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Review

Living Under the Volcano: Effects on the Nervous System and Human Health

Alicia Navarro-Sempere [1], Raúl Cobo [1], Ricardo Camarinho [2,3], Patricia Garcia [2,4], Armindo Rodrigues [2,3], Magdalena García [1] and Yolanda Segovia [1,*]

[1] Department of Biotechnology, Faculty of Sciences, University of Alicante, 03690 San Vicente del Raspeig, Spain; alicia.navarro@ua.es (A.N.-S.); raul.cobo@ua.es (R.C.); m.garcia@ua.es (M.G.)
[2] FCT, Faculty of Sciences and Technology, University of the Azores, 9501-801 Ponta Delgada, Portugal; ricardo.ad.camarinho@uac.pt (R.C.); patricia.v.garcia@uac.pt (P.G.); armindo.s.rodrigues@uac.pt (A.R.)
[3] IVAR, Institute of Volcanology and Risks Assessment, University of the Azores, 9501-801 Ponta Delgada, Portugal
[4] cE3c—Centre for Ecology, Evolution and Environmental Changes & CHANGE—Global Change and Sustainability Institute/Azorean Biodiversity Group (cE3c-ABG), University of the Azores, 9501-801 Ponta Delgada, Portugal
* Correspondence: yolanda.segovia@ua.es

Abstract: Volcanoes, during their explosive and post-explosive phases, as well as through continuous degassing processes, release a range of pollutants hazardous to human health, including toxic gases, fine particulate matter, and heavy metals. These emissions impact over 14% of the global population living in proximity to volcanoes, with effects that can persist for days, decades, or even centuries. Living conditions in these regions often involve chronic exposure to contaminants in the air, water, and soil, significantly increasing the risk of developing neurological disorders. Prolonged exposure to elements such as lead (Pb), mercury (Hg), and cadmium (Cd), among others, results in the accumulation of metals in the brain, which increases oxidative stress and causes neuronal damage and severe neurotoxicity in animals. An examination of metal accumulation in brain cells, particularly astroglia, provides valuable insights into the developmental neurotoxicity of these metals. Moreover, microglia may activate itself to protect from cytotoxicity. In this review, we consider the implications of living near an active volcano for neurotoxicity and the common neurodegenerative diseases. Additionally, we encourage governments to implement public health strategies and mitigation measures to protect vulnerable communities residing near active volcanoes.

Keywords: volcanogenic pollution; health effects; neuroinflammation; heavy metals; neurotoxicity

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1. Introduction

Pollution is the leading environmental cause of illness and premature death worldwide [1]. According to the WHO, diseases caused by chronic exposure to environmental pollution were responsible for 4.2 million premature deaths in 2019. The relationship between chronic exposure to environmental pollution and the risk of developing various diseases is widely documented in the scientific literature. In this context, air pollution stands out as a complex chemical mixture originating from multiple sources, including emissions from engines, coal combustion, biomass burning, and secondary photochemical products such as ozone, as well as natural sources. Living in urban areas with high pollution levels has been linked to an increased risk of cognitive decline in old age [2,3];

Alzheimer's disease [4]; Parkinson's disease [5]; various lung conditions such as asthma, obstructive diseases, and fibrosis [6]; and cardiovascular conditions, which represent between 40 and 60% of premature deaths due to air pollution [7]. The most vulnerable groups to the harmful effects of air pollution are the elderly, pregnant women, children, and individuals with pre-existing conditions [8]. Currently, there is extensive literature on the health effects of anthropogenic air pollution. However, it is important to note that not all air pollution originates from human activities; there are natural sources, some of which contribute significantly to air pollution globally, such as volcanic systems [9].

Throughout history, humans have maintained a dual relationship with volcanoes: on the one hand, fear due to the danger posed by volcanic eruptions; on the other, the benefit derived from the use of their soils, known for their high fertility. This is, in fact, the main reason why many communities, roughly 14.3% of the global population, settle near active volcanoes. This statistic highlights the relevance of volcanism for society in terms of human health and environmental quality.

The scientific community has historically been concerned with the effects of volcanic activity, focusing on volcanic eruptions and other associated phenomena such as seismic activity, which can be catastrophic. However, in recent years, there has been an increase in research focusing on the effects of exposure to gases and other contaminants released by volcanoes during post-eruptive periods, as these emissions can persist for years, decades, or even centuries. During periods of quiescent activity, volcanic contaminants are released into the environment through fumaroles, thermal springs, or diffuse soil degassing, making these forms of volcanism a significant source of environmental pollution [10,11]. Furthermore, these pollutants can be transported thousands of kilometers and can have effects even at low but constant concentrations.

Similar to pollution caused by human activities, volcanic contaminants have an impact on the health of people who are in sustained contact with them. These types of contaminants primarily affect the respiratory system, as inhalation is the main entry point into the body [12]. Furthermore, chronic exposure to active volcanic environments has been linked to a higher incidence of chronic bronchitis [13] and other respiratory conditions in humans [14]. Changes in histomorphology [15,16] and proinflammatory processes [17] have also been described in the bronchi of exposed animals.

Not only have effects on the respiratory system been observed, but damage has also been evaluated in the reproductive system [18] and even an increase in the incidence of certain types of cancer, such as lip, oral cavity, pharynx, and breast cancer [19]. In the central nervous system, which has also been studied in animals chronically exposed to volcanic contaminants, there has been documented accumulation of metals, such as mercury and lead [20,21], as well as changes in nervous tissue consistent with neuroinflammatory processes [22,23] (Figure 1).

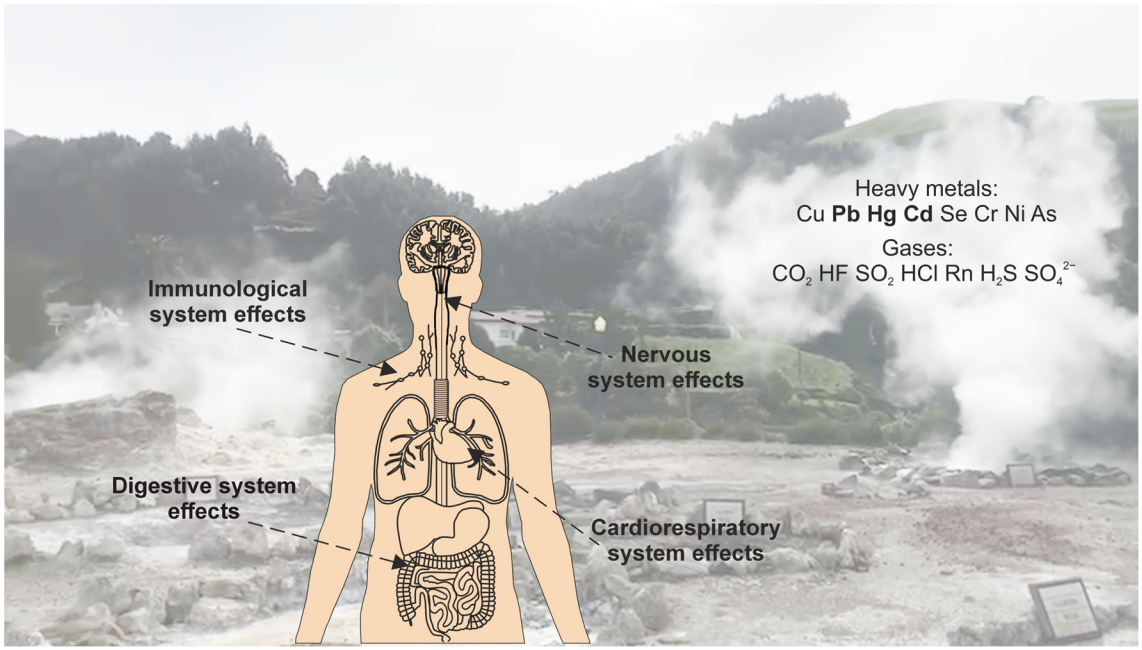

Figure 1. Effects of volcanic-origin contaminants on health.

2. Volcanogenic Pollutants

Volcanic pollution can exert detrimental and long-lasting effects on populations situated kilometers away from its source [24], impacting a broad range of geographical regions [25]. These emissions consist of complex mixtures of gases (H_2O, CO_2, SO_2, H_2S, CO, HF, HCl and ^{222}Rn), heavy metals in vapor form, and particulate matter, which may be released during volcanic eruptions as well as inter-eruptive periods via fumaroles, hot springs, and diffuse soil degassing [9,26]. Given that geothermal activity can persist for extended periods, and the perceived benefits of hot springs encourage human settlements, there is a heightened risk of toxic exposure to these pollutants. Furthermore, the interaction of these contaminants with the atmosphere may exacerbate environmental challenges such as climate change and the acidification of water bodies, which can, in turn, adversely affect aquatic life, harming ecosystems such as coral reefs, fish populations, and aquatic vegetation [27]. Similarly, acidification can compromise water quality, limiting its suitability for human consumption, agriculture, and industrial applications [28].

In this context, Weinstein et al. [29] emphasize that these pollutants also interact with organisms at the histopathological level, potentially causing damage to cells and tissues through various pathways: direct contact with skin and mucous membranes, interaction of particles and gases with the upper respiratory tract, metabolic disruption, or genotoxicity.

2.1. Gases

Carbon dioxide (CO_2)

CO_2 is the second most abundant volcanic gas after water vapor [26]. This noxious gas is, therefore, a constant hazard both during volcanic eruptions and in dormant or inactive volcanic regions [30]. Concentrations of CO_2 below 5% are not lethal but are sufficient to accelerate respiration by stimulating the brain's respiratory centers. At concentrations between 5% and 10–11%, individuals may experience lethargy and headaches [31]. Finally, concentrations exceeding 10% can be fatal [32]. Houses built on diffuse degassing zones in volcanic areas can accumulate CO_2 emanating from the soil, reaching dangerous indoor concentrations [33,34].

Sulfur dioxide (SO_2)

It is a colorless and irritant gas whose presence and effects in volcanic areas can vary depending on the quantity emitted, the atmospheric layer into which it is released, and the climatic conditions of the region [31]. This gas causes both acute and chronic pathologies, particularly affecting the respiratory system, and is one of the main contributors to chronic bronchitis in active volcanic regions [35]. Prolonged exposure to this gas can lead to epithelial hyperplasia and submucosal hypertrophy in the respiratory system [36].

Hydrogen sulfide (H_2S)

It is a colorless, harmful, and asphyxiating gas with a distinctive odor of rotten eggs, a characteristic aroma in volcanic regions with fumarole fields. According to the Agency for Toxic Substances and Disease Registry, inhalation of the gas rapidly affects vital systems such as the respiratory, cardiovascular, and nervous systems during short-term exposure (up to approximately 1–2 weeks) to high concentrations (>150 ppm) and can cause long-term health effects or death. At low concentrations, it can cause conjunctival irritation and depressive states [37], whereas at high concentrations, it irritates the upper airways, leading to pharyngitis, bronchitis, and even pulmonary oedema [31]. Moreover, a positive association between H_2S exposure and nervous system diseases has been reported [38].

Carbon monoxide (CO)

It is a harmful and asphyxiating gas that reaches high environmental concentrations in areas near fumaroles in active volcanic areas [29]. At low concentrations, this gas is already toxic to humans as, once inhaled, it rapidly penetrates red blood cells and binds to the iron component of hemoglobin, forming carboxyhemoglobin. This affinity is 200-times greater than that of oxygen, which quickly reduces the ability of erythrocytes to transport it. As red blood cells become saturated with CO, the individual experiences headaches and nausea, which can progress to coma and death if not treated promptly.

Hydrogen fluoride (HF)

It is a colorless and irritating gas for the skin, eyes, and mucous membranes. It is also commonly associated with volcanic activity [39]. Once it enters the body through respiration, it accumulates in the bones. In small doses, it can be beneficial, but high quantities or prolonged exposure to low doses can lead to fluorosis in teeth and bones [31]. The systemic effects of hydrogen fluoride are due to increased fluoride concentrations in the body, which can alter levels of calcium, magnesium, and potassium in the blood. In addition to the musculoskeletal system, excess HF has been linked to effects on development and on the renal, endocrine, and nervous systems [39].

Hydrogen chloride (HCl)

At ambient temperature, hydrogen chloride is a colorless or slightly yellow, corrosive, non-flammable gas, heavier than air, with a strong irritating odor. It is one of the most corrosive gases emitted by volcanic systems, which even at low doses, causes inflammation, hyperplasia, and ulceration of the nasal mucosa, larynx, and trachea. Although not classified as carcinogenic, due to its aggressiveness and acidic nature, it can induce oxidative stress, particularly in respiratory system cells [31].

Radon (^{222}Rn)

Radon is a naturally occurring radioactive gas that is produced by the decay of uranium, thorium, and radium in soil, rock, and water. In volcanically active environments, it is continuously vented to the atmosphere from fumarolic fields or diffused through soil. During the radon decay process, alpha, beta, and gamma radioactive particles are released and can be inhaled and deposited on the bronchial epithelium of exposed individuals;

considering that the alpha particle can disrupt the DNA structure within cells of the lining epithelia, and especially lung cells, exposure to this irradiation can contribute to an increased risk of lung cancer [40]. Inhabitants of geothermal areas, who are chronically exposed to radon, have an increased risk of developing mutations in epithelial cells, which are associated with carcinogenesis [41]. Additionally, exposure to this element has also been considered a risk factor for nervous system pathologies, such as multiple sclerosis [42] and primary brain tumors [43].

2.2. Particulate Matter and Heavy Metals

According to the WHO [44], particulate matter (PM) is one of the most concerning pollutants due to its detrimental effects on human health and its heterogeneity in variety, form, and size. Particles are classified based on their diameter into: PM10 (diameter < 10 μm), PM5 (diameter < 5 μm), PM2.5 (diameter < 2.5 μm), PM1 (diameter < 1 μm), ultrafine (diameter < 100 nm), and nanoparticles (<50 nm). Volcanic systems are a significant source of these particles, releasing everything from nanoparticles to coarse particles into the atmosphere [45–48].

PM can generate a wide range of adverse health effects, including respiratory, cardiovascular conditions, and damage to the central nervous system [49–53]. Although the molecular interaction between PM and the body is not yet fully understood [54], it has been identified that particles can directly damage respiratory system cells. Furthermore, they trigger inflammatory processes through the secretion of reactive oxygen species (ROS), causing oxidative stress, activation of inflammatory cells, and secretion of pro-inflammatory cytokines, thereby contributing to a cascade of pathological responses [55].

Simultaneously, volcanic systems emit not only PM but also heavy metals, which pose a significant risk to human health [37]. These metals include lead (Pb), mercury (Hg), copper (Cu), zinc (Zn), selenium (Se), and cadmium (Cd) [29]. Although short-term exposure to these metals can cause adverse health effects, the main concern lies in their ability to accumulate in tissues and organs, leading to severe health consequences [56]. This accumulation can even disrupt the central nervous system, causing various disorders [57,58].

3. Impacts on Central Nervous System

Prolonged exposure to environmental pollutants leads to a constant accumulation of ROS and other pro-inflammatory mediators, such as cytokines, which contribute to oxidative stress and trigger neurodegeneration and cell death processes. The CNS is particularly vulnerable to these alterations due to its high oxygen consumption, which accounts for approximately 20% of the total absorbed by the body, and its high composition of polyunsaturated fatty acids, which are susceptible to lipid peroxidation. Furthermore, the selectivity of the blood–brain barrier limits the diffusion of essential antioxidants, such as vitamin E, exacerbating its susceptibility to oxidative damage [59].

The resulting oxidative stress affects key mechanisms, such as structural damage to DNA, proteins, and lipids, contributing to the development of neurodegenerative diseases like Alzheimer's [60–62], Parkinson's disease [63–65], and multiple sclerosis [66–69]. Additionally, it has been documented that these exposures affect neurodevelopment, increasing the prevalence of autism spectrum disorders [70–72].

Among volcanic-origin pollutants, heavy metals pose a significant threat due to their environmental persistence, bioaccumulation capacity, chemical stability, and high toxicity [73–75]. In the human body, these metals are transported and accumulated in cells and tissues by binding to proteins and nucleic acids, destroying macromolecules and altering cellular functions [76]. These alterations can affect multiple organs, even with low-dose exposures [77]. The CNS is particularly sensitive to the toxicity of heavy metals,

which tend to bioaccumulate in this tissue, triggering severe neurological disorders [78] (Table 1). Recent studies have demonstrated the presence of metals such as mercury (Hg) in the brain [20,21] and in the spinal cord [79], as well as lead (Pb) and cadmium (Cd) in the brain [21] of mice exposed to active volcanic environments. These metals generate free radicals that promote oxidative stress and cellular damage [80]. They also form stable complexes with protein thiol groups, altering enzymatic activity by blocking the synthesis of essential products and exacerbating cellular damage [76,81]. Furthermore, they interfere with proper protein folding, particularly the refolding of chemically denatured proteins, as reported for Pb, Cd, Hg, and As [82].

Table 1. Volcanic metal pollutants: sources, entry pathways, and CNS effects.

Metal	Key Volcanic Sources	Route(s) of Exposure	Main CNS Effects
Mercury (Hg)	Emitted as vapor (elemental, inorganic, methyl) via fumaroles/hot springs	- Inhalation (~80% lung absorption) - Crosses BBB	- Chronic neuroinflammation - Neuronal damage, glial activation - Associated with neurodegenerative disorders
Lead (Pb)	- Volcanic emissions (quiescent/eruptive) - Present in ash and soils	- Inhalation (main route, up to 90% retention) - Secondary via ingestion/dermal contact	- Cognitive deficits, neuronal death - Hippocampal/cortical accumulation - Neuroinflammation (TNF-α, IL-1β)
Cadmium (Cd)	- Released by volcanic activity - Present in soils/fumarolic gases	- Inhalation leading to bloodstream transit and BBB crossing	- Oxidative stress, astrocyte dysfunction (↑GFAP) - Hippocampal damage (dentate gyrus) - Increased neurodegenerative risk
Other Metals (Cu, Zn, Se)	- Frequently co-emitted with Pb, Hg, Cd - Detected in volcanic ash, gases	- Inhalation (often adsorbed onto PM) - Possible dietary intake	- Toxic in excess, disrupt neuronal homeostasis - Can induce oxidative stress - May exacerbate effects of Pb, Hg, Cd

Abbreviations: BBB, blood–brain barrier; CNS, central nervous system; GFAP, glial fibrillary acidic protein; IL, interleukin; PM, particulate matter; TNF-α, tumor necrosis factor alpha.

Mercury

Mercury is released into the environment through various sources, with volcanic activity being one of the primary natural origins [83]. This element is found in nature in air, water, and soil in three main forms: elemental (Hg^0), inorganic (Hg^+; Hg^{2+}), and organic or methylmercury (MeHg) [84]. Hg^0, although liquid at room temperature, quickly volatilizes into Hg^0 vapor, with this form being much more dangerous than the liquid one [85]. This gas is absorbed at 80% by the lungs when inhaled [86], circulating throughout the body via the bloodstream as it passes through the plasma membrane of blood cells. This gaseous form of mercury can cross the blood–brain barrier (BBB) [87] as well as the placental barrier [88]. Although absorption through the gastrointestinal tract is also possible, inhalation has been confirmed as the primary route of entry in animals living in active volcanic areas, as mercury accumulations have been found in the lungs of these organisms [12]. Mercury induces toxicity by damaging mitochondria through the depletion of glutathione [89], a powerful endogenous antioxidant. This mitochondrial dysfunction reduces ATP synthesis

and increases the peroxidation of lipids, proteins, and DNA [90]. These factors appear to play a key role in the development of conditions such as amyotrophic lateral sclerosis and Alzheimer's disease [89,91,92]. Also, prolonged exposure to this heavy metal can induce neurodevelopmental alterations [93–96].

Lead

This heavy metal is among the agents emitted by a volcanic system, even during its quiescent stage. Although it can be absorbed through the skin, its entry into the body mainly occurs through the gastrointestinal tract and the respiratory system [85]. Once inhaled, up to 90% of the lead particles present in the air are retained in the body [97]. Once in the lungs, this metal is captured by resident macrophages, which generate Pb^{2+} ions in their phagosomes that can be released. These ions travel through the bloodstream until they reach the BBB, which they are able to cross [98]. The high vulnerability of the nervous system to lead has already been described in the literature [99]. In the brain, this metal is deposited and accumulates in specific areas such as the hippocampus or the cerebral cortex [100]. It has already been reported in the literature that continuous exposure to Pb induces the activation of astroglia and microglia [101]. This gliosis creates a neuroinflammatory environment characterized by the release of pro-inflammatory cytokines like IL-1β or TNF-α, and the activation of microglia [102,103], which can lead to neuronal death [104]. Also, Pb exposure has been associated with the formation of amyloid plaques due to an increase in beta amyloid protein in nerve tissue [105]. In line with this, a relationship between early-life Pb exposure and susceptibility to developing neurodegenerative diseases during adulthood has been suggested, specifically for Alzheimer's disease [106].

Cadmium

Volcanic activity has been identified as one of the main sources of emission of this metal into the atmosphere [107]. In animals living in active volcanic areas, inhalation has been confirmed as the primary route of exposure, as evidenced by the detection of cadmium in the lungs of these organisms [108]. Its impact on the CNS is particularly relevant since, once inhaled, cadmium reaches the BBB via the bloodstream, which contains transporters and receptors that facilitate the entry of this heavy metal into the nervous system [109,110]. Once in the CNS, Cd can alter synaptic function, modify neurotransmitter signals and induce mitochondria disruption. Thus, Cd creates an oxidative stress environment in the CNS, initially affecting astrocytes, which respond by increasing the expression of glial fibrillary acidic protein (GFAP) [73]. Although the CNS has antioxidant mechanisms that provide protection against acute exposures, these are insufficient in cases of chronic exposure [111,112].

In addition to the direct effects on the CNS, environmental pollution can also influence the gut microbiota, a microbial community crucial for overall health and neurological function [113,114]. The gut–brain axis, a bidirectional communication pathway between the gut and the brain, is affected by changes in the microbiota [115], which can be induced by environmental pollutants, including heavy metals emitted by volcanic systems.

Exposure to pollutants such as Hg, Pb, and Cd can alter the composition and diversity of the gut microbiota, leading to dysbiosis, a condition characterized by microbial imbalance that compromises vital functions, such as neurotransmitter production, regulation of systemic inflammation, and maintenance of the intestinal barrier [116,117]. This imbalance can increase intestinal permeability, facilitating the passage of pro-inflammatory mediators and toxins into the bloodstream, which triggers systemic inflammatory responses that affect the CNS. Changes in the gut microbiota have been associated with alterations in the brain and with neurodegenerative disorders [115]. Recently, microbial metabolites derived from the gut have been explored for their potential to modulate the blood–brain barrier [118],

which could offer new therapies to maintain its integrity and improve brain physiology. Moreover, it has been demonstrated that pollution-induced dysbiosis contributes to the development of neurological diseases such as Alzheimer's, Parkinson's, and autism spectrum disorders [117]. In a recent study, Frye et al. [70] found a significant relationship between air pollution exposure during pregnancy and altered mitochondrial metabolism in children with autism spectrum disorder, which could increase the risk of developing diseases or make them more susceptible to future triggering factors. Additionally, a connection has been identified between exposure to air pollution and the composition of the infant gut microbiome, which could have important implications for health and early development [119]. In this context, the gut microbiota may act as a critical mediator in the effects of pollutants on the CNS, amplifying their impact through systemic inflammation and the disruption of key metabolic pathways.

Overall, the interaction between environmental pollution, the gut microbiota, and the gut–brain axis emerges as a central mechanism in the neurotoxic effects of pollutants, highlighting the urgent need for research exploring interventions based on microbiota modulation as an effective strategy to mitigate these adverse effects.

Cellular Mechanisms Involved

Volcanic pollutant-induced neuroinflammation is a key process in neurotoxicity. The exposure to these emissions activates glial cells such as astrocytes and microglia, triggering inflammatory responses in the CNS [22,23]. In this process, activated microglia adopt a pro-inflammatory phenotype that amplifies oxidative stress and cell death, while astrocytes contribute to the homeostatic imbalance of the extracellular environment, exacerbating neuronal dysfunction. These mechanisms, along with the persistence of pollutants in neural tissue, perpetuate a cycle of chronic inflammatory damage.

Microglia

Microglia are the resident immune cells in the CNS and are considered the first line of defense. Although their activation is a protective response for the brain, sustained or chronic activation can lead to irreversible damage within the CNS [120]. Microglia rapidly detect alterations in the CNS, such as the presence of pathogens, apoptotic neurons, misfolded proteins, and even environmental pollutants [121–124]. In response to these stimuli, microglia transition from a resting state, characterized by a small soma and long processes [125], to an activated state, adopting an amoeboid morphology with larger somas and shorter processes. In addition to this morphological change, activated microglia secrete pro-inflammatory cytokines, such as IL-6 and TNF-α, as well as ROS and other molecules that are damaging to the CNS microenvironment [126]. Environmental pollution is now well recognized in the scientific literature as a source of neuroinflammation and microglial activation [127,128]. Chronic exposure to these pollutants has been shown to cause changes in microglia within the CNS, such as amoeboid-shaped cells [128–132], increased expression of the marker IBA-1 [74,129,130,133–138], and CD68 [127,139–141], as well as elevated levels of the cytokine TNF-α [74,129–131,133,134,142–144]. These changes in microglial cells, resulting from exposure to anthropogenic air pollutants, have also been observed in the dentate gyrus of the hippocampus in animals chronically exposed to natural pollutants, such as those of volcanic origin [23]. Thus, microglial activation is evident in individuals chronically exposed to these environments, and although such activation is essential for repairing damaged tissue, prolonged activation may prove detrimental to neuronal populations.

Astrocytes

Astrocytes play a fundamental role in the CNS, performing essential functions such as maintaining homeostasis and cellular defense. One of their main tasks is participating in the formation of the blood–brain barrier through their astrocytic end-feet, which enables them to regulate the transport of molecules and, in this way, ensure the nutrition and support of neurons. Additionally, astrocytes have a crucial role in eliminating excess neurotransmitters, particularly glutamate [145], with approximately 90% of this neurotransmitter being cleared by these cells [146]. This function is especially relevant because excess glutamate leads to excitotoxicity, a common feature in various neurological disorders.

On the other hand, astrocytes also act as immune-competent cells, capable of detecting threats in the CNS and responding by proliferating and secreting cytokines [147]. As such, they are considered key players, along with microglia, in neuroinflammatory processes. In this context, chronic exposure to atmospheric pollutants is a known source of neuroinflammation, which may lead to alterations in the astrocytic population of the CNS. To assess the proliferation of these cells and, consequently, the presence of reactive astrogliosis, the expression of the GFAP protein is studied [148], with its expression having been increased in response to exposure to anthropogenic atmospheric pollutants [72,127,149–151]. In line with these studies, Navarro et al. [22] reported an increase in GFAP expression in the dentate gyrus of the hippocampus in animals exposed to active volcanic areas, also noting a change in the morphology of these astrocytes.

These animals exhibited longer astrocytic processes compared to those from a reference region, a morphology considered reactive and associated with neuroinflammatory processes [152]. However, unlike GFAP expression, these individuals showed a decrease in the expression of the enzyme glutamine synthetase. This enzyme is crucial for astrocytes to metabolize glutamate into glutamine and prevent excitotoxicity, so its reduction indicates astrocytic dysfunction and an inability to perform one of their most important functions. Indeed, alterations in the expression or activity of glutamine synthetase have been linked to various neurodegenerative diseases, such as Alzheimer's disease [153].

4. Conclusions

Exposure to volcanic pollutants, such as toxic gases and heavy metals, induces significant neurotoxic effects on the CNS. These pollutants cause oxidative stress and chronic inflammation, promoting the development of neurodegenerative diseases. Moreover, the impact on the gut microbiota and its connection to the brain exacerbates these effects, highlighting the need to investigate strategies to mitigate this health damage in volcanically active areas.

5. Limitations and Future Perspectives

A major limitation of this study is the lack of research specifically addressing the neurotoxic effects of volcanic pollutants on the central nervous system (CNS). Existing studies are geographically concentrated and often lack comprehensive, longitudinal approaches, which hinders the extrapolation of findings to other volcanically active regions and limits our understanding of the underlying mechanisms. This gap highlights the urgent need for interdisciplinary research that integrates environmental science, toxicology, and neuroscience to better evaluate the risks associated with chronic exposure to volcanic emissions.

Despite the significant health impact of these pollutants, chronic exposure to volcanic pollution has largely been overlooked by both the scientific community and policymakers. To enhance public health management, raising awareness of the risks associated with volcanic emissions is essential. A key initial step would be to expand the installation of

sensors capable of detecting gases and atmospheric pollutants, generating real-time data on air quality. Furthermore, advancing the precision of exposure assessment methods, along with improving meteorological and dispersion models, is necessary. As practiced in other regions worldwide, providing real-time information on pollution levels would be highly beneficial, utilizing communication channels such as radio, television, and social media to ensure the public receives this information promptly [154]. Additionally, understanding the local geochemical context is critical, as imbalances in chemical elements—both from diet and atmospheric aerosols—can lead to health disorders, including effects on the nervous system [155]. Therefore, knowledge of local geochemical patterns is essential for identifying potential risks and their underlying causes. In this framework, epidemiological and biochemical studies should be conducted, including blood, urine, and stool analyses, as well as evaluations of the gut microbiota, allowing for the implementation of further measures based on the results to more effectively safeguard public health.

By addressing these limitations through enhanced research and public health initiatives, we can establish a stronger evidence base to develop targeted strategies for mitigating the neurotoxic impacts of volcanic pollutants and protecting vulnerable communities residing near active volcanoes.

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