CHAPTER 9

VOLCANIC EMISSIONS AND HEALTH

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I. AN INTRODUCTION TO VOLCANIC TOXICOLOGY

Volcanic vents and fissures provide a conduit by which magma—the molten rock, gases, and water within the earth—may interact with human biological systems (Figure 1). Because of the range of materials that are ejected during eruptions, the consequent effects on human health are diverse. Contact may occur dramatically and immediately for people living close to the vent, such as during pyroclastic flows or the emission of large tephra projectiles. Alternatively, effects on health may occur slowly or at great distances from the eruptive site as a result of dispersal of volcanic material such as ash, dust, and aerosols.

The vast majority of volcanogenic fatalities in the past few centuries have resulted from "proximal" events—such as pyroclastic flows, lahars, and suffocation or building collapse from ash or debris—and "distal" events, such as tsunamis, which may spread for hundreds of miles from the active site, and indirect consequences of eruptions, such as famine or infectious disease outbreaks. Simkin et al. (2001) placed the death toll over the last 500 years at above 250,000, with seven eruptions dominating the historical record (including Tambora in 1815, Krakatau in 1883, Pelee in 1902, and Ruiz in 1985). Apart from the obvious thermal and physical injuries resulting from an eruption, ejecta may also contain toxic elements and compounds that disrupt biological systems. These compounds may be released in the form of volcanic gases or carried with volcanic matter falling from eruptive columns or plumes. Some of the material ejected may induce disease by undergoing radioactive decay: among the best documented of such products is radon. Although these toxins are often not the major causes of mortality in volcanic eruptions, they may persist and have the potential to cause long-term morbidity.

Considering the pathological consequences of exposure to volcanic toxic compounds is a complex task. There is no simple, predictable path between the "emergence" of a toxin from the magma to the eventual health consequences it imparts to a particular individual. This chapter reviews the varied mechanisms by

FIGURE 1 Vent of an active volcano: White Island, New Zealand. (Photo: Michael Durand).

FIGURE 2 Krafla fissure eruption, Iceland. (Photo: Olle Selinus).

which the emissions associated with volcanism may compromise health and generate disease. In general, these mechanisms may be influenced by one or more of the following factors:

1. Eruptive variables
2. Toxin-specific properties
3. Patterns of toxin dispersal and persistence
4. Biological variables

Eruptive variables—The nature of the eruption (or other volcanic event) influences the duration of emissions, the chemical composition of the toxic compounds expelled, and the range of dispersal. For example, eruptions may be broadly grouped as *explosive* (releasing large quantities of gas, hot ash, and dust, as with Mount St. Helens), *effusive* (associated with large lava flows but less dramatic outpourings of gas and dust, as with the basaltic volcanoes of Hawaii), or *mixed* (a combination of the two patterns). Activity may be measured using the Volcano Explosivity Index, which incorporates many variables including the volume of tephra, the eruption type, and duration (Newhall & Self, 1982).

Toxin-specific properties—These primarily pertain to the chemical and physical properties of toxic compounds. Volcanic products vary in terms of particle size, concentration, pH, and water solubility. All of these factors can influence the bioavailability of toxins, and thus their patho-physiological effects.

Patterns of toxin dispersal and persistence—In terms of evaluating possible effects on human populations, physical proximity to the vent or eruptive site is an important component of risk assessment. Populations who fall within the "near-vent" range may be exposed to the full array of ejected materials, often at high concentrations (Figure 2). By contrast, areas that are distal from the volcanic site (or are less vulnerable to volcanic products for some other reason, such as the presence of a natural barrier) tend to be exposed to a smaller range of toxic compounds, and at concentrations less likely to result in injury. There are exceptions, however, with toxins such as fluoride adhering to fine, wind-dispersed ash particles and thereby occurring in highest concentrations some distance from the vent. In this discussion, near-vent is used loosely to refer to an area extending tens of kilometers from the eruptive site.

The mode of toxin dispersal should also be considered. Eruptive products may travel along many routes, and in a variety of chemical forms, before finally appearing in human biological systems. Carriage in the atmosphere and hydrosphere are the most common

modes of dispersal, but poisoning may also occur as a result of volcanic products entering the soil and food chain.

The duration of exposure plays one of the most crucial roles in determining health outcomes. For example, some insults may be short-lived and reversible, as with conjunctival irritation from ash particles, or may be chronic, as with inhalation of silica particles resulting in the life-long respiratory problems of silicosis. Some toxic compounds, such as radon, may persist in volcanic products (and continue to cause injury) long after the eruptive event ceases. These patterns of injury, and the body systems that are predominantly affected, are discussed in detail in Sections III, IV, and V.

The properties of the environment also affect the pattern of dispersal and settlement of volcanic toxins, including features of the physical environment (geography, air pressure, and climate) or man-made surroundings (for example, the opportunities for asphyxiant gases to enter low, enclosed spaces such as cellars).

Biological variables—The mechanism of damage must be considered at a histopathological level. Volcanic products may produce injury in human tissues and cells, either individually or in combination, in the following ways: (1) direct physical interaction (e.g., skin contact with acidic gases); (2) initiation of a chronic process of damage and repair (e.g., fibrosis from the deposition of respirable silica particles in lung tissue); (3) metabolic disruption (e.g., with carbon monoxide toxicity); or (4) genotoxicity and genetic alteration (e.g., from exposure to carcinogenic agents, such as radon).

Finally, the characteristics of the affected individuals play an important role in determining health outcomes. Important parameters include age, the presence of coexisiting cardiac or respiratory diseases, and access to appropriate diagnostic services. There is also an inherent response spectrum, both phenotypic and genotypic, in any human population and some individuals are more susceptible than others. Such variation in susceptibility is discussed in Section III.C.

Figure 3 illustrates the major determinants of health outcomes in a simplified schema that shows: (1) the spatial range of health effects (in relation to proximity to the vent), (2) the primary mode of human exposure, and (3) potential duration of health effects following contact with the toxic compound. A comparison is provided for three common volcanic products: free silica, a respirable mineral; sulfur dioxide, a noxious gas (and its associated sulfuric acid aerosol); and fluoride compounds (including the highly acidic gas, hydrogen fluoride, HF).

II. The Health Effects of Tephra Dispersal

A. Atmospheric Dispersal of Toxic Compounds

Tephra dispersal is a major cause of morbidity following eruptions. Tephra thrown into the atmosphere may cause disease through the fallout of particles from eruption columns or plumes on human populations, or through the movement of individuals into eruptive clouds (such as aircraft passengers and crew). The emission of large fragments of rock, such as "blocks" and "bombs," may cause severe physical injury including lacerations and fractures. Heavy fallouts (especially of pumice) can lead to burial and asphyxiation, either directly or through a roof collapse. Smaller particles of pumice, scoria, and ash may be distributed over a wide area around the eruptive site, and in some cases plumes may affect settlements situated hundreds of kilometers away. This chapter considers the health effects of tephra by primarily examining the effects of ash and dust (the constituents of tephra less than 2 mm in diameter). The pathophysiological effects of the compounds discussed are summarized in Table I.

The eyes are particularly vulnerable to the emission of fine tephra particles. Common ocular injuries include abrasions of the cornea and conjunctivitis from accumulation of ash in the conjunctival sac (Blong, 1984). Ocular irritation has been reported in people using contact lenses, because of the interposition of matter between the contact lens and the eye. Swelling of the eyelids and other facial tissues around the eyes have been reported less frequently. Symptomatically, ash produces higher reported rates of ocular redness, itchiness, throbbing pain, and discharge. Superficial tissues such as the skin, lips, mouth, and other mucous membranes may also be exposed. Nasal and throat irritation occur more frequently, and higher rates of nasopharyngeal irritation and nasal stuffiness were observed in a group of loggers following the Mount St. Helens eruption (Baxter et al., 1986). A less well-documented effect of tephra is skin irritation, including in the axillary area, following deposition of volcanic ash particles on the skin—the so-called "ash-rash." Irritation of superficial mucous membranes, eyes, and exposed skin commonly resolves shortly after the exposure to the ash ceases, and longer term toxic injury to such body structures following ash-fall is unusual.

The lungs may be exposed to any particulate matter able to penetrate into the respiratory passages. This

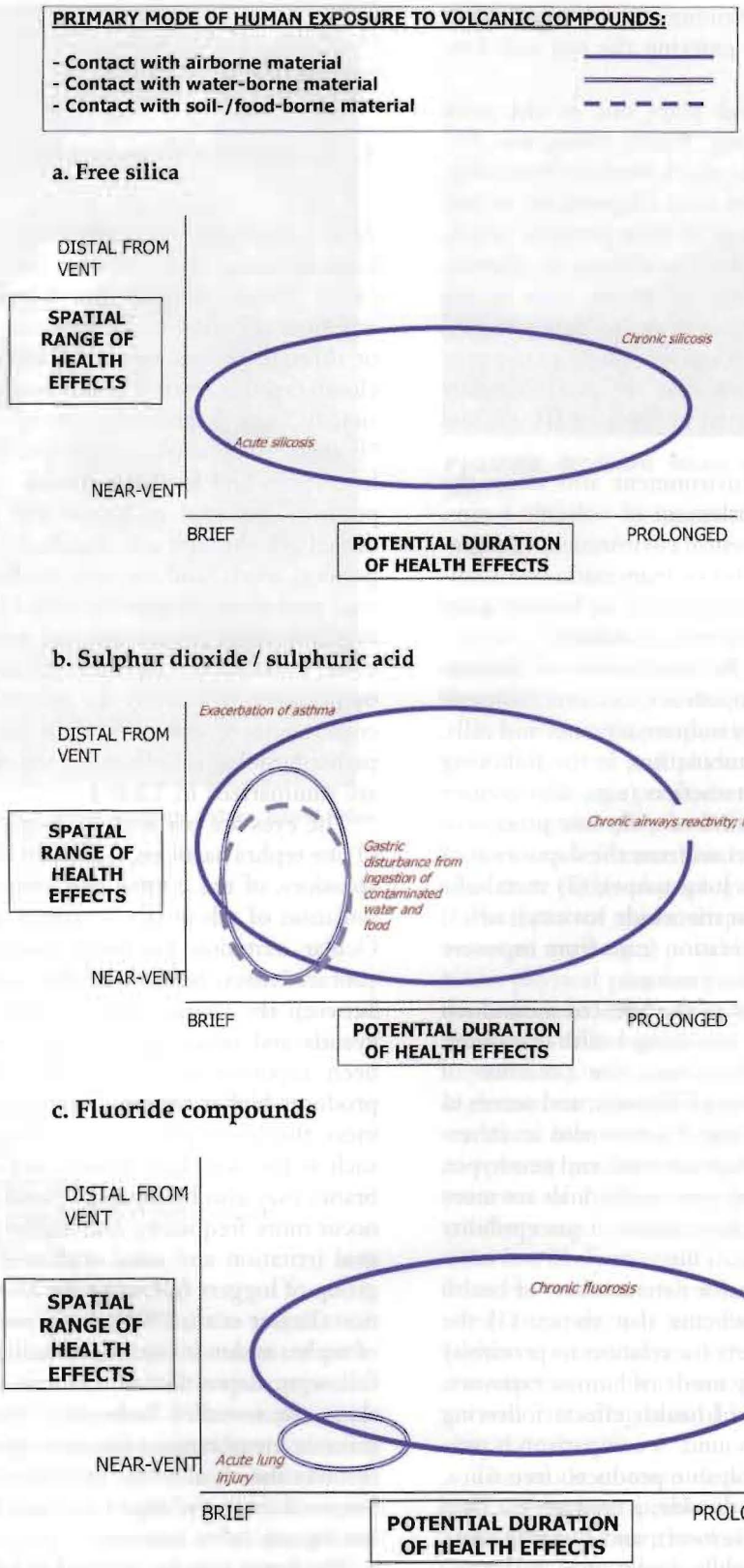

FIGURE 3 Major determinants of health outcomes for silica, sulfur dioxide, and fluoride.

TABLE 1. A Review of Major Toxic Compounds of Volcanic Origin and Their Potential Pathophysiological Effects

Toxin (active form)	*Mode of dispersal to human populations*	*Mechanism of injury*	*Acute systemic effects*	*Chronic or recurrent systemic effects*
Sulfur compounds				
Sulfur dioxide, SO_2; sulfur trioxide, SO_3; sulfuric acid, H_5SO_4	Gas emissions during eruptions, lava flows, degassing episodes	Acidic irritant	**RESP**: upper airway irritation, pneumonitis, pulmonary edema, acute ARDS → ≤ **HEENT**: nose and throat irritation, conjunctivitis **DERM**: skin irritation	**RESP**: recurrent or prolonged exacerbation of respiratory disease, bronchiolitis obliterans → ≤
Hydrogen sulfide, H_2S	Gas emissions during eruptions	Irritant, asphyxiant, inhibition of metabolic enzymes	**GENERAL**: headache nausea, vomiting, confusion, collapse, paralysis of respiratory centers → ≤ **GIT**: diarrhea **GU/REPRO**: pain on urination **RESP**: cough, shortness of breath, pulmonary edema **HEENT**: eye and throat irritation	
Fluoride compounds				
Fluoride compounds (including related acidic gases, aerosols and liquids)	Gas emissions during eruptions, ash leaching	Acidic irritant on inhalation or contact with skin, conjunctiva, or mucous membranes	**GENERAL**: hypocalcemia (low serum calcium), low serum magnesium, collapse and shock → ≤ **RESP**: (e.g., HF inhalation) coughing, laryngeal spasm, bronchitis, pneumonitis, pulmonary edema, acute ARDS → ≤ **GIT**: nausea, vomiting, gastrointestinal hemorrhage → ≤ **GU/REPRO:** nephritis **HEENT**: eye and throat irritation **DERM**: severe, slow healing burns; may be absorbed through skin causing internal effects	**RESP**: permanent lung injury from toxic inhalation **HEENT**: mottling and/or pitting of teeth **MSS**: osteoporosis, osteosclerosis, calcification of ligaments and tendons, kyphosis of spine with bony exostoses

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Chlorine compounds				
Hydrochloric acid, HCl	Gas emissions during eruptions, lava (e.g., with seawater contact)	Acidic irritant	**GENERAL**: collapse → ≤ **RESP**: coughing, laryngeal spasm, pneumonitis, pulmonary edema, acute ARDS → ≤ **HEENT**: eye and throat irritation	**RESP**: permanent lung injury from toxic inhalation
Carbon compounds				
CO	Gas emissions during eruptions	Noxious asphyxiant; binds to hemoglobin	**GENERAL**: collapse, coma, "cherry red" skin → ≤ **NEURO**: headache, impaired dexterity	**NEURO**: permanent neurological impairment from brain injury
CO_2	Gas emissions during eruptions	Inert asphyxiant	**GENERAL**: asphyxia, collapse → ≤	
Free silica and silicates				
Free silica	Ash plumes	Mineral dusts initiate inflammatory response and fibrosis (scarring)	**RESP**: acute exacerbation of respiratory disease (e.g., asthma)	**RESP**: chronic silicosis
Metals				
Mercury vapor, Hg	Gas emissions during eruptions	Oxidant irritant	**RESP**: bronchitis, pneumonitis, pulmonary edema **NEURO**: neurotoxicity, (may lead to acute or chronic mercury intoxication → ≤)	**NEURO**: neurotoxicity

Key to major body systems:
CVS: Cardiovascular system
DERM: Dermatological system (skin and adnexa)
GENERAL: Includes multiorgan, metabolic, and endocrine effects
GIT: Gastrointestinal system
GU/REPRO: Genitourinary and reproductive systems
HEENT: "Head, eyes, ears, nose and throat," including the scalp, face, eyes, and adnexa, ears, nasal cavities, sinuses, pharynx, oral cavity, or dentition
MSS: Musculoskeletal system
NEURO: Neurological system
RESP: Respiratory system
→ ≤ = Potential cause of mortality

respirable portion of tephra refers to particles less than 10 μm in diameter, and those under 2.5 μm may penetrate further into the lungs into the terminal bronchi and alveoli. The proportion of respirable ash varies greatly across eruptions. Higher levels of total suspended particles (TSP) caused by ash-fall may escacerbate some pre-existing respiratory complaints including asthma and bronchitis (Baxter et al., 1981; Baxter, 1983; Yamo et al., 1990). An increase in the frequency of bronchitis was also detected after the 1996 Mount

Ruapehu eruption in New Zealand, despite the small amount of ash and the relatively low respirable portion (Hickling et al., 1999). The probable mechanism by which ash produces such respiratory symptoms is by provoking hypersecretion of mucus and bronchoconstriction (narrowing of the air passages): both are reversible, however, and diminish once exposure ceases (Buist, 1988).

Dispersal of tephra may also produce health effects over a longer duration, and respiratory diseases are among the most common subacute, recurring, and chronic problems in this context. Baxter et al. (1983) reported higher rates of asthma and bronchitis exacerbations for over 3 months after the 1981 Mount St. Helens eruption. There appeared to be a clear correlation with respirable ash, which persisted in the ambient air of surrounding settlements long after the eruption. This effect was less prominent following the Mount Pinatubo eruption, despite the high volume of tephra. This lower degree of respiratory morbidity may have occurred because subsequent rainfall acted to settle volcanic dust and limit opportunities for respiration of particles. Alternatively, the chemical nature of the respirable portion of ash may have differed significantly between the two eruptions: such differences are difficult to predict and quantify, but may be important with respect to health protection (see Section VII, this volume).

In terms of chronic lung pathogenesis, one of the most troublesome compounds produced by volcanic activity is silica. Certain forms of silica, such as cristobalite and tridymite, occur in lava and may be formed when amorphous silica or quartz is heated to high temperatures. Inhalation of fine particles of crystalline silica, including quartz, is a well-established cause of both acute and chronic inflammatory reactions in lung tissue (see also Chapter 18, this volume). If large quantities of silica are inhaled over a short period, exposure may result in the appearance of inflammatory cells in the interstitium and filling of alveoli with proteinaceous material. If exposure to silica is prolonged, the particles may evoke a chronic inflammatory reaction which recurs until the lung tissue displays extensive signs of fibrosis (tissue repair with collagen fibers). In the chronic phase of silicosis, the classical pathological features of the disease are lung nodules, which are masses of concentric collagen fibers with a rim of silica dust and macrophages (white cells) containing trapped silica particles. These whorled masses slowly enlarge and may impinge on airways and the vasculature of the lung.

The mechanisms by which silica evokes this reaction are probably related to the chemical reactivity of the particle surface. SiOH groups form bonds with, and ultimately damage, proteins and phospholipids embedded in cellular membranes. Cell death may result. Furthermore, silica interacts with macrophages, causing the activation of these cells and the release of inflammatory mediators, such as interleukin-1. This process triggers the migration of other inflammatory cells to the alveoli, which amplifies the body's response and encourages intense deposition of collagen fibers.

During the Mount St. Helens eruption, the free silica content was 3–7% of the "sub-10 μm" (that is, potentially respirable) fraction of ash. Due to the relatively low free silica content, it was estimated that the chances of silicosis developing even in high-exposure groups (such as cleanup crews or loggers) was negligible (Baxter et al., 1986). The risk of silicosis is thought to be much greater for the residents remaining on the island of Montserrat, which has experienced many years of eruptions starting from 1995. At this location, the free silica content has been much higher (10–24% by weight of the sub-10 μm fraction), and the exposure is much more prolonged (Baxter et al., 1999; Baxter, 2001). Populations living in close proximity to Mount Pinatubo are also thought to be vulnerable following exposure to high levels of respirable silica.

Other particles of volcanic origin, including silicates such as talc, may also cause prolonged fibrotic changes, and these are included in a broader classification of lung diseases known as pneumoconioses. (This term encompasses inflammatory lung reactions that occur from contact with respirable mineral dusts, but that are distinct from asthma or bronchitis that may follow mineral dust exposure.) Following the Mount St. Helens eruption, workers exposed to tephra over a long duration, such as loggers and road gangs around the eruptive site, were examined for the advent of pneumoconiosis. One deceased victim appeared to have been suffering from distinctive granulomatous lesions of the lungs, a chronic inflammatory reaction similar to that observed in rats exposed to respirable tephra (Green et al., 1982). However, a longitudinal study of survivors found that respiratory symptoms largely disappeared three years after exposure, and no permanent changes in chest x-rays were observed relating to ash exposure (Buist et al., 1986). Animal studies indicated that volcanic ash from various eruption sites (including Mount St. Helens ash) displayed less cytotoxicity than free silica, and was more readily phagocytosed by macrophages in the alveoli (Vallyathan et al., 1984).

It is has also been suggested that respirable dusts from volcanic eruptions may have carcinogenic effects. Apart from the example of radon (described below), ash may

contain fibrous zeolites (including erionite). These asbestiform minerals have been linked to endemic pleural disease and a high rate of mesotheliomas (a highly lethal malignancy which typically infiltrates the pleura or peritoneum) (Rohl et al., 1982).

B. Hydrospheric Dispersal of Toxic Compounds

During and following volcanic activity, tephra particles may affect water supplies in two principal ways. First, tephra particles (such as ash or dust) may be deposited onto bodies of water, which include irrigation or filtration plants, thus rendering the water highly turbid and unusable. Secondly, tephra may carry a variety of adsorbed chemicals. Plumes of ash and dust, which may cover hundreds of kilometers, may effectively disperse such "stowaway" toxic agents. After the ash settles to the ground, these toxins may be dissolved—often by rain water—and thus leach into the environment. The dominant chemicals which may adsorb to tephra, and thus act as leachates, include chlorine (Cl), sulfur (S) compounds, sodium (Na), calcium (Ca), potassium (K), magnesium (Mg), and fluorine (F). Other elements that are present in smaller quantities are manganese (Mn), zinc (Zn), copper (Cu), barium (Ba), selenium (Se), bromine (Br), boron (B), aluminum (Al), silicon (Si), and iron (Fe). Furthermore, even light tephra-falls may have significant effects on water pH. During the 1953 eruption of Mount Spurr (Alaska), a 3–6 mm tephra fall on Anchorage caused the pH of the town water supply to fall to 4.5, returning to 7.9 after a few hours (Blong, 1984). Such excursions in pH and turbidity can alter the chlorine demand significantly at treatment plants, adding a significant, albeit indirect, microbial water hazard.

Some of these elements and compounds have safety levels established in drinking water and could potentially cause harm if ingested in quantities exceeding these concentrations. There is a paucity of conclusive studies relating to the health effects of ash leachates, although some effects may be inferred from accidental or occupational exposures. An example is provided by fluoride, a relatively common volcanic product, for which numerous cases of toxicity have been reported in various settings.

Fluoride is rapidly and effectively absorbed following ingestion (up to 90% absorption from the gastrointestinal tract), and most of the ionic fluoride remaining in the body has a high affinity for calcified tissues, particularly bones and developing teeth. Despite its usefulness in reducing dental caries, it may produce toxicity if ingested in high concentrations. Acute fluoride poisoning can result in death, such as following the accidental contamination of public water systems (Gessner, 1994). Acute symptoms of excessive fluoride ingestion include nausea, vomiting, excessive sweating, and abdominal discomfort (probably from the formation of the corrosive hydrofluoric acid in the stomach) (Grandjean, 1982). Clinical and laboratory evidence indicates that kidney damage (nephritis) may also result, thus disturbing water and sodium balances in affected patients. Higher concentrations of fluoride may produce cardiovascular convulsions, collapse, and shock partly as a result of fluid loss from vomiting and gastrointestinal bleeding. Fluoride may also lower levels of calcium in the blood, possibly by a process of precipitation in which insoluble CaF_2 is formed. Painful and involuntary muscle contractions may result from the depletion of available calcium. Although not recorded in humans following volcanic eruptions, these signs and symptoms are well-established in grazing livestock in Iceland (see below) (see also Chapter 12, this volume).

Chronic ingestion of fluoride may cause a number of low-grade symptoms, including loss of appetite, headache, vertigo, and joint pain. The most characteristic effects, however, involve the dentition and skeletal system. Although not directly a result of recent ash leaching, elevated fluoride levels in potable water adjacent to the Furnas volcano in the Azores have resulted in dental fluorosis, a condition characterized by mottling of the teeth (Baxter & Coutinho, 1999b). Mottling—the appearance of chalky white patches distributed irregularly over the teeth—develops if chronic fluorosis occurs during the development phase of permanent dentition. The enamel is structurally weak, and may eventually become pitted, in which case the affected teeth acquire a corroded appearance. Such dental changes are often the only signs of chronic fluorosis.

In areas where waters are highly fluoridated, however, more serious problems may emerge. For example, teeth and gums may degenerate. Skeletal symptoms start to appear, which include back pain and limitation of spinal movement. Studies of fluoride exposure in various contexts have indicated that, as a result of the fixation of bone calcium by fluorine, bones undergo a number of changes: increased hardening and rigidity (sclerosis) of bones; the development of bony "projections" (osteophytes and exostoses) on the ribs, pelvis, and vertebrae; increased thickness of long bones; and calcification of the ligaments (Grandjean, 1982). The results of long-term fluoride exposure may thus be crippling.

Other evidence relating to emission of ash rich in soluble fluorine have been obtained from studies of effects on other mammals. It is known that the ingestion of high concentrations of fluoride is highly toxic to livestock (Baxter, 1983). Following the 1947 eruption of the Icelandic volcano Hekla, a mere 1-mm deposition of fluoride-rich ash was sufficient to kill thousands of sheep. Icelanders in the eighteenth century also suffered from the long-term consequences of fluorosis following the eruption of Laki (1783). During this eruption, in which over 140 cones formed along a 27-km fissure, it is estimated that eight megatons of fluoride compounds were discharged. (Fluoride may also be expelled from volcanic vents as unadsorbed gas, including fluorine, F_2, and HF; volcanic gases are discussed subsequently.) The deposition of high concentrations of fluorine on pastures and waterways proved fatal for numerous animals. Death occurred from at least two causes. First, the volcanic discharge resulted in extensive crop damage, and thus stock starved from loss of feed. In addition, ingestion of fluoride from pastures and waterways caused direct toxicity (see also Chapter 20, this volume).

Records of the period indicate that fluoride may also have directly poisoned the human population. A contemporary account describes a curious disorder affecting farmers living around the eruption site. These symptoms included severe swelling in many joints that was associated with painful cramps. The most striking effects, however, appeared to affect the oral cavity: sores appeared on the palates and tongues of the victims, and eventually teeth and blackened portions of gum fell from their mouths. Although it is difficult to exclude the effects of scurvy, Blong (1984) suggested that the illness may be the result of toxic fluorosis. Recent studies of meltwater around ash particles from Mount Hekla, Iceland, show fluoride levels up to 2000 ppm. Although rapidly diluted to about 200 ppm in standing water and 20 ppm in running surface water, these fluoride concentrations could readily produce symptomatic fluorosis if ingested by humans (N. Oskarsson, personal communication).

Determining areas at risk from fluoride leachates remains difficult, and the relationship between concentrations and distance from the volcanic vents is highly variable. For example, during the Mount Ruapehu eruption in New Zealand in 1995, it was concluded that the levels of leachable fluoride in adjacent water supplies did not pose a threat to human health (Weinstein & Patel, 1997). Following the Popocatepetl eruption on May 12, 1997, however, levels of fluoride were maximal at a distance of 13.4 km from the eruptive site. It was predicted that had such ash entered the water supplies, fluoride levels would have exceeded the national standard of 1.5 mg/L (Armienta et al., 2001). Such results suggest that the possibility of significant water contamination by fluoride should be considered during and following the eruptive phase.

C. Toxic Compounds in the Soil and Food Chain

Volcanic material may also enter the human body by direct ingestion of contaminated foods or soils. As with the hydrospheric effects, toxic chemicals may be carried into the soil or food supplies by adsorption to tephra particles and subsequent leaching. (This is discussed in Section III. Emissions of volcanic gases also contribute to chemical deposition.) Usually ingestion of these compounds is unintentional, but humans may consume volcanic products such as mineral-rich muds for therapeutic or nutritional reasons.

Selenium is an example of a volcanic compound that may potentially produce health effects through foodborne exposure. This element may be emitted in substantial concentrations (e.g., 630 kg of aerosolized selenium per day during a 1976 eruption of Mount Etna [Faivre-Pierret & Le Guern, 1983]), thus providing unusually large Se loads for soils and edible plants. Chronic toxicity from the consumption of foods with high levels of selenium have been reported which include cases with probable associated fluorosis in some districts in China (Yang et al., 1988). Signs of chronic exposure include loss of hair and nails, skin lesions, and abnormalities of the nervous system. Elevated selenium levels may increase the risk of dental caries. The exact mechanism of selenium toxicity remains uncertain, although it has been suggested that the element may cause disruption of sulfur metabolism, inhibition of protein synthesis, or oxidation of sulfhydryl groups. The role of selenium and cancer is debated. Some studies indicate that there is a protective effect against cancer, and others have argued that selenium displays pro-oxidant—and thus possibly carcinogenic—effects (Spallholz et al., 1994) (see also Chapter 15, this volume).

The effects of tephra on the food chain and the hydrosphere intersect through the damage caused to aquatic life. During the 1979 eruption of Karkar, Papua New Guinea, heavy tephra-falls (and possibly small lahars) tainted the headwaters of surrounding rivers, which resulted in the death or contamination of aquatic life. However, edible fish, eels, and prawns continued to

be consumed by local farmers, despite complaints of the sulfurous taste (Blong, 1984). The health effects of this form of food contamination are unknown.

III. THE HEALTH EFFECTS OF VOLCANIC GAS EMISSIONS

A. Description of Volcanic Gases

The gaseous substances produced by volcanic activity are varied, and may be classified as follows:

1. Gases and vapors: The gaseous state of an element which normally exists in a liquid or solid form and can be readily reverted to this form by decreases in temperature or increases in pressure (such as mercury)
2. Aerosols: Droplets or particles suspended in a gaseous medium
3. Fumes: Aerosols of solid particles, usually less than 0.1 μm in size, usually formed by escape of volatiles from molten materials
4. Smoke: Volatile gases and particles, usually less than 0.5 μm in size, produced by combustion (Kizer, 1984).

The pathophysiological effects of the compounds discussed are summarized in Table I.

Steam, from both magmatic and superficial sources (such as overlying lakes or groundwater), is the most common volcanic gas. Other, often very toxic, gases are also emitted during eruptive events and may impact on human health (Figure 4). There are numerous accounts of volcanic gases causing death. In Japan, for example, 49 people have been killed over the last 50 years by becoming overwhelmed by volcanic gas emissions (Hirabayashi, 1999).

FIGURE 4 Volcanic gas emissions. (Photo: Michael Durand).

Among the most notorious gases are those which are heavier than air, such as CO_2 and H_2S. These may pool at ground level and result in asphyxia. HF, hydrochloric acid (HCl), hydrogen (H), helium (He), carbon monoxide (CO), and radon may also be produced in considerable quantities. Vaporized metals, such as mercury (Hg), are also found in plumes. Gases may be released even in the absence of obvious volcanic activity (degassing).

In assessing the impact of volcanic gases, it is not always possible to separate the toxicological effects of each gas released during a particular eruption. Numerous gases may be emitted simultaneously, or the gaseous components or concentrations may vary over time as the eruptive process evolves. For example, the sequence of activity around Rabaul, Papua New Guinea, over the last seventy years included: relatively quiet solfataric emissions of CO_2 and H_2S (prior to 1937); a violent steam explosion resulting in over 500 fatalities (between May and June 2, 1937); the appearance of vigorous vents producing large clouds of sulfur dioxide, with periodic emissions of hydrogen chloride, gradually increasing until another major eruption commenced (1937–1941); a release of CO_2 in a pit crater at the Tavurvur site, causing the death of six persons who had been collecting eggs (1990); the eruption of the two major vents, producing large quantities of ejecta, widespread building collapse, the evacuation of over 50,000 residents from Rabaul, and an accompanying SO_2 plume with an estimated size of 45,000 km^2, with a maximum SO_2 mass of 80 kilotons (1994); and the emission of a large bubble of flammable gas, presumed to be methane, which ignited and initiated bush fires (1997).

Although not as dramatic as full-scale eruptions, other types of geothermal activity (including hot springs and fumaroles) also generate a variety of toxic gases (Sparks et al., 1997). Because such activity may often continue unabated over prolonged periods, and because the benefits of hot springs may encourage the presence of humans, there is a risk of toxic exposure. For example, fumaroles are very numerous in some areas (such as Yellowstone Park) and may emit high levels of numerous gases including CO_2, SO_2, HCl, and H_2S. A full discussion of geothermal toxins is beyond the scope of this chapter.

In terms of adverse impacts on human health, volcanic gases may be classified into the following groups: gases with *irritant effects* on the respiratory system; those

which act as *inert asphyxiants*; and those which combine both properties and act as *noxious asphyxiants*.

B. Inert Asphyxiants

Carbon dioxide, CO_2, illustrates the effect of an inert asphyxiant gas: it replaces oxygen, but does not have a directly toxic effect on biological tissue. Concentrations of CO_2 are particularly high near emission vents (Faivre-Pierret & Le Guern, 1983). The degassing of volcanic soil may result in the collection of carbon dioxide in cellars, huts, and in low-lying areas (Baubron & Toutain, 1990; Baxter & Coutinho, 1999). Low concentrations (e.g., under 5%) produce accelerated breathing, and often feelings of discomfort, by direct activation of the respiratory centers in the brain. Headache and vertigo are early symptoms. If sufficient concentrations are reached (for example, concentrations of 7–10% for a few minutes), fainting occurs. Elevated levels of CO_2 in the bloodstream (hypercapnia) eventually result in circulatory failure and death from acidosis.

In Java's Dieng Volcanic Complex, it is believed that emissions of CO_2 from a fissure resulted in the deaths of 149 people (Le Guern & Faivre-Pierret, 1982). The dramatic effect of CO_2 emissions is also illustrated by two disasters in the Cameroon at Lake Monoun in 1984 and at Lake Nyos in 1986. It is thought that large volumes of CO_2 emerged from these crater lakes and, carried downward by gravity, engulfed whole villages in their path. As a result, 37 people were asphyxiated near Lake Monoun, and the death toll reached 1700 at Lake Nyos. The survivors from the Lake Nyos disaster reported falling in a deep state of unconsciousness for up to 36 hours. It appears that no long-term respiratory effects occurred in the survivors, although some sustained burns by falling into cooking fires during a period of CO_2-induced coma (Wagner et al., 1988; Afane et al., 1996). In areas with the highest CO_2 concentrations, a variety of animal life ranging from insects to livestock also perished. Although the exact mechanism of release is unclear, it is probable that the gas release was not in fact secondary to underlying volcanic activity. A widely accepted hypothesis suggests that soda springs deep in these crater lakes create accumulated pockets of CO_2, which may be abruptly emitted at periodic intervals.

C. Irritant Gases

Volcanic gases which have primarily *irritative* (directly injurious) *effects* include the hydrogen halides, HF and HCl. At low doses, eye and throat irritation may occur. At higher levels, both may cause ulceration of the respiratory tract upon inhalation and corrosive burns upon contact with the skin or mucous membranes. Cutaneous burns from contact with HF are particularly severe and notoriously slow to heal. Fatalities may occur from pulmonary edema and laryngeal spasm (discussed below).

Although there are few clinical accounts of direct toxicity from these gases in a volcanic context, an indication of likely health effects is provided by fumes from Hawaii's fumaroles and basaltic lava, which contain high concentrations of both HCl, and, to a lesser degree, HF (Murata, 1966; Kullmann et al., 1994). As discussed in Section V, HCl and HF concentrations are often highest in dense plumes arising close to the ocean, from which sea winds carry mist clouds to adjacent villages of Kalapana and the Hawaii Volcanoes National Park. The HCl-/HF-acidic aerosol may exacerbate pre-existing lung disease (Ostro et al., 1991), even at great distances from volcanic vents (Mannino et al., 1996). On the island of Ambrym, Vanuatu, cutaneous burns resulted from acid rain following a 1979 emission of eruptive gases high in HCl and sulfur compounds. Gastric upsets were also reported from the ingestion of acidic cistern water (Scientific Event Alert Network, 1989).

At a pathophysiological level, irritant gases affect the respiratory tract in a number of ways. The relatively more soluble gases, which include HF and HCl, tend to be removed by mucus linings before they reach the alveoli. Therefore, such gases predominantly cause inflammation in the upper airways, which results in symptoms such as cough and reactive bronchoconstriction. These effects, although often relatively short-lived, may be severe, particularly in people with hyperreactive airways (such as asthma sufferers). Less soluble volcanic gases, including hydrogen sulfide and mercury vapor (both discussed below), are less likely to be "cleared" by the mucus secretions of the upper airways. Thus, there is greater risk of penetration to the level of the smaller airways and alveoli.

As the concentration of respired irritant gases rises, there is a greater chance that alveolar damage will result. The most vulnerable targets are the epithelial cells lining the airways, the mucosal tissues underlying these cells, and endothelial cells lining the pulmonary blood vessels (including the capillaries of the alveoli). Damage to these cells results in an increase in vessel permeability. Ultimately, the alveoli fill with protein-rich fluid from plasma, which interrupts the process of effective gas exchange. In addition, there may be denudation and sloughing of alveolar epithelium and the mucous membranes of bronchiolar walls.

During the acute phase, the spectrum of disease caused by irritant gases is highly variable and includes pneumonitis, which results in a dry cough, shortness of breath, and evidence of patches of inflammation on chest radiography. The condition may often resolve without long-term sequelae such as pulmonary edema (the accumulation of unwanted fluid in the respiratory organs, for which there are numerous other etiologies). In other cases, pulmonary edema emerges within a day of exposure, and may be associated with the formation of mucus plugs and collapse of areas of the lung. Because of compromised oxygen intake, patients may experience breathlessness and acute (or adult) respiratory distress syndrome (ARDS). This syndrome is associated with severe alveolar injury, edema, and hemorrhage. It has a mortality rate of around 50%, and represents the end point for a number of disease processes apart from toxic inhalation.

If the victim survives the acute stage of gas exposure, a number of further clinical outcomes may occur. Often the symptoms may rapidly reverse once the gas is removed and full recovery occurs. If the gas has produced severe epithelial damage, however, cellular regeneration may take weeks. A secondary effect of this alveolar disruption is that the lung tissues become more vulnerable to bacterial invasion, and thus chest infections may intervene in the post-exposure period. Occasionally, toxic gas exposure results in permanent fibrosis (scarring) of lung tissue. Another potential outcome from toxic inhalation (described in greater detail below) is prolonged airway hyperreactivity, although the specific cause of this disorder remains uncertain.

Sulfur dioxide, SO_2, is well-established as a cause of acute and chronic disease. Both the gas, and the sulfuric acid aerosols into which it forms, are highly irritant, particularly to the eyes, nasal passages, throat, and respiratory tract. High SO_2 concentrations may also act to cause asphyxia, although in volcanic contexts the effects appear primarily irritative.

Sulfur dioxide exposure may provoke exacerbations of asthma, even at low concentrations. Increased airway resistance has been noted in asthmatics exposed to SO_2 at concentrations of 0.5 ppm when exercising, and at 1 ppm during rest. In non-asthmatic controls, an increase in airway resistance has been provoked at 5 ppm (Bethel et al., 1983). Cough and eye irritation occurs at concentrations of 20 ppm or above. At higher doses, pneumonitis, pulmonary edema, ARDS, and ultimately death may result.

In the context of volcanically active sites, SO_2 concentrations of 1 ppm have been recorded far downwind (30 km) from the Masaya Volcano in Nicaragua (Baxter et al., 1982a). The effects of long-term emissions have been examined at Kilauea, Hawaii, which has been erupting for 15 years and (in 2001) continues to eject 1500 tons of sulfur dioxide per day. Episodes of increased SO_2 in the ambient air have exceeded health standards 80 times in the last 15 years and appear correlated to ongoing eye irritations, throat pain, and respiratory problems, including asthma exacerbations (Elias & Sutton, 2001).

At the histopathological level, sulfur dioxide has a diverse range of respiratory effects. Although larger aerosols are filtered out by the nose and nasopharynx and are rapidly removed by mucociliary clearance, smaller SO_2 particles (particularly those less than 10 μm in diameter) may be deposited in deeper airways. Sulfur dioxide also acts to increase levels of mucus secretion and viscosity. Animal studies also indicate that the gas impairs the ability of macrophages to destroy bacteria in the alveoli, thus increasing the risk of respiratory infection.

Apart from affecting mucus production, SO_2 may produce airway narrowing through other mechanisms. As mentioned, people with high levels of airway hyperresponsivness, such as those with asthma and/or atopy (tendencies to allergic responses), are particularly vulnerable. Sulfur dioxide may provoke the recruitment of inflammatory cells, including histamine-secreting mast cells and eosinophils, which persist long after exposure ceases. Such cells contribute to the risk of recurrent airway constriction, particularly if provocation with gases (or other agents and allergens) recurs. This bronchoconstrictive response may enter a chronic phase (sometimes after only one exposure of a particular irritant gas). Other irritant gases and aerosols, such as nitrogen dioxide (discussed below), may precipitate a similar response. If such sequelae occur without a previous history of asthma or allergy, some authorities apply the term reactive airways dysfunction syndrome (RADS). Other changes from SO_2 exposure include chronic neutrophil infiltration and edema (fluid accumulation) in the mucosa, which is more distinctive of chronic bronchitis.

Two related, but less common, syndromes resulting from exposure to SO_2 (and other volcanic products, such as mercury vapor) are bronchiolitis obliterans and bronchiolitis obliterans-organizing pneumonia (BOOP). Both conditions are related to a delayed inflammatory and reparative process occurring some time after the initial injury. Bronchiolitis obliterans is associated with the appearance of plugs of granulation tissue (capillary-rich tissue associated with healing) in small airways, together with the presence of fibrous

scarring. A similar process occurs in BOOP, except that the alveoli and their adjacent tissues are also involved. The different patterns may be associated with variation in host responses to the toxic insult (Epler et al., 1985).

In addition to acting directly, irritant gases may create health problems by their interaction with other atmospheric processes. For example, droplets of rainfall or mud may pass through the irritant gases present in toxic clouds or ash plumes, thus creating acid rain. The health effects of this process were experienced by people on Kodiak Island, who suffered from falls of both ash and sulfuric acid rain following the Katmai eruption on the Alaskan mainland in 1912. A number of Kodiak islanders, 160 km downwind from the active vent, suffered from "stinging burns" when this rain contacted their lips or skin (Blong, 1984). Acid rains in other sites have also resulted in eye irritation (e.g., with Masaya in Nicaragua [Baxter, 1982a]) and apparently hair loss (during the 1917 eruption of Boqueron). A secondary health risk is posed by the interaction between acid rains and heavy metals: acid rain has reacted with zinc in galvanized roofs and tainted water supplies as a result (Baxter et al., 1982b).

As with tephra fallout, chemicals dispersed with volcanic gas emissions may affect water and food supplies. During the 1783 eruption of Laki in Iceland, apart from the high levels of expelled fluorine, an estimated 150 megatons of sulfur dioxide was discharged. This vast quantity of gas, and the aerosolized sulfuric acid it formed, had destructive consequences for vast tracts of surrounding pasture lands. The event tipped the balance in the already marginal farming environment of Iceland. The ensuing period of crop damage resulted in massive livestock losses: half of all the island's cattle and horses and four-fifths of the sheep perished. One-fifth of Iceland's population, around 10,000 people, died as a consequence of famine (called the haze famine from the persistent presence of sulfur compounds in the atmosphere) with toxic fluorosis probably also contributing to some fatalities.

D. Noxious Asphyxiants

The pungent gas hydrogen sulfide, H_2S, is a noxious asphyxiant: that is, it acts as both an asphyxiant and a powerful irritant. Its metabolic effect is to inhibit cytochrome oxidase, one of the enzymatic drivers of cellular metabolism (Jappinen et al., 1990).

At low concentrations, H_2S may cause irritation of the conjunctivae and mucous membranes. Short exposures at concentrations of 2 ppm have not shown any statistically significant effects on respiratory function (Jappinen et al., 1990), and other effects of chronic exposure at low levels form the basis for ongoing studies (P. Shoemack, personal communication). However, once the H_2S concentration in ambient air increases, the effects become more definitive, and exposure even for a few seconds may be fatal. For example, at the Kusatsu-Shirane crater in Japan, a high school teacher and two students were overcome and killed by H_2S emitted from nearby fumaroles that had concentrated in a bowl-shaped depression (Scientific Event Alert Network, 1989). Early signs of poisoning include headaches, ocular and respiratory irritation, and loss of smell (anosmia). Apart from these effects, inhalation of the gas also directly damages the respiratory tract and precipitates pulmonary edema in the lungs. At 1000 ppm, fainting occurs. Ultimately, H_2S causes cessation of breathing by direct action on the respiratory centers of the brain. Those who recover from poisoning may suffer from long-term neuropsychological effects. These sequelae are probably related to acute hypoxic effects on the brain either during poisoning or subsequently from pulmonary damage.

E. Health Risks from Other Volcanic Gases

Information relating to the effects of exposures to other volcanic gases is limited. After symptomatic (or fatal) episodes of inhalation, it may be difficult to determine the nature of the gas or combinations of gases that were present, and whether the concentrations were high enough to cause illness. Other gases produced during eruptive or degassing events that may *potentially* reach concentrations sufficient to produce symptoms in humans include: carbon monoxide, CO; nitrogen dioxide, NO_2; carbon disulfide, CS_2; methane, CH_4; and ammonia, NH_4.

Carbon monoxide—Like hydrogen sulfide, CO is an example of a noxious asphyxiant. Although concentrations in plumes are usually low, the gas may reach high concentrations in certain eruptions and near fumaroles (Tazieff & Sabroux, 1983; Williams & Moore, 1983). CO is toxic to humans in small doses. Once inhaled and absorbed, it rapidly permeates across the membrane of red blood cells and binds to the iron component of hemoglobin. Its affinity for hemoglobin

is profound, over 200 times greater than oxygen, and it rapidly reduces the oxygen-carrying capacity of blood. Symptomatically, low levels of blood saturation with CO produce headache, nausea, and impaired judgment and dexterity. As the percentage saturation exceeds 30–40%, severe nausea, vomiting, confusion, hyperventilation, and collapse ensue. Coma and death occur if treatment is not provided rapidly. The characteristic "cherry red" skin associated with CO poisoning occurs because of the persistent saturation of both arterial and venous blood with this "hemoglobin-loving" gas (which forms carboxyhemoglobin). Survivors of CO poisoning may suffer from permanent neurological and behavioral symptoms, which include disorientation, mood changes, and movement disorders.

Nitrogen dioxide—Significant concentrations of NO_2 have been detected in plumes from the Mount St. Helens eruption (Olsen & Fruchter, 1986). If high concentrations are inhaled, hydrolysis of NO_2 results in the production of nitric acid, particularly in the lower respiratory tract (including the alveoli). Nitric acid in turn produces nitrates and nitrites, both of which are capable of producing cytotoxicity and disruption of cellular membranes. The clinical outcomes of NO_2 inhalation are in some respects comparable to those of contact with SO_2. Acutely, victims may suffer from chemical pneumonitis, pulmonary edema, ARDS, or fatal lung injury. Susceptible individuals may suffer from exacerbations of asthma, increased need for bronchodilators, and decreased pulmonary function (Moseholm et al., 1993). As with SO_2, emergency room visits and hospital admissions for asthma are also increased by exposure to NO_2. Delayed or chronic outcomes of toxic inhalation, which may appear even after apparent "recovery," include chronic airway obstruction and bronchiolitis obliterans.

Carbon disulfide—CS_2 has been detected in volcanic plumes, although concentrations are usually low. This gas acts as a powerful toxin and produces headaches, muscular weakness, and delirium.

Methane—Methane, together with CO and CO_2, was among the gases which hampered rescue efforts during the 1973 eruption on the island of Heimaey, Iceland (Williams & Moore, 1983). It may also be concentrated around fumaroles. Primarily, it is an inert asphyxiant and acts by replacing oxygen in breathed air.

Ammonia—Exposure to this highly water-soluble gas results in severe irritation to the eyes, skin, and upper airways. Injuries are produced by thermal and chemical (alkali) burns as ammonia reacts to form hydroxyl ions on exposed mucosal surfaces.

IV. VOLCANIC DISPERSAL OF METALS AND TRACE ELEMENTS

The dispersal of toxins such as fluoride and selenium have been discussed above. In addition, there may be widespread dispersal of other elements and metals with identified risks for humans. For example, using a plume dispersal model for a 1976 eruption of Mount Etna, a variety of toxic aerosols was estimated to have exceeded permissible air concentrations at a distance of 10 km from the vent. These included lead (Pb), mercury (Hg), copper (Cu), zinc (Zn), selenium (Se), and cadmium (Cd). Arsenic (As) exceeded recommended concentrations at a distance of 5 km (Faivre-Pierret & Le Guern, 1983). Daily aerosol outputs were also estimated, which included 360 kg of lead, 110 kg of arsenic, 75 kg of mercury, and 28 kg of cadmium per day. These last four elements, which have well-established toxic effects even in small doses, are discussed briefly below as well as in greater detail in other chapters in this book.

Metal vapors, such as those produced by mercury, may act directly as an irritant gas. It has been suggested that mercury is sometimes present in concentrations sufficient to be hazardous if emissions were inhaled over a prolonged period of time (Baxter, 1983). Mercury vapors act to cause pulmonary edema and bronchitis and are neurotoxic once absorbed.

Arsenic can be deposited in soil or water (including seepage into subterranean wells) following volcanic or low-level geothermal activity (Welch et al., 1988). Chronic arsenic poisoning may affect many organ systems. For example, in affected populations in Taiwan and India, ingestion of drinking water high in arsenic has been associated with a variety of skin lesions, which include increased or decreased pigmentation and keratosis. Gangrene has also been reported (blackfoot disease). The carcinogenic role of arsenic has been suggested in a range of studies: exposure increases the risk of cancer of the skin, lung, liver, bladder, and kidneys (see also Chapter 11, this volume).

Lead produced by volcanic processes may potentially be inhaled or ingested in contaminated water and foodstuffs. The health effects, particularly on the neurological development of children, have been well-documented.

Like lead, cadmium enters the human body through inhalation or ingestion. Cadmium deposited in the soil crosses readily into plants and thus the food chain. Having entered the body, the toxin tends to be retained

in the liver and kidneys and is excreted extremely slowly. The effective half-life of cadmium in humans may exceed 30 years. Cadmium thus has the potential to result in prolonged illness, and toxic effects may occur in virtually any organ. The more common manifestations of acute ingestion or inhalation include vomiting, cramps, respiratory difficulties, and ultimately loss of consciousness. Chronic effects include anemia and renal disorders. More controversial is the connection between elevated cadmium and hypertension.

It is important to note that the valency state (species) of these metals and metalloids can be a key determinant of their potential biological activity. Although we have no further details about the role of valency in relation to volcanic emissions, the issue is discussed in more detail for some elements in other chapters.

FIGURE 5 Lava from the Krafla fissure, Iceland. (Photo: Olle Selinus).

V. VOLCANISM AND RADIATION HAZARDS

Radioactive decay of volcanic material may also have consequences for human health. Ash may have high uranium content and carry adherent particles of radon, an alpha-radioactive gas that has been linked to the development of lung cancer (Baubron & Toutain, 1990). Exposure may also occur from use of volcanic material, such as for building. In the Azores, radon has been found in high concentrations in dwellings within the Furnas caldera. Radon is discussed in detail in Radon in Air and Water, this volume.

VI. TOXIC EXPOSURE WITH OTHER ERUPTIVE EVENTS

A. Lava Flows

One of the more visually dramatic outcomes of volcanism is the ejection of fluid or semi-fluid material, such as basaltic lava (Figure 5). In some locations (for example, Hawaii), eruptions may be associated with fountaining of molten material, in which globules of plastic lava are sprayed over a kilometer high. These may feed into lava lakes and lava flows that course away from the volcano. The direct threats to health posed by lava flows are primarily thermal injuries. Often fatalities occur because of unexpectedly rapid flows because escape routes have been cut off, or from steam explosions created when the lava strikes a water source.

Lava flows may result in illness less directly by exposing humans to toxic chemicals. For example, the basaltic lava flows in Hawaii are often associated with the release of sulfur dioxide and aerosolized droplets of sulfuric acid. As discussed, HCl and (a lesser degree) HF may also be formed, particularly when molten lava strikes the ocean, thus creating falls of acid rain from the steam plume (Mannino et al., 1996). Lava may also act to taint subterranean wells by the process of leaching. The toxicity and health effects of compounds released from lava flows do not differ from those already discussed, although the exposure may at times be intense, such as in the area around an erupting crater. Emergency crews often work in close proximity to lava flows. On Heimaey in 1973, for example, crews spent many days on or near lava flows, applying cooling waters in a successful attempt to solidify and direct flows away from the main town.

B. Pyroclastic Flows

Pyroclastic flows are intensely hot ionized gas flows that contain dispersed fragments of debris, which may travel at speeds up to 200 km/h (Houghton et al., 1999). The exact composition and temperature varies greatly, but usually some of the fragmented rock within such flows is within the respirable range. The gas content will usually include H_2O (which may be superheated), CO_2, SO_2, and H_2S. Such flows present an immediate risk to humans close to the vent. With their considerable

kinetic energy, these deadly "volcanic hurricanes" simultaneously sear and blast objects in their path. The fatality rate of those caught in such flows is usually extremely high, and the common causes of death include asphyxiation (often from burial), trauma, and severe burns (especially of the respiratory system). For example, during the 1902 eruption of Mount Pelée on the island of Martinique, such a superheated gas cloud rapidly enveloped the city of St. Pierre, resulting in over 30,000 fatalities.

Pyroclastic flows result in varying degrees of thermal injury to the skin ranging from superficial erythema, to deep penetration into the subcutaneous tissues, to the extreme of complete incineration. Victims are commonly described as appearing dried and "mummified," rather than charred (the outcome usually observed with fire injuries). Respiratory effects appear to occur as a result of intense heat, oxygen deficiency, ash inhalation, and toxicity of the gas. Asphyxia from plugs of ash in the upper airways was described as the cause of death in those caught in the flow of the Mount St. Helens eruption. Survivors of the devastating flows from Lamington, Papua New Guinea, in 1953, have suffered from symptoms suggestive of pharyngeal burns, including throat pain, shortness of breath, and inability to swallow (Taylor, 1958). Health effects subsequent to the acute injury include pneumonia, tracheobronchitis, and ARDS, presumably from irritation and secondary infection of injured respiratory tissues (Eisele et al., 1981).

Flows, surges, and debris avalanches also impact human health because of their capacity to disperse toxic compounds. For example, during the Mount Pinatubo eruption in 1991, pyroclastic flows contributed to the volcanic material that covered a wide area and filled surrounding valleys. Subsequent erosion, often triggered from monsoon rains, then acted to mobilize the volcanic chemicals for many years subsequently.

C. Volcanic Activity and Aquatic Environments

Apart from the interaction between tephra and the hydrosphere described above, volcanic and aquatic processes may intersect in other ways (Figure 6). Crater lakes, for example, can act as a reservoir of toxic compounds which, in some circumstances, may affect human populations. Some lakes, such as the Poas volcano, Costa Rica, sit atop a degassing system. At this site, steady emissions of sulfur dioxide pass through a shallow lake, which is often hot and intensely acidic (pH <1). These emissions, together with particles of rock dissolved in the acid lake water, are periodically dispersed out of the crater and have been linked to respiratory problems in downwind communities (Baxter, 1997). At Poas and Kiwa Ijen in Java, crater lakes have contaminated water supplies with fluoride and other elements (Baxter, 2001).

FIGURE 6 Crater lake formation adjacent to a volcanic vent. (Photo: Michael Durand).

A fast-moving and potentially lethal consequence of volcanic eruptions is the lahar. These torrential flows of mud, water, and debris wash down the sides of the volcano, and may occur in association with crater lakes or the melting of snow and ice during or after eruptive events. For communities situated in the path of lahars, the opportunities for timely warnings may be limited—sometimes with lethal consequences. Lahars from some volcanic lakes, such as Kelut in Indonesia, may be hot and often acidic. Five thousand people died from a lahar generated from the 1919 eruption of Kalut. Those caught in the flow often suffer from drowning, suffocation while entrapped, or severe trauma from penetrating wounds and fractures (Baxter, 1990). Following the Nevado Del Ruiz lahar in Colombia, burns were also noted and may have been acidic in origin (Lowe, 1986).

For submarine eruptions, rafts of pumice and areas of discolored water on the surface may be the only evidence volcanic activity has occurred. At the other end of the hazard continuum, such events may produce tsunamis, which pose a major threat to coastal populations even at considerable distances from the vent. Submarine activity may manifest itself in other ways, as illustrated by the destruction of a Japanese research vessel *No. 5 Kaiyo-Maru* in 1952. The vessel had been traveling to monitor a submarine eruption 420 km south of Tokyo when radio contact with the vessel was lost.

Examination of the debris (which included boat fragments containing embedded rock particles) suggested that an explosion beneath the ship had caused the disaster, in which all 31 crew members were killed.

Apart from such infrequent events, little is known about the threat to health posed by submarine emissions of gases and particles. It is probable that most ejected material is thoroughly diluted in seawater and thus poses little risk for humans. However, in some regions, activity may occur near coastal populations. In the Antilles, for example, recently active submarine sites include Kick 'Em Jenny, situated 8km north of Grenada, and other vents near Martinique, where periodic episodes of "boiling water" have been reported (Roobol & Smith, 1989). Along the shallow Reyjkanes oceanic ridge near Iceland, bubbles rich in methane and CO_2 may rise to the sea surface (German et al., 1994). In Papua New Guinea, weak tremors and increased fumarole emissions of SO_2 and HCl around the Kadovar volcano were associated with reddish discoloration of the surrounding seawater, which was possibly associated with iron hydroxide. Coastal villagers reported that this discoloration persisted from June 1976 to 1978 (Mori et al., 1989). These eruptions and emissions, although under water, are in effect near vent phenomena. The potential for toxicity remains, albeit tempered by the aquatic environment.

VII. VOLCANIC MONITORING AND HEALTH PROTECTION

HEKLA, perpetuis damnata estib. er nivib. horrendo boatu lapides evomit (Hekla, cursed with eternal fires and snow, vomits rocks and a hideous sound)

—an early surveillance report of the volcano Hekla (from 1585 map of Iceland)

A. Introduction

From the perspective of health protection, the purposes of volcanic monitoring are threefold: (1) to provide an early warning system of potential health hazards, thus providing a preparation period for resident populations, health services, transport services, electricity utilities, etc., that are located in the hazard zone; (2) to minimize illness from contaminated water supplies, tainted or fouled foodstuffs, and air pollutants, particularly for susceptible groups; and (3) to provide accurate records of adverse health events for use in epidemiological and clinical research.

The phases of volcanic monitoring may be usefully classified as follows:

- Pre-eruptive phase: This period extends from the elevation of alert levels above the baseline state, hence indicating increased likelihood of volcanic activity, to the actual start of the eruption.
- Eruptive phase: The period surrounding the eruptive event (or degassing episode), including event imminence, the primary volcanic event, and periods of ongoing volcanic activity.
- Post-eruptive phase: The period over which volcanic activity wanes, allowing recovery and rehabilitation measures to take precedence.

The current discussion will be based on the assumption that these phases provide the loci of intervention designed to protect the public health.

B. The Pre-Eruptive Phase

This phase is primarily directed at preparing at-risk populations for an impending volcanic event. Early volcanic activity is usually monitored by geologists and involves assessments of seismic activity, ground deformation, gas emissions, geophysical variables, and hydrology. Using hazard maps, the primary aim during the pre-eruptive stage is to predict the temporal and spatial pattern of an eruption, together with the scale and path of any pyroclastic flows, lahars, or lava flows. Disaster scenarios will usually include the need for, and possible extent of, evacuation from the danger zone. Secure locations and safe travel routes for displaced populations should also be considered.

An important strategy during the pre-eruptive phase is the dissemination of information regarding potential health effects and strategies for minimizing exposure. Ideally, this process of training and educating should be continuous (even in periods of quiescence), and only escalated in the event of increased alert levels. (In Japan, for example, evacuation drills are conducted routinely in areas at risk.) Baseline tests of water and air quality should be conducted.

From an epidemiological perspective, this period may be an opportunity for selection of groups for prospective studies (for example, pre-eruptive baseline screening of people with asthma who will be followed through and subsequent to the eruption and ash-fall).

C. The Eruptive Phase

Early warning systems should be established for affected communities in order to ensure that the public, and organizations designed to preserve the societal infrastructure, are prepared for the arrival of the eruptive phase. For example, hospital staff and general practitioners should be informed of possible consequences of tephra fallout, including an increased frequency of motor vehicle accidents, ocular problems, and exacerbations of asthma.

Where risks are minimal (such as from light ash-fall), it may not always be necessary to recommend major changes in daily routines. Some groups of susceptible individuals, such as those with pre-existing respiratory disease, may benefit from consultation with health professionals. Instruction of disease management in the event of ash-fall or increased levels of gaseous emissions should be initiated if appropriate. For some patients, such as those with asthma who may react to raised levels of sulfuric acid aerosol, medical advice may be as simple as recommending that bronchodilator medications (such as inhalers) are always readily at hand. In those with chronic illness, strategies for ongoing medical management are particularly important should evacuation become necessary. More general advice may be required each day, including avoidance of areas of likely ash-fall. Ongoing status reports of hazards and strategies to avoid risk should be provided using newspapers, radio and television broadcasts, and public notices. In at-risk areas, it is imperative that such communication plans *pre-exist* the volcanic event to avoid the need to generate the information on an ad hoc basis.

Table II summarizes health impacts of eruptive events and relevant health preservation strategies. The hazards are separated according to proximity to the eruptive site using the general categories near vent and distal from vent.

Given the range of health consequences from exposure to volcanic ash and dust, monitoring tephra dispersal is an important component of eruption-phase management. The size and composition of tephra constituents vary between volcanoes and from eruption to eruption; this makes it difficult to predict the chemical composition of any given volcanic product. Although the health effects of tephra fallout are often not immediately life-threatening for populations distant from the eruption site, resulting illness may be both widespread and chronic in nature. As discussed, ash may cause ocular injury and exacerbations of respiratory disease. Prior warnings of ash-fall, together with regular announcements on air quality, will enable susceptible individuals to minimize their exposure or to seek medical advice where appropriate. Rapid analysis of particulate levels is also necessary to address or alleviate public concerns regarding other chronic outcomes such as silicosis. As with communication strategies, it is imperative that protocols for these analyses pre-exist the eruption.

As described above, tephra may also have disruptive effects on water supplies. Following some eruptions there is a direct risk of toxicity, which includes acute fluorosis. Water quality may also be rapidly degraded by increased turbidity and fluctuations in pH levels. Apart from direct testing and pH monitoring, it may often be possible to anticipate potential effects on water quality by estimating volumes of ash falling in water catchment areas and by using results from rapidly performed leachate studies. Such analyses are of importance not only to populations drawing water from large catchment areas, but also to those using non-public water supplies (such as for crop irrigation) that are not routinely monitored for quality.

Other health hazards of ash include the mechanical effects (particularly of wet ash), which cause roofs to collapse resulting in trauma. Heavy ash-falls may also result in traffic accidents, a product of both poor visibility and ash coating on roads (Dent et al., 1995). Cleanup crews are particularly at risk, and falls from roof cleaners often cause greater morbidity than any direct effect of the eruption.

Table III summarizes the strategies for tephra monitoring, which indicates how such information may assist in health protection for vulnerable populations.

As discussed in the previous section, volcanic gas emissions—and the aerosols that are derived from them—also pose a major threat to health. Therefore, as with tephra, gas production at the vent should be monitored in order to alert downwind communities of the potential hazards on a daily basis. Information of gas (and tephra) dispersal production will usually be combined with weather predictions. Combining "climatic" and "volcanic" forecasts—including predictions for acid rain or combined ash and fog contributing to poor visibility—will allow communities to implement appropriate precautions on a day-by-day (or hour-by-hour) basis.

The monitoring of gases and pH often requires specialized equipment that may not be available for permanent use in all settlements at risk of exposure (Environment m.f.t., 1994). A possible solution may be the provision of a mobile air quality team that responds to predictions based on geologic and climatic data.

TABLE II. Comparison of Proximal (Near Vent) and Distal Health Impacts and Corresponding Health Preservation Strategies

Eruptive event	*Consequence*	*Health impact*	*Health preservation strategies*
Near vent			
Explosion	Blast, rock fragments, shock waves	Trauma, skin burns, lacerations	Evacuation, movement to secure shelters
	Lightning	Electrocution	
	Forest and bush fires, combustion of buildings and vehicles	Burns, smoke inhalation	
Pyroclasts and other thermal emissions	Pyroclastic flows	Skin and lung burns	Evacuation
	Ash flows and falls	Asphyxiation	
Drainage of crater lakes, melting ice, snow, or rain accompanying eruption	Mudflows, floods	Engulfing, drowning	Evacuation, diversion barriers
Lava	Lava flow	Engulfing and burns (rare)	Evacuation, diversion barriers
	Forest/bush fires	Burns	Evacuation
Gas Emissions H_2O, SO_2, CO, CO_2, H_2S, HF	Pooling in low lying areas and inhalation	Asphyxiation	Evacuation, avoidance
		Airways constriction (exacerbation of asthma, COPD)	Respiratory protective equipment
Radon	Radiation exposure	Lung cancer	Evacuation
Distal from vent			
Ash-fall	Ash/dust less than 10 μm in diameter	Asthma, exacerbation of pre-existing lung disease	Wear high-efficiency masks, minimize exposure, protect homes and offices from ash infiltration
	Siliceous dust	Silicosis if free silica content high and exposure prolonged	Respiratory protective equipment
	Water contamination with fluoride, possibly also heavy metals (e.g., cobalt, arsenic)	Gastrointestinal upset and electrolyte disturbance (may be fatal)	Avoid water that has not yet been approved, avoid surface water, use water from wells
	Food contamination	As above	
		Foreign bodies in eyes	Goggles for when heavily exposed
		Conjunctivitis, corneal abrasions	
Gas Emissions	Acid rain	Eye and skin irritation; possible toxic contamination	Protection during rainfall, avoid collection of rain water for drinking, especially from metal roofs, etc.
	Dispersal of irritant and/or asphyxiant gases	Exacerbation of pre-existing lung disease (especially asthma) exacerbation from acidic aerosols)	Prophylactic use of medication
		Mucosal and conjunctival irritation; suffocation	Avoidance/evacuation

Adapted from Baxter et al., 1986.

TABLE III. Strategies for Monitoring Tephra Dispersal: A Health Protection Perspective

Monitored component of tephra fallout	*Location of monitoring*	*Optimal time for monitoring*	*Health-related objectives of monitoring*
Ash production and ash-fall predictions	At the vent	Immediately	a. To alert areas downwind of the probability and degree of ash-fall b. To anticipate and minimize the effects of traffic disruption and mechanical injury (e.g., roof collapse)
Mapping of ash-fall (isopach)	Throughout ash-fall area	As soon as possible after ash-fall in order to determine load of ash in water catchment areas, pastures, etc.	a. To maintain water quality, including monitoring of turbidity and pH b. To minimize the risk of ingesting toxic substances in drinking water (e.g., fluoride)
Proportion of particle size which is respirable (<10 μm)	Priority to plume-vulnerable areas with greatest population density	Immediately after an eruption	To quantify the level of respirable ash to assess the risk of acute and chronic illness (e.g., silicosis)
PM-10 (overall measure of particulate matter <10 μm, i.e., from volcanogenic and other sources, such as man-made pollutants)	a. Priority to plume-vulnerable areas with greatest population density	a. Continuously and preferably at locations where humans are likely to receive maximum exposure	a. To monitor daily levels and provide information to individuals with respiratory disease (e.g., to stay indoors on certain days); also for research purposes in linking the respirable portion of ash to health effects
	b. Personal monitors on individuals exposed to much ash	b. Whenever exposed to ash	b. To monitor total exposure and any associated health effects; this is especially important if the ash has a high free silica content
Free silica content of respirable portion	a. Priority to plume-vulnerable areas with greatest population density	a. Preliminary studies immediately after an eruption	a. To assess, and hopefully alleviate, public anxiety concerning silicosis
	b. Individuals that will be exposed to high levels (e.g., cleanup workers)	b. More detailed studies at later date	b. To determine the relative risk of silicosis for individuals exposed to varying concentrations and types of free silica
Particle morphology	At a range of distances from the vent	At a later date	To assess the possible requirements for long-term health monitoring and/or to initiate prospective studies (e.g., relationship between exposure to fibrous zeolites and pleural disease)
Leachate studies	a. In water catchment areas supplying public drinking water	a. Immediately after ash-fall	a. To assess risk to water quality in the catchment region, and to initiate clearance of toxins and particles; this is often required urgently because water demand is likely to be high during the process of cleanup
	b. In water catchment areas of private water supplies (e.g., irrigation, wells)	b. Ideally as soon as possible; however, the assessment based on ash depths and leaching studies may be all that is practicable	b. To assess potential effects on water quality

D. The Post-Eruptive Phase

As the post-eruptive phase commences, and recovery becomes possible, it is critical to continue monitoring for as long as toxic compounds remain present in the environment. The conclusion of the "disaster phase" does not indicate the cessation of monitoring requirements. Collection of geological and health-related data should be ongoing for epidemiological reviews in order to anticipate which measurements are important for any subsequent eruptions. It may be necessary to specifically review exposure patterns in high-risk groups (such as cleanup workers) and in areas where dust will be disturbed (e.g., roads, schoolyards, city centers). In areas with persistent or repeated ash-falls, advice and hazard communication similar to that provided during the eruptive phase is important.

E. Utilizing and Integrating Data from Eruptions

In a coordinated response to volcanic hazards, ideally a number of information sources should be utilized. Geological monitoring may often help predict when and where an eruption will take place, as well as the type and scale of activity expected (Figure 7). After appropriate assessment of hazards, the information should be disseminated to all relevant organizations, including territorial authorities or regional councils responsible for identifying risk areas and likely effects upon their constituent populations. Regional authorities are usually also responsible for ensuring that the public are, and remain, informed and educated throughout each of the eruption phases. Finally, local public health representatives and medical providers should participate in the assessment of the possible eruptive effects on human welfare.

FIGURE 7 Monitoring volcanic emissions. (Photo: Michael Durand).

Problems may arise, however, with the interpretation and integration of geological and medical information to ensure beneficial health outcomes. A number of failures to obtain and integrate health-related data have emerged during recent volcanic events, including the 1995–1996 Ruapehu eruptions in New Zealand (Davies et al., 1998). For example, during the height of Ruapehu's activity, numerous measurements of ash-fall and gas emissions were undertaken, but these data were used almost exclusively for geological purposes. There were few avenues available to usefully relay such information or to discuss its implications with public health officials or medical practitioners. Furthermore, air monitoring of poisonous gases following eruptive activity was difficult, because many areas had no existing facilities in place to test pollutant levels during non-eruptive periods. Data regarding water contamination from falling ash was often unavailable because testing was completed at supply level, thus excluding many catchment areas that were vulnerable to ash-fall (such as small streams serving remote communities).

A review of recent disaster responses has therefore indicated a degree of mismatching between the acquisition of eruption data and its health-related utilization. Given the diverse range of effects of volcanism on humans and ecosystems, full and accurate risk assessment requires integration of data collected by numerous ministries, departments, and regional authorities. Protocols for sharing information and achieving a consensus should be established *prior to* a volcanic event. For an effective response, joint consultation and action should involve organizations responsible for agriculture, fisheries, forestry, water provision, power generation, environmental management, and health. Furthermore, consensus needs to be achieved regarding the process of information transfer to the public, which includes the nature and frequency of health-related information, the organization that should impart the message, and the most appropriate media to use.

Geological data which may be generated before, during, and subsequent to eruptions therefore represent only the first step in the process of decreasing "volcanogenic morbidity." The ideal end result would involve information collected from monitoring (e.g., gas production at the vent) being disseminated to all relevant regional organizations and health providers, and then to vulnerable communities. Recommendations

TABLE IV. Strategies for Monitoring Gas (and Aerosol) Emissions: A Health Protection Perspective

Monitored component of gas emission	*Location of monitoring*	*Optimal time for monitoring*	*Health-related objectives of monitoring*
CO_2	a. At vent	a. At regular intervals preferably daily	a. To minimize risk of asphyxiation from downhill flow of CO_2 into populated areas
	b. In areas where CO_2 (heavier than air) can pool	b. If the areas need to be accessed	b. To minimize risk of asphyxiation from CO_2 collecting in low-lying areas
	c. Soil degassing	c. Continuously	c. Same as for a
SO_2	a. At vent	a. At regular intervals preferably daily	a. To provide early warnings to vulnerable populations
	b. Priority to emission-vulnerable areas with greatest population density	b. Continuously during eruption or in response to early warning	b. To minimize risks of respiratory disease, including exacerbations of asthma
H_2S	a. At vent	a. At regular intervals preferably daily	a. To provide early warnings to vulnerable populations
	b. Downwind in built up areas	b. Continuously during eruption or in response to early warning	b. To minimize risks of respiratory disease, including exacerbations of asthma
	c. In areas where H_2S (heavier than air) can pool	c. If the areas need to be accessed	c. To minimize risk of noxious asphyxiation
HCl and HF	a. At vent	a. At regular intervals preferably daily	a. To provide early warnings to vulnerable populations
	b. Priority to emission-vulnerable areas with greatest population density	b. Continuously during eruption or in response to early warning	b. To minimize risks of respiratory disease, including toxic lung injury
	c. Areas where much water is collected from roofs	c. Continuously during eruption or in response to early warning	c. To minimize effects of acid rain dissolution of metal roof components (which may release heavy metals into drinking water)
Air pH?	a. At vent	a. At regular intervals preferably daily	a. To give indication of expected pH of ambient air downwind
	b. Priority to emission-vulnerable areas with greatest population density	b. Continuously during eruption	b. To minimize the effects of acidic aerosols on respiratory disease, including asthma
Radon	a. At volcano	a. Continuously	a. To minimize possible long-term sequelae of radon exposure, such as lung cancer

communicated to the public should take into account practical realities (including economic or geographical constraints) and should be simple, coherent, noncontradictory, and delivered in a prompt fashion. From a medical perspective, geologic monitoring has a pivotal, but often underutilized, role in helping those living in the shadows of volcanoes. In keeping with the integrative aims of this book, the authors hope that this chapter will help to encourage the cross-disciplinary use of geological and medical data from locations where populations are exposed to volcanic eruptions. Such an approach offers an opportunity not only to advance scientific understanding, but ultimately to better protect public health.

SEE ALSO THE FOLLOWING CHAPTERS

Chapter 2 (Natural Distribution and Abundance of Elements) · Chapter 10 (Radon in Air and Water) · Chapter 12 (Fluoride in Natural Waters) · Chapter 15 (Selenium Deficiency and Toxicity in the Environment) · Chapter 20 (Animals and Medical Geology) · Chapter 23 (Environmental Pathology)

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