Health Impact of Volcanic Emissions

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http://dx.doi.org/10.5772/intechopen.73283

Abstract

Volcanoes form along the edges of tectonic plates. Although it is true that volcanic eruptions are destructive, these eruptions also have benefits. Volcanic eruptions provide minerals to the surrounding soil, and these minerals are beneficial to agriculture and can be used as building materials. Exposure to volcanic emissions can threaten the health of inhabitants in many ways; dermal and ocular irritation, cardiopulmonary exacerbations in people who suffer from chronic diseases, and even cancer have been linked with exposure to volcanic emissions. When rainwater passes through volcanic ashes deposited on land surfaces, the leaching of metals leads to significant changes in the chemistry of the surface water, increasing the risk of drinking water and land contamination. In addition to the health effects, volcanic eruptions are known to lead to surface cooling at the regional and global scales because of the emission of fine ash particles; however, these emissions, being a source of sulfates, contribute to acid rain formation. Because volcanic ash is highly abrasive, this ash can lower visibility and cause considerable damage to the engines of transportation systems. To avoid fatalities, it is important to closely monitor volcanic activity and promote a culture of prevention at all levels of society.

Keywords: volcanic emissions, health impact, cardiopulmonary disease, environmental impact, public policies

1. Introduction

Volcanoes form along the edges of tectonic plates, making up a cluster known as the Ring of Fire (Figure 1). Currently, there are about 100 active volcanoes all over the world, and more than a 100 million people are potentially exposed to volcanic emissions, which pose a threat to
the health of those exposed [1]. As a consequence of an eruptive event, a diversity of volcanic material rises to the surface (Figure 2). As shown in Table 1, the emission of volcanic material threatens the safety of populations in two different ways. In addition, volcanic emissions are a global concern due to the impact of these emissions on health, property and climate [2]. A volcanic plume is a mixture of liquid and solid particles produced during the eruptive event; volcanic plumes can reach the stratosphere depending on the intensity of the eruption, which is measured as the volcanic explosivity index (VEI). The chemical reactions within the plume are intense, and aerosol particles formed by nucleation remain in the plume and are transported by the wind across long distances (Figures 3 and 4) [3, 4].

Humans experience multiple types of exposure (respiratory and gastrointestinal tract, skin contact) to volcanic emissions. The respiratory system is the most susceptible because of the large quantities of air we breathe (20 m$^3$/day). Moreover, the impact of such exposure depends on different factors; essentially, the physical and chemical characteristics of the toxic substances (e.g., heavy metals, salts, metal oxides, inorganic carbons, silicates, plastics or organics) are in question. In addition, the age, sex, respiratory pattern and health status of the exposed person are also determinant factors. Among the physical characteristics, the size of the ash particles emitted by volcanoes, which can be less than 2 mm (Figures 5–7), and the number, concentration and density of the particles as well as the dynamics of the gas flow in the airways will determine the region where the ash particles will be deposited and will determine the local adverse effects [5]. The health effects of the volcanic emissions depend on the physical and chemical characteristics of the emissions and on the corresponding toxicological properties. The health effects are a consequence of the inhalation of particles directly emitted from active volcanoes or of resuspension of the soil ashes during the cleanup after the eruption. The effects that such exposure has on human health can be classified as acute or chronic. Figure 8
shows the interaction of some components of volcanic emissions within the respiratory tract and the possible effects caused by these components coming in contact with segments of the respiratory tract.

Acute effects include eye and throat irritation, cough, dyspnea, wheezing, chronic obstructive pulmonary disease (COPD), cardiovascular events, psychological stress, reversible changes in healthy lung function, and acute exacerbations of previously existing respiratory conditions such as asthma [6, 7]. As the lung is not a closed system, tiny particles (<0.1 μm) called ultrafine particles (UFPs) can translocate to other organs, where these particles can have adverse effects [5]. In addition, components of the ash particles can be dissolved in the

![Figure 2. Material emitted during volcanic emissions, extracted from https://pubs.usgs.gov/fs/fs002-97/](https://publish.crcpress.com/doi/10.5772/intechopen.73283)
lung-lining fluid and can pass through the alveolar-capillary membrane; this interaction explains, at least in part, the relationship between particle exposure, cardiovascular disease and some neurological alterations [8]. Chronic effects are related to increased mortality rate for cardiopulmonary disease [6], increased medication use (e.g., asthma medication, analgesics) [9], and increased prevalence of some types of cancer (e.g., lip, oral cavity, pharynx and female breast cancers) [10]. Moreover, neurodegenerative diseases such as Alzheimer’s disease have also been linked to chronic exposure to volcanic emissions [11]. Animal models have reported that volcanic emissions are associated with impairment of spermatogenesis [12].

Toxicological studies have suggested that exposure to volcanic ash could also have a negative impact on immunological defense and could increase the occurrence of biological redox reactions [13], which could lead to more frequent infections and to organic damage [14] (Table 2). In addition to the inherent physical and emotional disruptions caused by eruptive events, the economic impact of these events has severe consequences at a regional level because the people most impacted are those who belong to low-income groups. In this chapter, we summarize the toxicological aspects of volcanic emissions and the impact that a potential multimedia exposure has on health, with emphasis on inhalation exposure.

2. Volcanic emissions

The type and physicochemical characteristics of volcanic emissions vary depending on the volcano. In fact, these characteristics and types depend on the volcanic morphology and the
Figure 3. Dynamics of volcanic emissions. Sulfur dioxide emitted during volcanic eruption can reach the stratosphere altering the chemistry of the atmosphere. https://volcanoes.usgs.gov/volcanic_ash/gases_aerosols.html.

Figure 4. Volcanic plume dispersion from Popocatépetl, México. NASA image courtesy Jeff Schmaltz, LANCE MODIS rapid response and Robert Simmon. Caption by Michon Scott. Mexico’s Popocatépetl volcano released a plume on March 8, 2013. The ash plume reached roughly 1500 m (4900 feet) above the volcano crater, or about 7 km (4 miles) above sea level. The plume blew eastward away from the volcanic summit. https://earthobservatory.nasa.gov/NaturalHazards/view.php?id=80621.
geological features of the region where the volcano is located. Generally, volcanic ashes are composed of magmatic fragments, consisting of both glass and minerals.

The mineralogical composition of volcanic ash consists of approximately 45–75 wt% silica (SiO₂) [15], making SiO₂ content useful as a classification parameter (Figure 9). There are also other major components in volcanic emissions, such as water vapor, hydrogen peroxide (H₂O₂), carbon dioxide (CO₂), sulfur dioxide (SO₂) (the dominant sulfur component), hydrogen sulfide

Figure 5. A sample of tephra erupted by Mount St. Helens on May 18, 1980. The tephra was collected between about 40 and 60 km downwind from the volcano. USGS image, D.E. Wieprecht. http://volcanoes.usgs.gov/lmgs/jpg/Tephra/30410914_075_caption.html.

Figure 6. Ballistic tephra fragment from Popocatépetl volcano (May 14, 2013). CENAPRED image. http://www.cenapred.unam.mx/pop0/2015/mar/p03041510.png.png.
(H_2S) (the second most important S species, converted to SO_2 in the atmosphere), sulfates (SO_4^{2-}), and carbonyl sulfide (COS) and its precursor carbon disulfide (CS_2).

COS has a residence time of several years in the atmosphere and is an important source of sulfate aerosols. The main halogen component of volcanic ash is hydrogen chloride (HCl), which is highly soluble and is rapidly washed out from the atmosphere. Hydrogen fluoride

| Cause | Effect |
|---|---|
| Decreases in C3 and C4 complement | Reduce phagocytosis processes and cellular activation |
| Impairs autophagy in human alveolar macrophages | Impairs bacterial killing, increase susceptibility to bacterial infections |
| Decrease activation of JNK and ERK pathways | Promote inflammation |
| Inhibitory effect of antimicrobial peptides and defensins 1 and 2 | Decrease bactericidal capacity |
| Induce cytokines TNF-α, IL-8, IL-1β | Promotes inflammation |
| Reduction in T-cell response | Impairs T-cell response, impairs antigenic presentation |
| Reduction in T-cell proliferation | Impairs T-cell response |
| Decreases immunoglobulines (IgG) | Immunity humoral altered, unknown |

Table 2. Effects of volcanic ash exposure on immune system responses.
(HF), which is another ash component, may be dangerous because it is introduced into the alimentary chain mainly through contaminated water, not to mention that fluoride is an emergent toxicant affecting smooth organs (e.g., lungs and kidney) [16]. Volcanic ash is a source of helium (He), radon (Rn), mercury (Hg), magnesium (Mg), manganese (Mn) and bromide (Br), increasing the risk for people who have difficulty in absorbing essential elements such as Ca, Fe and Zn and retaining other elements such as Cd and Mn [17].
3. Epidemiological impact of volcanoes

The effects of exposure to volcanic emissions depend on the summit elevation and topographic features and on the intensity and duration of volcanic activity as well as the population density near volcanoes. This last parameter determines the difference between volcanic eruptions being a spectacle or a natural disaster. It has been reported that 4.7 million people have been affected by volcanoes all over the world and a total of 14,726 injuries (range 11,549–17,917) and 91,834 deaths (range: 85,169–102,372) result from volcanic eruptions between 1900 and 2009 [18]. In this context, exposure to CO₂ emissions was responsible for 30 and 1700 deaths at Monoun and Nyos lakes, respectively [19, 20]. In Mexico, the Popocatepetl volcano is part of the trans-Mexican volcanic belt and represents a high-risk zone because of the human settlements that surround the volcano, which include the states of Mexico, Morelos and Puebla, which are 65, 41 and 45 km away, respectively, from the volcanic crater. The Popocatepetl volcano reawakened on December 21, 1994, after a dormancy period of 70 years; the volcano remains active, with a VEI of 3–4 being registered in January 2001. In addition to the impact that a Popocatepetl eruption could have on crops and wildlife, the 2010 population census indicated that there are 173,928 people living approximately 30 km from the crater of Popocatepetl [21] who could potentially be directly affected by the eruption. Furthermore, the ashes produced by a major eruption could affect an area containing more than 20 million people and have a large economic impact [22, 23].

4. Impact on morbidity

During volcanic eruptions, there are multiple interactions that occur between the sets of chemical products emitted into the atmosphere. These chemicals are commonly referred to as “vog,” a term that is a combination of the words “volcanic” and “fog” and is frequently used by volcanologists. Vog is composed of sulfuric acid (H₂SO₄); ammonium bisulfate (NH₄HSO₄) and ammonium sulfate (NH₄)₂SO₄; and solid material, less than 2.5 μm in size (PM₂.₅), that is rich in sodium sulfate, ammonium sulfate and sulfuric acid aerosols.

The impact of volcanic emissions on morbidity and mortality has been described by several authors; however, some effects cannot be fully explained by vog exposure. For example, Michaud et al. [24] found a close relationship between emergency department visits in Hilo, Hawai’i and at the Kilauea volcano from January 1997 to May 2001 and occurrences of asthma/COPD, cardiac conditions, flu, cold and pneumonia. However, after adjusting for day of the week and month of the year, only visits for asthma/COPD remained associated with air quality, which was measured as SO₂ and PM₁ levels. Furthermore, the association with SO₂ levels was stronger with a 3-day lag and that with PM₁ levels was stronger with a one-day lag. In another study, it was reported that 3 weeks after the eruption of the Guagua Pichincha volcano in Quito (Ecuador), the emergency room visits increased due to acute pediatric respiratory infection and asthma, mainly in children under 5 years old [25].

In Hawai’i, Shinkura et al. [26] determined a relative risk of 2.2 for neonatal mortality associated with the average monthly SO₂ concentrations. Volcanic emissions greatly contribute to
the environmental SO₂ concentrations in some cities, such as Miyakejima (Japan). In this regard, it is worth mentioning the results from the Yorifuji study [27], where the odds ratio of having a baby with low birth weight was 1.71 if there had been SO₂ exposure during the entire pregnancy period, including exposure to SO₂ derived from volcanic emissions.

These highly irritant species are associated not only with respiratory effects but also with ocular effects. Kimura et al. [28] evaluated the effects of volcanic ash on the ocular symptoms of 10,380 children aged 6–15 years who lived near Mount Sakurajima (Japan). The study was carried out from 1994 to 2003. The authors concluded that ocular symptoms, such as redness, discharge, foreign-body sensation and itching, were influenced by volcanic eruptions, and stronger correlation was found with those who lived 4 km away from the volcanic crater.

Camara and Lagunzad [29] also evaluated the negative impact on residents of the island of O’ahu in Hawai’i who were exposed to volcanic emissions from Mount Kilauea for at least 7 years. The authors found a combination of toxic and allergic reactions, characterized by signs and symptoms such as conjunctival injection, papillary reaction, clear mucous discharge, pulmonary edema, lid swelling, chemosis, itchiness, foreign-body sensation, tearing and burning sensation. Some of these symptoms were present in 100% of the people studied. The authors introduced the term “vog-induced conjunctivitis (VIC)” and recommended that physicians refer patients to an eye specialist when the patients exhibit the aforementioned signs and symptoms.

Cardiovascular effects have also been observed in people exposed to volcanic emissions; nonmedicated, nonsmoking and nonobese participants had significantly faster mean pulse rates, as did those aged ≥65 and having a body mass index < 25 kg/m², and a higher mean systolic blood pressure was observed in nonobese participants. Interestingly, indoor concentrations of volcanic air pollution were higher than the exposure limits recommended by the World Health Organization (20 μg/m³, ~7.5 ppb, 24-h mean for SO₂) [7]. As mentioned earlier, volcanic emissions include metals, and some of these metals are essential for life and participate in a wide range of biochemical reactions.

In addition, toxic heavy metals are also released and deposited in the airways and hair and can be retained in tissues, increasing the inflammatory, oxidative stress, DNA damage response and, ultimately, the carcinogenic response [10, 12, 30].

Adults and children living in active-volcanic areas of Azores (Portugal) and Etna (Italy) have shown significant increase in scalp hair concentrations of metals and metalloids such as Cd, Cu, Pb, Rb, Zn, V, U and, to a lesser extent, As and Mn [31, 32], and the risk of chronic bronchitis for the people living in the volcanically active area was much higher (males RR = 3.99; females RR = 10.74) than that for those living in volcanically inactive areas [10].

The literature reports two cases of lung damage associated with the inhalation of fresh volcanic ash [33, 34]. In both cases, the medical conditions improved, and the patients were allowed to go home. In the first case, the authors did not follow up, but in the second case, the patient returned to the hospital 6 months later with the shortness of breath and worsening chest pain; the authors associated the lung symptoms with a kind of silicosis.
5. Impact on the respiratory system

Volcanic gases affect people even after they return to their homes. This is the case with the citizens of Miyakejima who returned to the island four-and-a-half years after the eruption of the Mount Oyama in Japan. The volcanic emissions were mainly composed of SO$_2$, the levels of which reached 80,000 tons/day during the peak period. Although emission rates of SO$_2$ decreased after the initial eruption, the levels remained high compared to those at Sakurajima in Japan, Kilauea in Hawai‘i, and Stromboli and Etna in Italy [35–38]. Before the residents of Miyakejima returned to the island, the Tokyo Metropolitan Government established the Scientific Committee for the Assessment of Health Risks and Volcanic Activity. Iwasawa et al. [39] and Kochi et al. [40] started a follow-up study and evaluated the impact on the respiratory health of the residents. The authors found that the mean SO$_2$ concentration in the air ranged from 6.64 to 12.6 ppb with a maximum concentration between 1580 and 1880 ppb from November 2006 to 2011. The authors observed clear dose-response relationships with symptoms of irritation and established a threshold concentration of approximately 70 ppb in adults. In addition, in children aged 6–18 years, the authors observed a clear exposure-dependent increase in respiratory symptoms such as throat and eye irritation and/or pain, and the threshold concentration was established to be 30 ppb for this group.

Longo et al. [41] found a statistically significant positive association between chronic exposure to SO$_2$ and fine sulfate particles (PM$_{2.5}$) emitted from the Kilauea volcano (Hawai‘i) and increased prevalence of cough, phlegm, rhinorrhea, sore/dry throat, sinus congestion, wheezing, eye irritation and bronchitis. The chronic exposure to volcanic SO$_2$ emissions increased the risk of acute bronchitis in children aged 0–14 years, with a cumulative incidence ratio of 6.56 [41]. However, Tam et al. [42] found that chronic exposure to respirable acidic particulates is associated with a reduction in the FEV1/FVC ratio; however, this effect was not statistically significant. The authors did not find any association with the diagnosis of asthma or with persistent wheezing or bronchitis in the last 12 months in the group studied.

Rojas-Ramos et al. [43, 44], in a 7-month prospective study of 80 nonsmoking farmers exposed for 1- to 5-day periods to volcanic ash from the Popocatépetl volcano, found an increase in the occurrence of respiratory symptoms such as sore throat, cough, expectoration and dyspnea. Of the 80 exposed people, 44 showed a restrictive spirometric pattern and two showed an obstructive pattern; the proportions of these symptoms were considered to be higher than those observed in persons exposed to tobacco and wood smoke. After 7 months of exposure, the lung function returned to normal values.

In contrast, Benítez et al. [45] did not observe significant differences in spirometric parameters before and after volcanic eruption in Bariloche, Argentina. The data obtained before and after the event did not correspond to the same individual, which could lead to an underestimation of the effect.
6. Toxicological effects

In vitro and in vivo toxicological studies of volcanic emissions have been performed and have focused on cytotoxicity, immunological response, production of reactive oxygen species and pulmonary response.

The physicochemical characterization of ash particles from different volcanoes is important in toxicological studies. Volcanic ash particles have a wide distribution in size and composition. Due the high content of SiO$_2$ in volcanic ash, many investigators have been interested in the fibrotic effects.

It is well known that particles less than 10 $\mu$m (PM$_{10}$) in size, which are found in volcanic ash, can enter the respiratory tract, including the alveolar region with a deposit of 10% of the ash particles; the tracheobronchial region with a range of 1–4.5% of ash deposit; and the head airways with a deposit of 15–60%, increasing the risk of in situ lung damage (Figure 8) [46]. Furthermore, the deposition of ash particles along the respiratory system could cause systemic alterations because the smallest particles can translocate from airway tissues to other organs via the blood stream and to the brain via the axon extensions in the nose [47, 48].

Studies in mice exposed to volcanic emissions have provided evidence of increased alveolar damage (decrease in alveolar space and perimeter, increase in alveolar septal thickness, inflammation and particle deposits) compared to mice exposed to environmental pollutants [49]. Inflammatory lung reactions were noted in lymph nodes after 13 weeks of exposure to volcanic ash, and the reaction lasted until 49 weeks after exposure [50].

One single instillation of ash from Mount St. Helens (40 mg) was performed in female fisher rats that were then killed at 109 days; the exposure induced an inflammatory response, leading to increased lung and lymph node weight. A mononuclear cell inflammatory reaction, cell necrosis, hyperplasia of type-II pneumocytes and evident damage of type-I epithelial cells were observed in the lungs. Microgranulomas were observed in rats exposed to volcanic ash, and a significant increase in fibrosis was observed. These responses suggest that the toxicity of ash is associated with the levels of crystalline silica. The toxicity of ash from Mount St. Helens on lungs has been extensively studied and reviewed by Martin et al. [51]. Discordant results have also been obtained by several authors. For example, THP-1 macrophages exposed to volcanic ash from Mount St. Helens had low levels of LDH release, minimal suppression of cellular metabolism, and negligible apoptosis and necrosis as well as IL-1$\beta$ production [52, 53]. The last result was not consistent with the reported cristobalite toxicity and concentration in the ash from Mount St. Helens.

Since macrophages in the lung constitute the first line of defense to foreign substances and materials, and the mineral content of volcanic ash particles determines the bioreactivity and the intensity of immunological response, these results could indicate a gradual change from an acute to a chronic inflammatory process that involves the biochemical and biomolecular modification (e.g., proteins or lipids) of the lung-lining fluid, altering the protective effects of this fluid [54]. The bactericidal capacity of macrophages is also impaired, which could be related to the type and levels of metals that can affect the activity of antimicrobial peptides.
present in the lung-lining fluid [55, 56]. Dodson et al. [57] reported an electron-microscopic analysis of interaction of ash with human macrophages from smokers and nonsmokers, and these authors did not observe morphological changes associated with cytotoxic effects of the eruption of Mount St. Helens.

The exact mechanism by which volcanic ash could damage lung tissue is not clear, and the literature shows contradictory results. One of the proposed mechanisms involves the generation of reactive oxygen and nitrogen species, especially hydroxyl radicals (HO●) [58], related to interactions with some metals, both essential and nonessential to biochemical and physiological functions [59], that are present in volcanic ash [60]. Horwell et al. demonstrated that the production of hydroxyl radicals from volcanic ash was independent of the iron content of basaltic volcanic ash, but the production was associated with the uncoordinated iron ions exposed at the surface of the ash [38].

Volcanic ashes from Eyjafjallajökull and Grímsvötn were not oxidative and had low acute cytotoxicity (20–30%) in human alveolar type-1-like epithelial cells (TT1) after a 24-h exposure to 50–1000 μg/ml, and a pro-inflammatory response was observed, with increased levels of Il-6, Il-8 and MCP1 after acute exposure when evaluated at 24 and 96 h [61].

The cytotoxic effect of Monserrat volcanic ash was tested in the A549 cell line via the evaluation of the mitochondrial reduction of tetrazolium salts (MTT assay). The authors reported a reduction in cell viability (20–30% with respect to cell controls) of the cell line exposed to 500 μg/ml for 24 h [62].

We have not found reports of in vitro cytotoxicity studies with the ashes of Popocatepetl. Experiments that are ongoing in our laboratory on the cell viability of the A549 cell line exposed for 48 h to ash collected in 2013 from Popocatepetl showed, by using the MTT assay, elevated, concentration-dependent cytotoxic effects (Figure 10) (Aztatzi-Aguilar et al., in preparation). The cytotoxic effect of ash from Popocatepetl could be attributable to the high fluoride content reported by Armienta et al. in ash leachates from the Popocatepetl volcano (5–513 mg/kg).

Figure 10. Cytotoxic effect of Popocatépetl’s ash on A549 cell line. Exposure of A549 cell line to volcanic ash showed an increase in cell death in a dose-dependent manner (MTT assay).
and by D’Addabbo et al. in ash from Mount Etna (10 mg/L) [63, 64]. Rodents exposed to ash from Popocatepetl at 10 mg/m³ for 60 days (4 h/day) showed reduced body weight and changes in differential blood count, with increased lymphocyte counts and reduced neutrophil counts [65]. D’Addabbo et al. did not observe any toxicity of Popocatepetl ash leachates on *Xenopus laevis* embryos; however, the authors observed a 40% increase in larval malformation [64].

Volcanic ashes from Mt. Vesuvius led to decreased lung antioxidant defenses, had low acute cytotoxicity in human alveolar type-1-like epithelial cells and had a chronic pro-inflammatory response [61]. This effect could lead to an increase in alveolar-capillary permeability, which correlates with lung injury and with impaired gas exchange [66]. People living near volcanoes are also exposed to anthropogenic pollutants; the exposure to volcanic ash from the Soufrière Hills volcano mixed with diesel exhaust particles (DEP) increased the pro-inflammatory response of cellular culture, as determined by the enhanced release of TNF-α, IL-8 and IL-1β [67]. Discordant results have also been obtained by several authors. For example, THP-1 macrophages exposed to volcanic ash from Mount St. Helens had low levels of LDH release, minimal suppression of cellular metabolism, and negligible apoptosis and necrosis as well as IL-1β production [52, 53]. The last result was not consistent with the reported cristobalite toxicity and concentration in the ash from Mount St. Helens.

### 7. Geotourism

In 2002, the Travel Industry Association of America introduced the term geotourism, which is increasing in popularity and represents a new trend in travel. Unfortunately, this kind of travel carries the risk of injuries ranging from mild cuts and abrasions to thermal burns, broken bones, hypothermia and altitude sickness, which are life-threatening conditions [68–70]. Lung emphysema associated with exposure to high concentrations of SO₂ or CO has been reported as a cause of death in some tourists [71, 72].

In addition to geotourism, such fatalities have also been reported in workers during winter months, when snow can block volcanic vents, making them nonvisible. Moreover, snow can trap toxic fumes and form dangerous, gas-filled pits [73].

### 8. Environmental effects

The effects of volcanic emissions are not limited to living organisms. There is plausible information regarding stratospheric ozone depletion related to the emission of hydrochloric acid from major explosive eruptions [74], where volcanic ash can play the role of a temporary atmospheric reservoir of chloride salts that may generate reactive chlorine species, contributing to catalytic O₃ destruction [75]. Furthermore, the reactivity and activation of volcanic ashes are influenced by the mineralogical and chemical composition of the ashes [76]. Additionally, it is important to note that the acidification of surface waters caused by ash precipitation can
increase the leaching of toxic elements such as fluoride and arsenic, leading to the poisoning of aqueous environments and increasing the risk of fluorosis [63, 77, 78].

9. Conclusions

There is no doubt that people will continue to live near volcanic flanks, and this is for many reasons:

1. Because of poor urban planning.
2. Because governments and stakeholders fail to provide alternatives.
3. Because citizens cannot afford a safer place to live in.
4. Because of the influence of religion on people’s behavior and risk perception.

Whatever the cause, governments must work together with residents to identify the hazards in the environment and the vulnerabilities of the communities in order to improve public policies to lower the risk from volcanic exposure or other natural disasters.

Acknowledgements

This work was supported by National Council of Science and Technology (CONACYT) grant FOSISS-SALUD-220014-01-233950 and CÁTEDRA-CONACYT-280. The authors wish to thank Drs. Ana Lillian Martín Del Pozzo and Hugo Delgado Granados from the Department of Volcanology of the Institute of Geophysics, National Autonomous University of Mexico (UNAM), for supplying the ash samples.

Conflict of interest

The authors declare that they have no competing interests.

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