposed miners and are subject to multiple uncertainties. For example, the cumulative radon exposure values for miners are often many times higher than those for the general public, the exposure rate is higher for miners than for the general public, the breathing rate and type of breathing (i.e., more oral breathing by
miners as opposed to nasal breathing) often differs between miners and the general public, differences in the size of particles to which the radon decay products attach, sex difference (i.e., most miners are men), age differences (i.e., miners generally are over age 18), higher rates of smoking among miners, and the greater exposure to other lung carcinogens among miners. Because of the uncertainties in projecting miner-based risk estimates to nonworker populations, and in order to obtain direct information on the risk posed by residential radon exposure, numerous investigators have performed case-control epidemiological studies that compared the concentration of radon in the homes of cases (i.e., individuals with lung cancer) to the concentration of radon in the residences of age- and sex-matched individuals without lung cancer. Summaries of the findings from 22 major residential case-control studies are available elsewhere (Darby et al., 2005, 2006; Krewski et al., 2005, 2006). Although the risk estimates for protracted radon exposure and lung cancer incidence varied among the studies, 19 of 22 exhibited increased risk estimates at an average long-term radon exposure that was even below (i.e., 2.7 pCi/L) the USEPA's Radon Action Level of 4 pCi/L (Lubin, 2010). Pooling of residential radon studies performed both in North America and Europe (Darby et al., 2005, 2006; Krewski et al., 2005, 2006) yielded quantitative risks estimates that are very comparable to those projected from the radon-exposed miner studies. The pooled epidemiological analyses yielded statistically significant findings for the relationship between protracted radon exposure and lung cancer at concentrations even below the USEPA's Radon Action Level. These findings further support the need to reduce radon exposures for workers involved with uranium mining and processing to as low as reasonably achievable (ALARA).

Consistent with the prevalence of exposure and its adverse effects, residential radon decay product exposure is believed to be the second leading cause of lung cancer overall, the primary cause of lung cancer among individuals who have never smoked, and the leading environmental cause of cancer mortality in the United States (USEPA, 2009, 2011b; Lubin, 2010; Field, 2011). Moreover, even relatively low-level residential radon concentrations (i.e., less than 2 pCi/L) present a numerically substantial (i.e., on the order of 10,000 excess deaths per year) population-based health risk because of the large population exposed in the United States. To reduce the lung cancer deaths from residential radon exposure by 50 percent, the radon concentration in all the homes in the United States would have to be lowered to ≤ 2 pCi/L (NRC, 1999b; Lubin, 2010). As noted in the USEPA's Physician’s Guide for radon (USEPA, 1993b),

Recognizing that radon is a significant public health risk, scientific and professional organizations such as the American Medical Association, the American Lung Association, and the National Medical Association have developed programs to reduce the health risks of radon. The National Institute for Occupational Safety and Health (NIOSH) reviewed the epidemiological data and recommended that the annual radon progeny exposure limit for the mining industry be lowered (NIOSH 1987).

**Radon Releases from Uranium Mining and Processing**

While radon is ubiquitous in the Earth’s crust, it is generally more concentrated in or near uranium mining and processing operations. Communities living near uranium tailing piles may have increased environmental radon levels (ATSDR, 2008). Sources of radon at uranium mining and processing sites include tailings, uranium ore, waste rock, open cuts or underground mines, the processing facility, and water retention ponds (Mudd, 2008). In many cases, tailings represent the predominant source of radon emission (i.e., off-gassing) from a mining site. Radon emanation is heavily influenced by the specific material’s radium activity, moisture content, porosity, and density (Mudd, 2008). The Code of Federal Regulations (10 CFR § 20.1301) restricts the total effective dose (TED) to individual members of the public from licensed processing facility operations to less than 100 mrem per year. Radon and its decay products are specifically excluded from compliance with the dose criteria outlined in the Code of Federal Regulations (40 CFR § 190.10a). However, 40 CFR Part 61, Subpart B limits the effective dose from radon decay products to 10 mrem/yr for members of the public.
On November 10, 2011, a USEPA contractor, S. Cohen & Associates (SC&A), provided the agency with modeled data for radionuclide emissions from processing facility tailings and risk estimates to the population under various scenarios. One of the sample exposure scenario sites selected by SC&A (2011) included a site in Virginia, and SC&A indicated that this site was chosen because of the large number of uranium deposits in Virginia. Specifically, Culpeper County, Virginia, was selected as the Eastern Generic sample study site within Virginia, “because of its high population density and its past experience as a uranium mine lease site.” The location was also selected to exclude members of the general population living within 1 km of the site. The model used in the report included the following input data: an estimate of the 2010 population living within 80 km of the Culpeper County, Virginia site, meteorological data at the site, and an estimate of the amount of radon released on a yearly basis from the site. The maximum estimated radon release rate of 1,750 Ci/yr from the White Mesa, Utah, mine and processing facility tailings site was used as a surrogate measure of the maximum release rate for the Culpeper County site. Based on the estimated release rates and the standard modeling performed by the USEPA contractor, the reasonably maximally exposed individual (RMEI) (i.e., member of the public within 80 km expected to receive the greatest exposure to radon decay products) was estimated to receive a dose of 28 mrem/year, with a 1.6 in 100,000 chance of developing a latent cancer fatality; while the maximum estimated population dose living within 80 km of the site was 200 person-rem/yr, with a 1.4 in 1,000 chance of developing a latent cancer fatality.

The extent to which the estimated radon release rate assumed by SC&A (2011) for the Culpeper County site would approximate potential radon releases from tailings and waste rock in Virginia is not known. Radon emission rates from various types of underground mines and processing facilities are presented in other reports (e.g., UNSCEAR, 1993; Mudd, 2008). The NRC (1986) reviewed existing information regarding the potential for radon and radon decay particle release from uranium tailings, and noted that the relationship between the concentration of radionuclides in a tailings pile and the radon flux from a pile is complex and, moreover, the relationship has considerable variability by site. Although modeling can serve a role, overly heavy reliance should not be placed on general models of radon emission and dispersion without site-specific information. More recently, UNSCEAR (2009) also recognized that significant deviations of selected model parameters (e.g., population density, emission rates) are possible, and that while careful management of tailings in the future would be expected, variations in management of tailings could result in increases or decreases of estimated exposures by at least an order of magnitude. In concluding their section on mining and processing dose estimates, the UNSCEAR (2009) report indicates that, “Further surveys of site-specific conditions would be useful to establish realistic parameters for the worldwide practice” (UNSCEAR, 2009, p. 182).

Because of the complexity and variability of factors that affect off-site releases (e.g., site characteristics, deposit type), as well as the variations in assumptions used by the investigators, the magnitude and geographic distribution of off-site exposure to radon and its decay products are difficult to quantify (UNSCEAR, 1993, 2009; Chambers, 1998a, b; Frost, 2000; Mudd, 2008). Accurate radiation exposure estimates specific to the Commonwealth of Virginia that could be used for reliable modeling, as well as risk estimates for off-site populations (i.e., non-mine or nonprocessing facility workers), would require information (e.g., source data, site characteristics, and operational specifics) that does not currently exist. Clearly, additional site-specific research would be required to develop baseline data and methods to assess the long-term potential for releases of radon and its decay products to the population in the adjacent environment. Compared with radon progeny exposure leading to alpha particle exposure, off-site gamma radiation exposure is generally only a concern for individuals in close proximity to uranium tailings.

**URANIUM HEALTH HAZARDS**

As noted previously, among the three naturally occurring uranium isotopes ($^{238}$U, $^{235}$U, and $^{234}$U), $^{238}$U exhibits greater than 99 percent relative abundance (ATSDR, 2011). Long-lived
Uranium Absorption, Distribution, and Excretion

Internal exposure to $^{238}$U can occur via inhalation, ingestion, or entry through a cut or other disruption to the skin. Dermal absorption of soluble forms of uranium through intact skin is also possible, but this pathway of exposure is not considered significant. The rate of inhalation and transport of airborne uranium within the body depends on both the particle size of the aerosol and the solubility of the uranium compound. For example, soluble forms of uranium (e.g., UF$_6$, UF$_4$, and UO$_2$(NO$_3$)$_2$) have moderate rates of absorption entering the bloodstream, followed by transportation to the kidneys and other organs (IARC, 2001). The majority (over 60 percent) of uranium in the blood is filtered in the kidneys and excreted in urine within 24 hours. Uranium compounds that are less soluble (e.g., UO$_2$, U$_3$O$_8$) tend to be retained in the lungs and tracheobronchial lymph nodes for many months or years, thereby creating an increased cancer risk from alpha particle exposure.

There is no conclusive evidence that uranium produces cancer in humans (ATSDR, 2011). Although uranium has not formally been classified as a human carcinogen by the International Agency for Research on Cancer (IARC), uranium-238 is considered a Group 1 carcinogen under the category of alpha-particle-emitting, internally deposited radionuclides (IARC, 2011).

Gastrointestinal absorption of uranium, with reported absorption rates that vary widely from 0.1 percent to 31 percent (Hamilton, 1972; Wrenn et al., 1985, 1989; Harduin et al., 1994; Limson Zamora et al., 2003), is affected by the solubility of the uranium ingested and previous food consumption (Sullivan et al., 1986; La Touche et al., 1987). The International Commission on Radiological Protection-69 (ICRP, 1995) model for the fate of uranium after it enters the bloodstream is based on both human and animal data. The model predicts that 12 percent of the uranium in the bloodstream is apportioned to the kidneys, 2 percent to the liver, 15 percent to bone, 1 percent to red blood cells, 30 percent to soft tissues with rapid turnover, 6.7 percent to soft tissues with intermediate turnover, and 0.3 percent to soft tissues with slow turnover rates. The ICRP-69 model also predicts that 63 percent of the uranium that enters the blood is promptly excreted in urine via the bladder, as noted previously (Royal Society, 2001). According to the ICRP (1995), of the uranium that is retained, 66 percent is deposited longer term in the skeleton, 16 percent in the liver, 8 percent in the kidneys, and 10 percent in other tissues. IARC (2001) notes that a portion of uranium deposited in skeletal bones may remain there for over 20 years, which poses a risk for cancer of the bone and leukemia. Additional information on uranium occurrence, routes of exposure and entry into the body, deposition, and clearance is presented in detail elsewhere (ICRP, 1991, 1995; Leggett, 1994; NRC, 1999b, 2008b; Royal Society, 2001; Brugge et al., 2005; ATSDR, 2011).

Adverse Health Effects of Uranium

Uranium has no known normal metabolic function or essential human elemental requirement. It has been shown to cause chemical toxicity, and because it emits predominantly alpha particles, uranium is a suspected human carcinogen (ATSDR, 2011). The Agency for Toxic Substances and Disease Registry (ATSDR) recently published a detailed review of adverse uranium health effects (ATSDR, 2011), concluding—as have other reviews—that the primary effect from uranium exposure is renal toxicity. Soluble uranium compounds and uranium compounds that
become soluble by forming a bicarbonate complex in the blood can produce impairment of the proximal tubules (ATSDR, 2011); renal toxicity associated with high doses of uranium can lead to death. However, if the renal tubular epithelium is damaged by acute or chronic lower level exposures, it can usually regenerate. ATSDR (2011) did not identify any human studies that assessed health effects of dermal exposure, as opposed to ingestion, of uranium.

The USEPA has set a maximum contaminant level of 30 µg/L for uranium in drinking water, as well as a maximum contaminant level goal of no uranium in drinking water, based primarily on its chemical toxicity (USEPA, 2012a). Several epidemiological studies have used aggregate data (Mao et al., 1995; Limson Zamora et al., 2009; Seldén et al., 2009) to examine potential adverse health effects of chronic exposure to uranium in drinking water. These studies reported renal effects possibly related to the uranium exposures, but no dose-response findings were observed. Results from the aggregate-based studies (i.e., studies that examine aggregated data at the population level and lack information on disease or exposure for a specific individual) need to be interpreted cautiously and are generally used for hypothesis-generating purposes, rather than hypothesis testing, because of their potential for biases due to their lack of individual-level information on both exposure and disease. Numerous epidemiological studies of miners and processors (discussed below) have noted adverse renal effects associated with uranium exposures from inhalation. ATSDR (2011) also noted that several of these studies analyzed potential reproductive effects (i.e., damage to sex chromosomes) related to inhalation of uranium, but provided limited empirical evidence of such a relationship.

Experimental animal data concerning systemic adverse health effects from inhalation, ingestion, and dermal absorption of uranium are more robust. Animal studies have provided a rich dataset that characterizes the renal toxicity (e.g., reduced glomerular filtration rate, renal enzyme changes) of uranium under controlled experimental conditions (Vicente-Vicente et al., 2010). Nonspecific neurological symptoms also have been observed in animals that have been exposed dermally or via inhalation of high concentrations of uranium (ATSDR, 2011). Of note, despite its renal toxicity, there are no reported studies of ototoxicity from uranium in experimental animals, although this question could be highly relevant to uranium and noise co-exposed workers.

**Occupational Exposures and Health Effects of Uranium**

In part because of the low specific activity of uranium, the renal health effects and potential respiratory effects of uranium exposure are most often attributed to the chemical properties of uranium (ATSDR, 2011). The primary clinically observed health effect related to uranium exposure is chemical-induced nephrotoxicity. The first observations concerning the nephrotoxicity of uranium began in the 1800s, when uranium was intentionally administered as a medical treatment for diabetes and other diseases (Hodge, 1973). “Uranium nephritis” was described as early as 1915 (Oliver, 1915). Although the causal link between nephrotoxicity and uranium exposure was established many years ago, few epidemiological studies with rigorous exposure assessments and sufficient sample sizes have been performed that examine the risk posed by uranium to workers in the uranium mining or processing industry. Additional epidemiological data relevant to this question among uranium miners and processors will be provided in a later section on silica exposure.

Assessing the causal relationships between uranium exposures in miners and adverse health outcomes presents a challenge because of confounding by occupational exposures to radon decay products, silica, and diesel exhaust. Uranium miners clearly have higher all-cause mortality rates compared with selected reference populations, and do not—as is the case with the majority of other retrospective occupational mortality studies—exhibit the tendency for workers to be healthier than the general reference population (i.e., the “healthy worker effect”). Boice et al. (2008) attributed this excess mortality to exposure to radon decay products, rather than uranium itself. In addition, data on lifestyle factors that will affect mortality risk (i.e., confounders), such as smoking and alcohol consumption, have not been available in many of the epidemiological studies for these cohorts, which precluded adjustment of these factors. As pointed out by the Royal Society (2001) report, only a limited number of epidemiological studies have been
performed examining the adverse health outcomes of workers who work with uranium and even fewer studies have looked at nonfatal health outcomes. As noted previously in regard to extrapulmonary cancer risk from radon decay product exposure, the ability to observe work-related health effects is reduced when epidemiological studies rely solely on death certificates as a measure of health outcomes.

The potential for exposure to uranium, as noted previously, is highest during processing. Several retrospective cohort mortality studies of uranium processing workers where exposure to radon decay products is expected to be less than that of underground miners, although not negligible, have been performed. These limited studies have failed to establish a consistent pattern of excess mortality among uranium processing workers (Archer et al., 1973a; Pinkerton et al., 2004; Boice et al., 2008). Findings from these studies related to silicosis are discussed in a following section. These studies, especially Archer et al. (1973a) and Pinkerton et al. (2004), should be interpreted with caution because of the limited sample size and lack of individual measures of exposure and smoking data.

Other sources of epidemiological data are important for assessing the potential health effects of occupational exposure to uranium itself. These data sources are needed because adverse health effects seen in mortality studies of underground uranium miners are dominated by radon-related exposures, and because studies of uranium processors have been limited by small sample sizes and poor exposure assessment. Thus, findings from the wider uranium industry are particularly relevant to the question of potential uranium-specific adverse health effects from uranium mining and processing. The findings from two systemic analyses of multiple epidemiological studies are described in the following text. These two analyses, by the Royal Society and the National Research Council, are summarized in this report because—despite their many limitations—they are the most scientifically rigorous data analyses that have been performed to date on this subject and often serve as the predominant findings referenced indicating that uranium exposure to workers does not infer a substantial adverse health risk.

The meta-analysis (i.e., an analysis that represents a combination of other analyses) performed by the Royal Society (2001) is particularly noteworthy. It included 14 studies (11 from the United States and 3 from the United Kingdom), and examined the adverse health effects associated with work in the wider uranium industry—including uranium processing, uranium enrichment, uranium fuel fabrication, phosphate fertilizer production, and employment at other uranium-contaminated sites. This review included approximately 120,000 workers with 33,000 observed deaths. Health outcomes included all-cause mortality, deaths from 13 specific cancer types, and from genitourinary disease as a primary cause of death. The authors of the meta-analysis noted selected risk elevations in individual studies, including increases in overall mortality (Frome et al., 1997; Ritz, 1999), kidney cancer (Dupree-Ellis et al., 2000), Hodgkin’s disease and bladder cancer (McGeoghegan and Binks, 2000), lung cancer (Frome et al., 1997; Ritz, 1999), prostate cancer (Beral et al., 1988), and a statistically significant dose-response relationship between internal lung dose and upper aerodigestive tract cancers as well as haematopoietic and lymphatic cancers (Ritz et al., 2000). The meta-analysis combining these studies nonetheless did not observe statistically significant increases in all-cause mortality, all cancer mortality, or mortality due to specific cancers, or genitourinary disease (a category that included kidney dysfunction). As the Royal Society (2001) researchers pointed out, the meta-analysis had numerous limitations, including lack of uranium exposure data, potential double counting of subjects that were common to more than one study, inclusion of subjects with little or no uranium exposure, lack of exposure information on toxicants other than uranium, and the tendency for workers to be healthier than the general reference population (i.e., healthy worker effect). Because of these limitations, the authors of the Royal Society report concluded that—based on the meta-analysis—it would not be justified to infer that adverse health effects associated with occupational uranium exposures do not exist.

The National Research Council (NRC, 2008b) also performed a review of uranium worker epidemiological studies that overlapped somewhat with the Royal Society’s (2001) earlier review. The NRC (2008b) report also noted many of the same limitations of these studies,
including the lack of uranium exposure data, limited information on potential confounders, and
the potential for a healthy worker effect blunting the ability to observe adverse health effect
associations. This meta-analysis of mortality outcomes among nearly 110,000 workers also
detected no significant excess mortality due to cancer or renal disease. The NRC reported that the
findings suggested that occupational exposure to uranium compounds does not support a
conclusion that uranium compounds had a highly carcinogenic or nephrotoxic effect in this
combined study population. Nonetheless, the NRC (2008b) report concluded that an increased
risk of lung cancer due to the inhalation of uranium particulates cannot be ruled out, especially
because alpha particles are known to be emitted by such dusts. ATSDR (2001) agreed that the
existing studies of uranium workers do not provide compelling evidence that occupational
exposure to uranium dust causes lung cancer. Nonetheless they note—reiterating what other
researchers also have stated (Archer et al., 1973b; Howe et al., 1986)—that because of the
concurrent exposure to radon and thoron progeny, the studies of such working populations are
inadequate for assessing the carcinogenic potential of uranium.

Other important information on uranium-associated adverse health outcomes in human
populations is limited, especially for environmentally exposed individuals (ATSDR, 2011;
Brugge and Buchner, 2011). This includes information regarding neurological effects,
immunotoxicity, developmental toxicity, reproductive toxicity, genotoxicity, and, finally, whether
children are more susceptible than adults to such effects if indeed they are present.

RADIUM HEALTH HAZARDS

Radium is a naturally occurring radioactive metal with chemical characteristics similar to
calcium. As noted previously, there are four naturally occurring isotopes of radium, including
radium-228 (228Ra), radium-226 (226Ra), radium-224 (224Ra), and radium-223 (223Ra).
Radium-224, -226, and -228 and their decay products are classified as Group 1 carcinogens (i.e.,
known carcinogenic to humans) (IARC, 2001). Because of the relatively short radioactive half-
lives of 224Ra and 223Ra of 4 and 11 days, respectively, as well as their lower relative abundance
as compared to 226Ra, these isotopes carry less occupational health risk than 226Ra with its
1,600-year half-life (Figure 5.1). In addition, 228Ra, produced in the 232Th decay chain, is
generally not considered a major health concern in uranium tailings as compared to 226Ra,
because of its lower relative abundance and much shorter half-life of 6 years (USEPA, 1983).

During uranium processing, a large percentage of the uranium is removed, leaving the majority
of the decay products in the tailings. Thorium-230 (230Th) is the immediate decay product
following 234U and is the longest-lived (i.e., radioactive half-life of 77,000 years) decay product
remaining in the tailings. The 230Th provides a constant source of 226Ra (Figure 5.2), which in
turn decays into radon (as previously discussed). In addition to the production of radon from
226Ra during mining and processing operations, 226Ra decay products (i.e., bismuth-214 and
lead-214) (Figure 5.2) in the waste or tailings can produce significant gamma radiation hazard
(USEPA, 1983) both in the processing facility as well as near waste areas or tailings. Gamma
radiation has the potential to increase the risk of cancer to varying degrees for most tissues and
organs (USEPA, 2011a). Because of its similarity to calcium, ingested 226Ra tends to concentrate
in bone. The International Commission on Radiation Protection estimates that about 15 to 21
percent of ingested radium is absorbed (ICRP, 1993).

Existing understanding of the potential adverse health effects related to ingested 226Ra is based
primarily on studies of radium watch dial painters who worked with radium in the early 1900s
(Martland and Humphries, 1929). These painters would routinely place the paint brush in their
mouths in order to get the fine tip needed to paint the watch dials, which led to significant
ingestion of 226Ra which was followed by systematic absorption and subsequent deposition into
the skeletal system. The primary adverse health effect in this group related to the high degree of
226Ra ingestion was bone cancer (i.e., osteosarcoma) (Rowland et al., 1978; Stebbings et al.,
1984; Rowland, 1994). The USEPA also noted that in addition to bone cancer, protracted
exposure to inhaled or ingested 226Ra is linked to increases in lymphoma, leukemia, and aplastic
anemia (USEPA, 2011c). Studies directly assessing the risk posed by $^{226}\text{Ra}$ to miners and processors are lacking, in large part because of the inability to separately assess the effects of exposures to $^{226}\text{Ra}$ relative to exposures to other radionuclides.

Along with exposure to radon decay products, inadequate containment of uranium tailings most likely represents the highest potential source of radiation exposure, related to uranium mining activities, to the general public. Landa and Gray (1995) note that “due to its high radiotoxicity and affinity for accumulating in bones,” $^{226}\text{Ra}$ is generally the uranium daughter product of “most concern in hazard assessments of water supplies and food chains” associated with uranium mining tailings. The stability of uranium mine tailings is an extremely important focus of industry best practices (see Chapter 8). In 1976, the USEPA set a maximum contaminant level (MCL) for a combined $^{226}\text{Ra}$ or $^{228}\text{Ra}$ concentration of 5 pCi/L in public water supplies. The USEPA estimated that if 10,000 individuals consumed 2 liters water each day at the MCL for 70 years, one additional death would be caused (USEPA, 2011c).

**Radiation-Related Adverse Health Effects in the General Population Living Near Uranium Mining or Processing Sites—Limitations of Epidemiological Studies**

The potential off-site (i.e., non-occupational) adverse health effects related to modern mining practices remains an area of great uncertainty. Several well-executed ecological studies have been performed that attempted to identify increases or decreases in mortality or cancer incidence related to exposures from uranium mining or processing operations (Boice et al., 2003, 2007a,b, 2010). The earliest study by Boice and colleagues (2003) compared the rates of cancer based on death certificates from Karnes County in Texas, which had three processing facilities and over 40 mines that were in operation for various periods between 1961 and the early 1990s, to mortality-based cancer rates in “control” counties as well as to the Texas and U.S. mortality-based cancer rates. The researchers reported that no unusual patterns of cancer mortality were detected, suggesting that the uranium mining and processing operations did not contribute to increased cancer rates in Karnes County.

Boice and colleagues used a similar study design to the Karnes County, Texas, study to examine the mortality and cancer risk posed by past uranium mining and processing operations in Montrose County, Colorado (Boice et al., 2007b) and for another study to examine the health risks for a population living near a uranium processing facility in Uravan, Colorado (Boice et al., 2007a). Except for an increased risk of lung cancer among males that was attributed to occupational radon exposure (i.e., working in mines) by the authors, no statistically significant increases in cancer or mortality rates were detected. A more recent study by Boice et al. (2010) investigated whether incident cancer or mortality rates were elevated in the population living near uranium mining and processing activities in Cibola County, New Mexico. The researchers did not find any evidence that the operation of the uranium mines and processing facilities increased the cancer or mortality rates for the nearby population.

Boice et al. (2007b) pointed out that definitive causal inferences cannot be established from these geographical correlation studies. Geographical correlation studies are hindered by the lack of individual-level exposure data, and so everyone within a certain region is assigned the same exposure. In addition, other risk factors (e.g., cigarette smoking, alcohol consumption) are also based on grouped data, and so adjustment for confounding at the level of the individual is impossible (Brugge and Buchner, 2011). Although epidemiologists rely on the use of geographically based studies to generate hypotheses, ecological epidemiological studies lack the ability to test hypotheses. As stated in epidemiological terms by Morgenstern (1995), “Despite several practical advantages of ecologic studies, there are many methodologic problems that severely limit causal inference, including ecologic and cross-level bias, problems of confounder control, within-group misclassification, lack of adequate data, temporal ambiguity, col-linearity, and migration across groups.”

**PRINCIPAL URANIUM MINING AND PROCESSING EXPOSURES OTHER THAN RADIONUCLIDES**

https://www.ncbi.nlm.nih.gov/books/NBK201047/
Silica

Silica overexposure is a potential hazard whenever resource extraction such as mining (underground or open-pit) or ore processing involves silica-bearing materials. The geology of uranium-bearing ore deposits is such that typically concomitant silica exposure cannot be avoided during mining and processing uranium. Many of the known uranium deposits in Virginia occur in granites that contain silica.

The primary health-effect-relevant route of exposure for silica is via inhalation. The concentration of silica dust that is crystalline (as opposed to amorphous) and in the respirable range (particles up to 10 microns can reach the airways, and particles smaller than 5 microns penetrate deeply into the lungs) is considered to be the most important exposure metric, and health protective standards are recommended on the basis of these attributes (e.g., NIOSH, 1978). The specific sources of silica dust generation in mining and processing operations can include drilling (including test bores); blasting; shotcrete formulation (this can include the addition of fine particulate “silica fume”) and application to mine surfaces; earth-moving, excavating, rock hauling and transport; crushing, processing, and sifting; and in the handling of tailings or mining debris. Other occupational activities that are nonspecific to mining or processing, but which are likely to involve silica exposure in conjunction with various phases of a large mining and processing project, include concrete finishing, sandblasting, and infrastructure construction (e.g., road building). Any mechanical operation that breaks apart silica-bearing materials not only can generate respirable dust, but may also produce freshly fractured silica—a form of the mineral believed to be of particularly high biological activity.

There are multiple silica-caused adverse health outcomes, predominantly—but not exclusively—disorders of the respiratory tract. Chief among these is silicosis, a progressive, life-threatening, fibrotic lung disease. The lung tissue changes that are the hallmarks of this disease are distinct to silica exposure. Pathological examination of lung specimens, however, is not required to make a clinical diagnosis of silicosis, which is frequently based on the occupational exposure history, lung function studies (such a measures of airflow, lung volumes, and the diffusing capacity), and radiographic assessment (which can include computerized tomographic [CT] imaging).

Silicosis has been endemic to mining and quarrying operations involving silica-containing materials, including among workers in uranium operations located in multiple regions of the world. One of the largest occupational cohorts of silica-exposed uranium workers derives from the “Wismut” operation in the former East Germany, with an estimated labor force of 400,000 (Schröder et al., 2002). This cohort has already been alluded to in the previous section on radon. As is noted in the report of that study by Schröder and coinvestigators, working conditions were reported to be particularly poor between 1946 and 1956; operations ceased in 1990. By 1999, silicosis had been recognized among more than 16,000 former workers (this total also includes silicosis complicated by concomitant tuberculosis).

Other studies covering the same period have documented elevated risk of silicosis mortality in cohorts of uranium workers. Such risk is typically expressed as the ratio of mortality standardized to the general population. The standardized mortality ratio (SMR) is a basic metric of epidemiological risk derived from mortality studies such as those done among uranium mining and processing cohorts. A recent report of further follow-up of the Colorado Plateau cohort (a large group study of former uranium miners from the U.S. Southwest) added 15 years of additional mortality follow-up data for the period 1991 through 2005, supplementing previous data for 1960-1990 (Schubauer-Berigan et al., 2009). This cohort has also contributed to the epidemiology of radon health effects discussed previously. For silicosis deaths, the SMR for whites in the 1991-2005 period was 64.7 and for American Indians was 33.3 (even higher than the elevated point estimates in the earlier period of 42.5 and 24.2, respectively). In total, there were 54 silicosis deaths, although there were 37 classified as other or unspecified pneumoconioses.

A large French cohort study of uranium miners has also reported silicosis mortality over a comparable time period (Vacquier et al., 2008). In that analysis, the SMR was 7.12, based on 23
silicosis deaths among more than 40,000 miners. This SMR point estimate, although elevated and statistically significant, is far lower than the U.S. estimated risk based on Colorado Plateau data. The lower estimate from France could represent statistical variation or could reflect a higher general population death for silicosis in France, reducing the SMR because the referent value used in the ratio was higher.

Another relevant analysis is that of a cohort of more than 4,000 Czechoslovakian uranium miners who had worked between 1948 and 1959 (Tomášek et al., 1994). In that cohort, among those with 25 or more years of follow-up, the SMR for nonspecified chronic respiratory disease (which would subsume silicosis, 60 deaths in total) was modest—but statistically significant—at 1.6 ($p < 0.001$).

Data on silicosis among uranium process workers, as opposed to uranium miners, are more limited. An updated analysis of 1,484 employees of seven uranium processing facilities in the Colorado Plateau—with nearly 60 years of follow-up from 1940 through 1998—presents a relatively robust database because of the size of the cohort combined with the duration of follow-up (this cross product is summarized as person-years; in this analysis, 50,000 person-years of follow-up). This cohort study is distinct from the miner cohort already described above, but was alluded to in the previous discussion of uranium health effects among processors. This analysis reported a statistically significant increased risk of all nonmalignant respiratory disease (SMR 1.43; 95 percent CI of 1.16-1.73 based on 100 observed deaths) and, within that category, an increased mortality risk for pneumoconiosis, including silicosis (SMR 1.68; 95 percent CI of 1.26-2.21) (Pinkerton et al., 2004). A study of a smaller subset of processors in another mining-processing cohort from New Mexico (718 who were included were likely to have been employed only as process workers without underground mining experience, also with up to 50 years’ follow-up) did not observe a statistically significant mortality risk for all nonmalignant respiratory disease, although the SMR point estimate was elevated (1.22; based on 24 observed deaths); pneumoconiosis mortality risk was not reported separately (Boice et al., 2008). Of note, the pooled estimate of respiratory nonmalignant disease, which can be derived by taking the published values available from these two studies and adding them together (yielding 124 observed deaths due to non-cancer-related lung disease and with only 89.9 such deaths expected based on population rates), yields an SMR of 1.38 (with an associated statistically significant 95 percent CI of 1.14-1.65, using a conservative statistical Poisson assumption of such deaths being rare events). This excess rate indicates that the risk of death from nonmalignant respiratory disease among these U.S. uranium processing workers was increased by nearly 40 percent.

Silicosis, in its classic form, is a chronic process that becomes clinically manifest more than a decade after initiation of first exposure. For example, an analysis of length of employment and onset of silicosis among Chinese workers exposed to uranium dust from 1956 to 2002 reported a mean time elapsed of 14 ± 8 years until diagnosis (Wu et al., 2004). That analysis also reported that among uranium “geological prospecting teams” the duration to disease onset was on average 4 years less than the 14-year interval noted above (10 ± 6 years), an observation that could be related to exposure differences between miners overall compared with the subset that worked as prospectors.

Earlier onset, more progressive silicosis associated with more intense exposure is sometimes termed “accelerated silicosis.” Although accelerated and classic silicosis differ in time course, they are believed to represent the same underlying pathological process. In contrast, “acute silicosis” is a pathological entity that can arise relatively soon after initial silica exposure, is often rapidly fatal, and is pathologically distinct from classic silicosis. Acute silicosis was first well described pathologically in the 1930s (Chapman, 1932). Decades later, an unusual idiopathic disorder of the lungs, pulmonary alveolar proteinosis (PAP), was described (Rosen et al., 1958). Since then, a number of case reports and case series have underscored the role of silica in at least a subset of classic PAP cases. To further complicate categorization and the medical terminology that is applied to these disorders, this subset of disease is sometimes referred to as “silicoproteinosis.” For example, in a review of 139 cases of PAP, approximately one-half had occupational exposures to various dusts, and 10 were clearly silica-exposed (Davidson and
Macleod, 1969). A case report of a mine drilling machine operator whose exposure included work as a test driller may be relevant because it underscores that associated exposures need not be massive (Sauni et al., 2007). Acute silicosis or PAP specifically associated with uranium mining has not been reported.

As is implicit in data from the German uranium mining cohort that combines silicosis and silico-tuberculosis (Schröder et al., 2002), silica exposure increases the risk of tuberculosis infection. This effect is attributed to silica-related immune dysfunction, particularly in pulmonary macrophages. This risk applies to tuberculosis (i.e., infection with *Mycobacterium tuberculosis*), as well as to infection with strains of atypical mycobacteria that do not typically cause disease in immunologically intact individuals. Silico-tuberculosis refers to frank silicosis with tubercular coinfection. It has become well recognized, however, that silica exposure, even without radiographic evidence of silicosis, is associated with increased risk of tuberculosis potent enough to warrant medication prophylaxis for this disease (Fielding et al., 2011). In the Colorado Plateau cohort, tuberculosis-related deaths manifested statistically elevated SMRs in the first study period (3.44 and 2.40 for Whites and American Indians, respectively), but no tuberculosis deaths were noted among Whites in the second follow-up period and only two deaths among American Indians (SMR 2.39, not statistically significant) (Schubauer-Berigan et al., 2009).

In the Czechoslovakian cohort, the tuberculosis SMR for those with ≥ 25 years follow-up was 3.6 (*p* < 0.01) (Tomášek et al., 1994). The lymph node burden of silica following exposure may explain this pattern of risk, as observed in a recent analysis of a sample number of cases from a histopathological autopsy archive of deceased German uranium miners (Cox-Ganser et al., 2009). Among 264 cases (enriched for the presence of lung carcinoma), only 98 (38 percent) were free of a substantial parenchymal lung tissue burden of silica; among the remaining 166, 52 had silica involvement confined to the lymph nodes. In areas with high endemic infection, the triad of HIV, tuberculosis, and silica exposure has emerged as a major public health challenge (Rees and Murray, 2007). Thus, assessment of the potential health burden of silica exposure among any already marginalized population should take into account the potential for these combined, interactive risks. This is relevant to socioeconomic gradients of health among disadvantaged populations within Virginia.

Silica is a Class I recognized human carcinogen by IARC criteria (IARC, 1997). Review of the extensive epidemiological dataset supporting that designation is beyond the scope of this summary. It is noteworthy, however, that although the analysis of silica-associated lung cancer risk in mining operations was an important part of the IARC review, these data generally excluded uranium-exposed workers, because this occupation involves exposure to radon decay products, a potentially confounding lung carcinogenic exposure discussed above. The sole exception was the inclusion in the IARC review of a lung cancer case-control study of radiographic silicosis in uranium miners from the Colorado Plateau (see Samet et al., 1994; IARC, 1997, Table 19, p. 108). Based on 65 lung cancer cases and 216 controls and adjusted for radon co-exposure, silicosis was associated with a 33 percent increased risk of disease (because of the study design, this comparison does not yield an SMR), but with wide confidence intervals, meaning that this increased risk was not statistically significant at the 0.05 level (odds ratio [OR] 1.33; 95 percent CI of 0.31-5.72). Since that time, however, there has been increased interest in analyzing the combined risk of silica and radon to assess a potential interactive risk for lung cancer. An analysis of lung cancer risk among workers from two Swedish iron mines—one with substantial radon co-exposure and the other with negligible radon—recently addressed this question (Bergdahl et al., 2010). That study supported the presence of lung carcinogenic risks for both silica and radon in the mine with higher exposure to radon. Although the authors did not discuss interactive affects, the relative risk of lung cancer for the highest radon exposure category was 3.9 and for the highest silica category 1.9, while in the highest exposure cell for both, the estimated relative risk was 9.3 (e.g., greater than the 7.5 cross product and thus consistent with an effect that is more than additive alone). An analysis of lung cancer mortality in the German mining cohort observed an independent association with silica exposure, but also did not assess potential interactions (Taeger et al., 2008). That study, however, demonstrates the high
Silica exposure, with or without frank silicosis, has been associated epidemiologically and in case reports with selected extrapulmonary disorders, in particular, collagen vascular disease and renal disease, including disorders with overlapping end-organ effects such as scleroderma (Ranque and Mouthon, 2010). There are no reports specifically analyzing the relationship of silica exposure to these extrapulmonary outcomes among uranium miners. Of potential relevance, the extended cohort analysis of the Colorado Plateau miners observed a three- to fourfold increased SMR for acute glomerulonephritis (a potentially life-threatening form of kidney disease) among Whites in both time periods studied; no deaths for this cause were reported among American Indians (Schubauer-Berigan et al., 2009). An additional analysis of end-stage renal disease (ESRD) incidence (as opposed to mortality) observed an elevated point estimate for the standardized incidence ratio (SIR) for nonsystemic ESRD, which would include glomerulonephritis, for both Whites and American Indians (1.4 for each), but neither was statistically significant. A similar SIR approach was taken in the analysis of Colorado Plateau uranium processors. In that cohort, the risk for all ESRD was reduced (SIR = 0.71), but was increased for ESRD of unknown etiology (SIR = 2.73); in both cases the confidence intervals were wide and did not exclude no-effect (Pinkerton et al., 2004). As was noted in a previous section reviewing potential uranium extrapulmonary effects, the potential for renal toxicity from uranium itself also represents a potential mechanism for adverse health outcomes in these cohorts.

Finally, silica exposure is associated with chronic obstructive pulmonary disease (COPD). This association, however, extends beyond silica itself to inorganic dusts more broadly defined. Dust exposure in underground mining (silica and coal dust) was found to be strongly linked to COPD risk in a systematic analysis that included exposure levels and smoking adjustment (Oxman et al., 1993). Since that pivotal analysis, a large number of epidemiological studies have emerged consistently supporting a causal association between employment in dusty trades and increased COPD risk (e.g., Balmes et al., 2003; Blanc and Toren, 2007). Limited uranium mining and processing cohort data support the more generally observed association of dusty trades with COPD. In the Colorado Plateau cohort study, COPD mortality among Whites was significantly elevated in both time periods (SMR = 2.07 and 1.85, respectively), although the authors of that study speculatively attribute this finding to smoking rates among the cohort relative to the referent population data used (Schubauer-Berigan et al., 2009). In the Colorado Plateau uranium processor cohort analysis, emphysema mortality was elevated (SMR = 1.96, 21 deaths observed), but not chronic and unspecified bronchitis (SMR = 0.91; only 2 deaths were observed, indicating low study power to detect an association) (Pinkerton et al., 2004). Because of the latency between initial exposure and silica-related diseases such as silicosis, lung cancer, and COPD, the epidemiological data summarized above represent exposure conditions that span decades. It is presumed that improved working conditions leading to reduced exposure account for the decline in silicosis mortality observed in the United States in the 1970s to 1980s, but it should also be noted that the years of potential life lost (YPLL) due to silicosis have remained relatively flat from the 1990s onward (CDC, 2008). Indeed, silicosis deaths continue to occur in the United States, and mining remains a major contributor to the problem. For example, among 1,416 persons 44 years and older in United States dying from silicosis during 1968-2004, one in five with occupation and industry data available was known to be a miner; moreover, two-thirds lacked any employment information at all, such that the mining contribution may have been even greater (Mazurek and Attfield, 2008). Also, arguing against attenuation of risk, mining morbidity data for U.S. coal workers’ pneumoconiosis—for which there is better surveillance than silicosis—indicate that over the last decade, severe dust-related disease among miners has actually been increasing in the United States (Wade et al., 2011).

Silicosis has been linked to environmental sources of silica exposure among persons without a direct occupational risk. Moreover, ambient elevations in silica have been detectable downwind
from sand and gravel facilities, an exposure source that may be comparable to open-pit mining or rock hauling and dumping processes (Dhiraki and Holmén, 2002). Government regulators have carried out formal risk assessments of the potential public health effects of ambient silica; for example, in 2005 the California Environmental Protection Agency Office of Environmental Health Hazard Assessment adopted a “Chronic Reference Exposure Level” for silica that was driven by such ambient exposure concerns. Of note, this level was based on silicosis, rather than cancer risk (California EPA, 2005). A number of other states also have ambient silica standards, some of which are more stringent than California’s (Wisconsin Department of Natural Resources, 2011).

**Diesel Emissions and Diesel Particulate Matter**

Exposure to diesel emissions is particularly relevant to the potential health effects of uranium mining because such exposures are ubiquitous in modern mining environments. The use of diesel engines in metal and nonmetal mines in the United States expanded greatly in the 1960s and 1970s; even by 1976, it was estimated that 60 percent of underground noncoal mines had diesel equipment. Diesel engine exhaust contains respirable carbonaceous particulates that adsorb organic chemicals, including the polycyclic aromatic hydrocarbons benzo[a] pyrene and 1-nitropyrene. These compounds are carcinogenic in rodents when administered topically or by implantation, an effect that has been attributed to lung “overload” (Mauderly et al., 1987). Research has also suggested that inhalation of high concentrations of whole diesel exhaust causes destruction of pulmonary defense mechanisms and promotes the development of lung adenocarcinomas in animal models, whereas at lower levels of exposure that do not interfere with pulmonary clearance, diesel exhaust does not appear to be carcinogenic (Mauderly et al., 1990). This observation has been interpreted to suggest that one possible mechanism for carcinogenesis associated with inhalation of diesel emissions might be particle overloading, with subsequent inflammation of the lung, rather than the mutagenic effects of the organic fraction of diesel exhaust. The body of the evidence, however, does not support a threshold mechanism for diesel-associated carcinogenesis (California EPA, 1998).

The health effects of diesel exhaust have been studied in numerous epidemiological studies of occupational groups exposed to diesel emissions, notably operators of diesel powered railroad locomotives, heavy equipment vehicles, trucks, and some buses. This evidence for lung cancer is most suggestive and has been reviewed and summarized by numerous agencies and individuals, notably the National Research Council (NRC, 1981), the International Agency for Research on Cancer (IARC, 1989), Schenker (1980), Steenland (1986), Muscat and Wynder (1995), Bhatia et al. (1998), and Hesterberg et al. (2006). Although the 1981 NRC study found no evidence for the carcinogenic effect of diesel exhaust in the epidemiological studies, by 1989, IARC concluded—based on its review of the evidence—that diesel exhaust was “probably carcinogenic to humans.”

The most comprehensive and rigorous systematic review and meta-analysis of the epidemiological data was that conducted by Bhatia et al. (1998). Based on 23 case-control and cohort studies with adequate data for inclusion, these authors concluded that the epidemiological evidence supports a causal association between risk for lung cancer and exposure to diesel exhaust. The overall meta-estimate (weighted by precision of the individual studies) indicated an increased relative risk (RR) for lung cancer associated with occupational exposure to diesel exhaust of 1.33 (95 percent CI of 1.24-1.44). Importantly, this increased risk persisted for subanalysis by type of study, smoking status, and type of comparison group for cohort studies. A positive “duration of employment–response” pattern was observed in the studies that stratified by employment duration. Although there was considerable heterogeneity among the studies included, the overall consistency of results from the individual studies and the meta-analysis are consistent with a causal association.

Because a lot of mining equipment today is powered by diesel engines, diesel exhaust—including diesel particulate matter—poses risks for multiple adverse health effects among workers thus exposed. This is particularly relevant to the confined environment of underground mining, but is also relevant to open-pit processes as well as to exposure from diesel-powered
equipment used in other aspects of mine and process operations (e.g., heavy vehicle transport equipment). Moreover, in certain mining environments, simultaneous exposure to three occupational lung carcinogens—diesel, radon, and silica—may occur (Bergdahl et al., 2010). In addition to the potential risk of lung cancer, cardiovascular and acute and chronic pulmonary effects of diesel emissions have been documented (California EPA, 1998; USEPA, 2002).

### Physical Injury

Mining presents a large risk of traumatic injury. The most common causes of fatal injury include rock fall, fire, explosion, fall from height, entrapment, electrocution, and mobile equipment injuries. Fatal injury can also be caused by underground mine flooding, collapse of bulkheads, and caving failure. Fatalities have largely remained constant at around 40 per year from 1988 to 2007 (Figure 5.6) (NIOSH, 2011).

![FIGURE 5.6](image)


Both the number and frequency of nonfatal injuries have been declining (Figure 5.7), although there were still over 7,000 injuries in 2007 out of a population of approximately 255,000 miners (NIOSH, 2011). In underground mines, the largest injury category (~30 percent) over the 4-year period from 2003 to 2007 was materials-handling incidents. One way to judge the severity of nonfatal injuries is by the number of workdays lost; between 2001 and 2008 the average injury required 48 days of lost time before the worker could return to work, whereas between 1983 and 2000 the average number of lost workdays was 33. According to the U.S. Labor Department, the average number of lost workdays from injury for all other occupations was 8 days.

![FIGURE 5.7](image)


### Electrical Hazards

As mine operators decrease their use of diesel-powered equipment in underground mines—to decrease exposure to diesel fumes—the need for additional high-voltage electricity to power equipment increases, increasing the potential for electrical accidents. Statistics indicate that in mines, electrical accidents occur less frequently than other sources of traumatic injury, but they are disproportionately deadly with a fatality rate of 1 in 22. Electrical accidents accounted for over 6 percent of deaths in mines between 2000 and 2009; a recent review indicated that electrical injury ranks fourth as the cause of death. Compared with electrical injuries in other industries, mining is among the most dangerous. There are various causes of electrical injury in mines, and so a multifaceted approach is needed to mitigate electrical hazards. This would include engineering, administrative controls, protective equipment, and training to address human factors. One promising area of research involves a detection system for proximity to high-voltage lines.

### Noise and Vibration

#### Noise—Occupational Exposure

In 2007, the most recent year with data available on the NIOSH website (NIOSH, 2011), hearing loss or impairment was the second most prevalent reported illness among miners (after joint,
tendon, or muscle inflammation or irritation). Overexposure to loud noise can cause temporary hearing loss by damaging the nerve cells in the cochlea of the inner ear. Although it is possible to recover from this temporary hearing loss, repeated damage to the nerve cells causes permanent sensory neural hearing loss.

Noise is also a safety hazard, because warning bells, whistles, or shouts could be masked by loud noise. The mining industry has the highest prevalence of hazardous noise exposure of any major industry sector (Tak et al., 2009). In a study of 31,325 uranium miners in Germany from 1946 to 1990, hearing impairment was found in 4,878 miners (16 percent) (Schröder et al., 2002). From 1991 to 1999, when noise controls were presumably in place, 129 of 4,619 miners (3 percent) had hearing impairment (Schröder et al., 2002). Uranium mining- or processing-specific noise-induced hearing loss data for the United States are not available.

As with any industrial safety hazard, minimizing exposure to noise through engineering controls is the best solution. A substantial amount of literature has been devoted to the engineering controls that have been designed to minimize noise from equipment such as pneumatic drills, roof-bolting machines, and other heavy equipment used in hard-rock mines. Plots of noise contours from common mining equipment have been compiled so that miners can predict the noise environment adjacent to such equipment. In the processing operation, rubber can be used in the machinery for crushing and grinding. This minimizes noise exposure and also provides reduced maintenance of equipment. If engineering controls are not practical, administrative controls—such as limiting the amount of time spent in the noisy environment—are an alternative solution. The last resort, after all other noise control measures have been tried, is to equip workers with personal hearing protection.

Standard computer programs are available to track worker noise exposure. Since uranium is a neurotoxin, it is possible that exposure to uranium, along with exposure to noise, increases the probability of noise-induced hearing loss (Janisch et al., 1990). MSHA has regulations that govern worker noise exposure, codified in 30 CFR Part 62. These regulations parallel OSHA noise regulations and have a permissible exposure level, action level, and hearing conservation program. There are requirements for periodic audiometric testing of workers as well as training.

**Noise—Public/Off-site Exposure**

Health effects of noise in a community setting are based upon speech interference and sleep interference, rather than noise-induced hearing loss. When ambient sound levels reach a level of 50 decibels (measured on the A-scale to simulate the human hearing range), they begin to mask normal speech (USEPA, 1974; Peterson, 1980). A speaker will have to raise his/her voice to be heard at a distance greater than 2 ft, and the listener will have to concentrate to understand the speech. Telephone use will be difficult, and consonant sounds will be difficult to distinguish. These speech interference effects may be considered a nuisance in a typical residential setting, but may be more critical in an educational setting. Although studies of noise reduction and its impact on student test scores suggest that there is an impact of reducing noise exposure on high school student performance, more study is needed on elementary and middle school children’s performance (Eagan et al., 2004).

Sleep interference exhibits significant variability between individuals, and is linked to the subjective nature of the response. Much of the research on sleep interference has been conducted to study the impact of aircraft noise near airports (FICAN, 1997), and this indicates that a dose-response relationship can be drawn, despite the high degree of scatter in the data. To address the concern about sleep interference, model ordinances designed to protect the public against sleep interference generally require sound levels after 11 p.m. to be below 50 decibels, with an assumption that there will be 15 decibels of attenuation due to housing construction bringing the sound levels in sleeping rooms to 35 decibels. Although buildings can decrease sound levels by about 15 decibels through use of typical window construction, if the building is not air-conditioned and windows are opened during warm weather, sound is transmitted through open windows with no attenuation.
Noise—Physiological Effects

Noise can act as an environmental stressor, affecting the autonomic and hormonal systems, and causing elevated heart rate, blood pressure, and vaso-constriction. Prolonged exposure to noise can lead to chronic conditions such as hypertension and heart disease. The World Health Organization has reviewed the literature relating to physiological effects, and published community noise guidelines that cover all sources of noise (WHO, 1999).

At the federal level, USEPA or a designated federal agency regulates noise sources, such as rail and motor carriers, low noise emission products, construction equipment, transport equipment, trucks, motorcycles, and the labeling of hearing protection devices (USEPA, 2012b). Primary responsibility for regulating community noise rests with states or local governments. In Virginia, some local governments have passed noise control ordinances, which are enforced by code enforcement officers.

During exploration for uranium, it is likely that there would be limited off-site community impacts. During construction, however, there are likely to be more off-site impacts due to drilling and earthmoving, and transportation of construction equipment could affect neighborhoods. The choice of mining technique will affect the noise contour of a mining facility, with open-pit mining having more neighborhood noise impact than underground mining. Processing (grinding of the ore) is a noisy operation, but the off-site impact might be minimal if it is a fully enclosed operation.

Vibration—Occupational and Off-site

Sound is the transmission of vibration in the audible range—from 20 Hz to 20,000 Hz—but energy present in the range below 20 Hz can still cause adverse health effects. Whereas sound is airborne, vibration is primarily structure-borne. Sources of vibration include construction equipment, drilling equipment, blasting, and processing (crushing/grinding) equipment. The health effects of whole-body vibration include fatigue, insomnia, stomach problems, headache, and “shakiness” shortly after exposure. Vibration reduction can be accomplished by using isolation and by installing suspension systems between the vibrating source and the operator. People who operate hand-held vibrating tools can experience changes in tendons, muscles, bones, and joints, and vibration can also affect the nervous system. These effects are known as “hand-arm vibration syndrome,” and the symptoms are aggravated by exposure to cold. Ergonomic tool designs are available. Proper selection and maintenance of tools, and administrative controls, such as job rotation and rest periods, can reduce the adverse health effects (Nyantumbu et al., 2007; California State Compensatory Insurance Fund, 2011; Heaver et al., 2011).

Elastic waves emanate from any mining blast, causing ground vibration with potential to cause structural damage off-site. Most commonly, ground vibration causes lengthening of existing cracks. Without a structural failure leading to physical injury, however, this would not be classified as a human health effect. Humans can perceive potentially annoying vibration levels far below legal limits, but existing regulations are not intended to eliminate such annoyances.

MISCELLANEOUS HEALTH IMPACTS

There are additional potential exposures associated with uranium mining and processing beyond those individually described above. These can be categorized as either exposures arising generically out of mining (or at least the type of larger construction project that subsumes modern mining), or alternatively, exposures that are likely to be more specific to uranium processing and ore purification (although this latter category can overlap with certain related mineral extraction processes). Modern mining practices, in general, can be associated with a variety of hazards including—explosive gases; shotcrete; isocyanates; carbon monoxide; welding, metalworking fluids, and other maintenance-related exposures; and mold-related illness. In uranium processing, uranium extraction is a chemically dependent process, with certain commonly used substances (e.g., sulfuric acid) that are known to be hazardous, whereas
other process chemicals have uncertain hazard status. A short description of these miscellaneous potential exposures is presented below.

**Nitrogen Oxides in Explosive Gases**

Beyond noise and physical trauma, explosive use produces nitrogen oxides as residues. Nitrogen dioxide inhalation can cause severe acute lung injury and lead to chronic lung sequelae, in particular a syndrome of airway destruction called “bronchiolitis obliterans” (Blanc, 2010). Exposure is likely to be highest in enclosed-space applications (e.g., underground detonations).

**Shotcrete**

The term “shotcrete” refers to various formulations of concrete-related materials used in high-pressure spraying applications. Shotcrete can be little more than a simple mix of cement and aggregate, which is associated with skin and eye chemical burns in mine spraying (Scott et al., 2009). In modern underground mining applications, however, shotcrete has evolved into chemical-intensive formulations that can include “plasticizers” to facilitate flow, accelerators to promote setting, and retardants to temper the accelerator effects, together with added fiber and finely ground silica fume (alluded to previously in the silica discussion). Shotcrete plasticizers can include ethylenediamine as an active ingredient. This organic chemical is a well-recognized sensitizer associated with asthma and dermatitis (White, 1978; Ng et al., 1991). Shotcrete accelerators can include diethanolamine [2,2’-iminodiethanol], also a sensitizing agent (Piipari et al., 1998; Lessmann et al., 2009).

**Isocyanates (in Polyurethanes), Epoxies, and Related Reactive Polymer Chemicals**

These materials are widely used in modern mining and tunneling techniques associated with bolt placement and other ceiling- and wall-stabilizing applications (Ulvestad et al., 1999). Exposure to these sensitizing materials can lead to asthma and probably carry risk of dermatitis as well (Nemery and Lenaerts, 1993).

**Carbon Monoxide**

Whenever internal combustion engine-powered equipment is used in or near enclosed or semiclosed areas, or with heavy outdoor use, excess carbon monoxide inhalation may occur (NIOSH, 1972). Exposure sources can include fork-lifts, gas-powered generators or compressors, gas-powered equipment, and motor vehicles. Air intakes near carbon monoxide sources can entrain the gas, leading to overexposure remote from the source. Motor vehicles can cause elevated ambient exposures to carbon monoxide (as well as diesel vapor and particulates as discussed previously) beyond the worksite itself, especially near heavily trafficked roadways or as a result of idling vehicles. Carbon monoxide can also be present in postexplosive detonation atmospheres, together with oxides of nitrogen (as described above).

**Welding, Metalworking Fluids, and Other Maintenance-Related Exposures**

Mining and processing operations require extensive onsite maintenance operations that include welding, machining, and various other equipment and parts maintenance and repair work. Welding exposures are complex, and a detailed summary is beyond the scope of this review. Note, however, that stainless steel and titanium welding (the latter because caustic process solution handling can require titanium alloys in working parts) can carry particular exposure risks, for example, from chromium, nickel, and titanium metal fumes (Antonini et al., 2004). These welding techniques can be routine work practices in uranium processing plant maintenance. Metalworking coolant fluid exposures are also complex, with health effects associated in particular with microbial contamination (Mirer, 2010). Other potential maintenance-related exposures include solvents, lubricants (including under high pressure), paints, and sealants.
**Arsenic**

Arsenic can be a common contaminant in uranium, as with many other metal-bearing ores. Based on existing knowledge of the uranium ore-bearing characteristics in Virginia (see Chapter 3), however, this does not appear to be a relevant uranium processing exposure in handling locally mined ore. Were uranium processing to involve feedstock from other sites, the potential for arsenic contamination would require further assessment. In areas of the world where arsenic has been present as a uranium contaminant, exposure has been a major issue of occupational health risk among mining and process workers. Although arsenic is a potent toxin with a myriad of adverse effects, its carcinogenic potential has been particularly salient among uranium miners, in particular because of their concomitant exposure to radon (Taeger et al., 2008; Tomášek et al., 1994).

**Other Metals—Vanadium, Selenium, Iron**

Vanadium is commonly used as a catalyst in sulfuric acid manufacturing, which is often carried out on-site at uranium processing facilities. Exposure would be most likely to occur in the context of maintenance or catalyst replacement. The primary target organ for vanadium’s adverse health effects in humans appears to be the airway, manifested by a bronchitis syndrome. In addition, IARC classifies vanadium as possibly carcinogenic to humans. Selenium can be a natural contaminant of mined materials and thus be a constituent of waste tailings; in addition to natural sources, iron can enter the waste stream as an intentional process additive. For both selenium and iron, the occupational toxic exposure potential does not constitute a relevant health risk in this industry, although such metals do pose a potential environmental hazard as is noted later (see Chapter 6).

**Mold-Related Illness**

Work activities that disturb soil, anticipated in any large-scale construction operation, have been associated with outbreaks of mold-related illness due to histoplasmosis or blastomycosis in areas where these environmental fungi are endemic. This could include parts of Virginia. Outbreaks occur among those directly involved in construction activities, but also among bystanders. In histoplasmosis exposures, bystanders have generally been adjacent (e.g., students attending a university with campus construction); however, at least one recent community-wide blastomycosis outbreak was linked to area-level roadway construction (Schlech et al., 1983; Carlos et al., 2010).

**Sulfuric Acid and Sulfur Dioxide**

Uranium processing can use either acid or sodium carbonate to dissolve (leach) uranium into an aqueous solution, as noted in the technical discussion of uranium extraction in Chapter 4. Acid extraction generally requires sulfuric acid in large enough quantities to require either onsite production or the transport of substantial quantities of the bulk product to the processing site. Sulfuric acid can also be used later in the processing sequence to “strip” uranium from its solvent carriers (a mix of tertiary amines, decanol, and kerosene; see below), and in the treatment of process wastes and effluents (“effluent polishing”). Sulfuric acid production requires a source of sulfur that is handled through either a contact process or a wet sulfuric acid process. Both are associated with potential exposures, including sulfur dioxide, vanadium catalyst (as noted above), and sulfuric acid itself. Sulfuric acid skin contact, as might occur in a chemical spill, would be likely to lead to a chemical burn. Sulfur dioxide and sulfuric acid aerosols are both potent respiratory tract and mucous membrane irritants. Heavy acute exposure (e.g., through a leak or other large industrial release—events that can occur either as a result of on-site manufacturing or during transport from off-site) can cause severe lung injury; moderate acute exposure can lead to irritant-induced asthma (Blanc, 2010). Lower-level acute sulfur dioxide exposure—including area-level ambient air pollution, as might occur through inadequately controlled plant emissions—could be anticipated to cause asthma exacerbation, based on the known capacity of sulfur dioxide to induce increased airway resistance among persons with
preexisting airway hyper-responsiveness, the basis for the health effects endpoint in U.S. National Ambient Air Quality Standards for this pollutant (Johns and Linn, 2011). Epidemiologically, sulfuric acid aerosol exposure is a known cause of chronic dental erosion. Epidemiological studies of sulfuric acid manufacturing worker cohorts have been limited to production processes in which the source of sulfur is sulfur contained in mineral ore.

**Acrylamide and Related Polymeric Flocculants**

These materials are used in uranium refining, together with mechanical separation techniques (e.g., countercurrent decantation and further clarification steps), to precipitate nonmetallic particulates from the process stream. Human-exposure-related adverse effects from polymeric flocculants, as relatively high-molecular-weight polymers, would not be anticipated among secondary occupational users (e.g., people involved in uranium processing) in contrast to the potential exposure risks among primary polymer manufacturers.

**Tertiary Amines**

Tertiary amines are used, with alcohols and kerosene, to chemically extract uranium from the aqueous solution that remains following the flocculation/decantation process. In this processing step, the uranium partitions into an organic solvent phase, while other metals remain predominantly in the aqueous solution (referred to as raffinate; see Chapter 4). The tertiary amines commonly used are either trioctylamine (which is widely known by the trade name Alamine 336, but also has other synonyms) or tridecylamine (Mackenzie, 1997). Both of these tertiary amines have similar chemical structures, with nitrogen linked to three identical aliphatic side chains of either 8 (octyl) or 10 (decyl) carbon atoms. Toxicity data specific to these tertiary amine moieties are extremely limited. The Toxnet National Library of Medicine Toxicology Data Network lists only one human exposure study for trioctylamine and none for triodecylamine. For the triodecylamine, a Russian study did not observe acute irritation to humans exposed by inhalation, even though mouse toxicity was observed not only when test animals were exposed by inhalation, but also by skin contact (Loyt and Filov, 1964).

As opposed to early steps in the uranium processing sequence, which can include open tanks with varying amounts of shielding, depending on the uranium concentration in the ore, solvent extraction typically takes place within a closed-circuit system. When used in such an enclosed system, occupational exposures are likely to be minimal under normal operating conditions, but excess exposure could occur in maintenance or quality control activities or through loss of integrity for an otherwise closed system (e.g., through a leak or other rupture). As solvents, these materials should be presumed to be readily absorbable through the skin, in addition to inhalation of vapor or through droplets suspended in the air. As a chemical group, aliphatic amines have been associated with causation of occupational asthma, indicating a structure–function relationship (Jarvis et al., 2005; Seed and Agius, 2010). Other tertiary amines have been shown to produce adverse ocular effects in exposed humans; the assessment of such endpoints, however, has not been reported for the specific octyl- and decyl-tertiary amines (Page et al., 2003).

**Decanol**

Decanol, a 10-carbon aliphatic alcohol, is used with the tertiary amines in the uranium solvent extraction process. Human health data specific to decanol are limited. It does penetrate intact skin and has been studied as a potential absorption enhancer in models of transdermal delivery for pharmaceuticals (Williams and Barry, 2004), even though in another study, it was found to be a human skin irritant (Robinson, 2002). In a rodent study, inhalation of decanol up to vapor saturation levels did not demonstrate sensory irritation (Stadler and Kennedy, 1996). In addition to being a synthetic organic chemical, decanol also falls within the category of microbial volatile organic compounds (MVOCs), produced as metabolites of fungi and detectable environmentally in sites of mold contamination—when 12 such MVOCs were tested in a lung cell-line model of toxicity, decanol proved to be the most toxic by a factor of 5 to 10 (Keja and Seidel, 2002). Decanol, along with other shorter chain aliphatic alcohols, was shown in a rat model to potentiate
the liver toxicity of chloroform, even though decanol was not toxic on its own (Ray and Mehendale, 1990). Although questions of potential human toxicity are raised by these studies, the same imitated exposure scenarios in an enclosed system, as noted for the tertiary amines, are also relevant to decanol’s application in uranium processing.

**Kerosene**

Kerosene is a hydrocarbon distillate of mixed hydrocarbon composition that is employed in uranium purification at the same process stage as tertiary amines and decanol (see Chapter 4). As noted previously, overexposure would only be likely to occur through perturbations in otherwise enclosed processes. Generically, adverse health effects of kerosene vapor inhalation or skin absorption are associated with higher level exposures, in particular through dermal contact leading to substantial systemic absorption (Bebarta and DeWitt, 2004). In addition, aspiration of petroleum distillates, as well as inhalation of their combustion products, is linked to acute lung injury (Blanc, 2010). These latter exposure scenarios, however, are not anticipated from the routine use of kerosene in uranium processing, although the latter is possible if there were to be a fire. Onsite storage of inflammable materials can be associated with risk of conflagration, and leaks of material at any stage of use (including stored material prior to use or in recycling systems or waste handling) can lead to groundwater contamination.

**Sodium Hydroxide, Hydrogen Peroxide, and Ammonia**

Sodium hydroxide (caustic soda) can be used in an alkaline process for the initial precipitant step after uranium is dissolved into solution, or it can be used to raise the pH of an acid solution in another processing stage (see Chapter 4). Industrial process solutions of sodium hydroxide are caustic and corrosive, requiring adequate skin and eye protection when handled and other safeguards against splashes, sprays, or aerosolization of concentrated solutions to prevent caustic eye, skin, or inhalation injury. Similar safety steps are relevant for high pH alkaline solutions (sodium carbonate/bicarbonate) if used in the initial process step of dissolving uranium.

Hydrogen peroxide can be used in both early and later uranium processing steps. In the initial leaching step, it facilitates solubilizing uranium by acting as an oxidizing agent (sodium chlorate and ferrous sulfate also can be employed as oxidants; adverse health effects would be limited to unlikely ingestion scenarios). Hydrogen peroxide can also be used as a reagent (along with magnesia) in the precipitation of aqueous uranium in its final purification as an alternative to sodium hydroxide or ammonia. Hydrogen peroxide at industrial concentrations (e.g., 50 percent or higher) is a powerful oxidant and highly irritating by inhalation, eye, or skin contact.

Ammonia can be used in uranium processing to neutralize acidified aqueous solutions containing uranium and precipitate the uranium. Concentrated (e.g., anhydrous) ammonia is typically handled in pressurized containers. Ammonia is an acute respiratory tract mucous membrane irritant that in high-level exposures can cause severe lung injury. Because of its high solubility, injury to the upper airways, including the nasal tract, is particularly associated with ammonia inhalation episodes (Blanc, 2010).

For three of the agents discussed above (sodium hydroxide solutions, hydrogen peroxide, and ammonia), overexposure can occur through transportation mishaps if manufactured elsewhere and delivered for use, through storage containment failure, or through unintended release associated with valve or piping failure. Because pressurized ammonia is released as a gas (whereas the others are liquids), of the three, ammonia has the highest potential for inhalation injury in an acute system failure. In addition, unintended contact mixing of these materials, in particular hydrogen peroxide, with certain other reagents on-site can lead to potentially hazardous interactions. Adherence to internationally accepted best practices (see Chapter 8) should seek to minimize the likelihood of adverse events such as transportation mishaps or equipment failure that might lead to unintended releases of irritant or toxic chemicals.

**FINDINGS AND KEY CONCEPTS**

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The committee’s analysis of potential human health impacts that might apply if uranium mining and processing were to take place in Virginia has produced the following findings:

- **Uranium mining and processing are associated with a wide range of potential adverse human health risks. Some of these risks arise out of aspects of uranium mining and processing specific to that enterprise, whereas other risks apply to the mining sector generally, and still others are linked more broadly to large-scale industrial or construction activities.** These health risks typically are most relevant to individuals occupationally exposed in this industry, but certain exposures and their associated risks can extend via environmental pathways to the general population.

- **Protracted exposure to radon decay products generally represents the greatest radiation-related health risk from uranium-related mining and processing operations. Radon's alpha-emitting radioactive decay products are strongly and causally linked to lung cancer in humans.** Indeed, the populations in which this has been most clearly established are uranium miners that were occupationally exposed to radon. The epidemiological data from studies of radon-exposed miners clearly demonstrate that protracted radon decay product exposure causes lung cancer in a dose-dependent manner, and that it can act independently of other known carcinogenic exposures as well as having a greater than additive effect (i.e., synergistic effect) with co-exposures to other lung carcinogens (e.g., cigarette smoking). As protracted radon decay product exposure increases, so do the rates of lung cancer (i.e., a linear dose-response relationship). The existing scientific evidence indicates that even very low exposure to radon decay products carries some risk, so there are incremental excess risks down to the lowest rates of environmental radon decay product exposure.

- **In 1987, the National Institute for Occupational Safety and Health (NIOSH) in the Centers for Disease Control and Prevention recognized that current occupational standards for radon exposure in the United States do not provide adequate protection for workers at risk of lung cancer from protracted radon decay exposure, recommending that the occupational exposure limit for radon decay products should be reduced substantially. To date, this recommendation by NIOSH has not been incorporated into an enforceable standard by the U.S. Department of Labor’s Mine Safety and Health Administration or Occupational Safety and Health Administration.**

- Radon and its alpha-emitting radioactive decay products are generally the most important, but are not the only radionuclides of health concern associated with uranium mining and processing. Workers are also at risk from exposure to other radionuclides, including uranium itself, which undergo radioactive decay by alpha, beta, or gamma emission. In particular, radium-226 and its decay products (e.g., bismuth-214 and lead-214) present alpha and gamma radiation hazards to uranium miners and processors.

- **Radiation exposures to the general population resulting from off-site releases of radionuclides (e.g., airborne radon decay products, airborne thorium-230 or radium-226 particles, $^{226}$Ra in water supplies) present some risk. The potential for adverse health effects increases if there are uncontrolled releases as a result of extreme events (e.g., floods, fire, earthquakes) or human error.** The potential for adverse health effects related to releases of radionuclides is directly related to the population density near the mine or processing facility.

- **Internal exposure to radioactive materials during uranium mining and processing can take place through inhalation, ingestion, or through a cut in the skin. External radiation exposure (e.g., exposure to beta, gamma, and to a lesser extent, alpha radiation) can also present a health risk.**

- Because $^{230}$Th and $^{226}$Ra are present in mine tailings, these radionuclides and their decay products can—if not controlled adequately—contaminate the local environment under certain conditions, in particular by seeping into water sources and thereby
increasing radionuclide concentrations. This, in turn, can lead to a risk of cancer from drinking water (e.g., cancer of the bone) that is higher than the risk of cancer that would have existed had there been no radionuclide release from tailings.

- A large proportion of the epidemiological studies performed in the United States, exploring adverse health effects from potential off-site radionuclide releases from uranium mining and processing facilities, have lacked the ability to evaluate causal relationships (e.g., to test study hypotheses) because of their ecological study design.

- The decay products of uranium (e.g., $^{220}$Th, $^{226}$Ra) provide a constant source of radiation in uranium tailings for thousands of years, substantially outlasting the current U.S. regulations for oversight of processing facility tailings.

- Radionuclides are not the only uranium mining- and processing-associated occupational exposures with potential adverse human health effects; two other notable inhalation risks are posed by silica dust and diesel exhaust. Neither of these is specific to uranium mining, but both have been prevalent historically in the uranium mining and processing industry. Of particular importance is the body of evidence from occupational studies showing that both silica and diesel exhaust exposure increase the risk of lung cancer, the main risk also associated with radon decay product exposure. Thus, workers in the uranium mining and processing industry can be co-exposed to several separate lung carcinogens, including radon decay products, silica, and diesel. To the extent that cigarette smoking poses further risk in absolute terms, there is potential for increased disease, including combined effects that are more than just additive. Moreover, because manual workers and lower socioeconomic status (SES) groups in the United States generally have higher rates of smoking, work-related lung cancer in uranium miners and processors may be related to socioeconomic status such that those with lower SES could comprise a particularly vulnerable subset of the population.

- Although uranium mining-specific injury data for the United States were not available for review, work-related physical trauma risk (including electrical injury) is particularly high in the mining sector overall and this could be anticipated to also apply to uranium mining. In addition, hearing loss has been a major problem in the mining sector generally, and based on limited data from overseas studies, may also be a problem for uranium mining.

- A number of other exposures associated with uranium mining or processing, including waste management, also could carry the potential for adverse human health effects, although in many cases the detailed studies that might better elucidate such risks are not available. For example, some of the materials used in this industry may be potential sensitizers that could cause asthma. Many of these exposures have not have been adequately evaluated in animal or human studies.

- Assessing the potential risks of multiple combined exposures from uranium mining and processing activities is not possible in practical terms, even though the example of multiple potential lung carcinogen exposures in uranium mining and processing underscores that this is more than a theoretical concern.

**Footnotes**

1 Radon decay product concentrations are expressed in working levels (WL). A WL is equal to the total alpha energy released from the short-lived radon decay products in equilibrium with 100 pCi of radon gas per liter of air. Thus, if a worker is exposed to 0.166 WL for 1 month (170 hours), that worker’s cumulative exposure for that month would be 0.166 working level months (WLM). Exposure at the end of 12 months at a monthly exposure of 0.166WLM would yield a cumulative exposure of 2 WLMs.

2 See 30 CFR §§ 57.5047, 57.5038.

3 An SMR value above unity indicates a risk estimate greater that the comparison population—a probability of...
less than 5 in 100 (p < 0.05) that the observed deviation from unity would be observed by chance alone is
generally taken to indicate a statistically significance elevated SMR; this can also be presented as a 95% confidence interval [CI], indicating where the observed SMR falls statistically. Note that the unity value for an SMR can either be presented as a value of 100 or a 1, with an SMR of 150 equating to 1.5, if the 100 × convention is not used. The values that are presented here have not been multiplied by 100.


5 See ; accessed September 2011.


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