Review Article

Air Pollution and the Auditory System at Risk: A Narrative Review

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Short running title: Air Pollution and the Auditory System at…

Highlights:

- Air pollution impact peripheral auditory system, leading to both conductive and SNHL
- Air pollution can adversely affect the CANS and auditory-cognitive disorders

ABSTRACT

Background and Aim: Air pollution has a negative impact on various body systems, including auditory system. The aim of this study was to provide a comprehensive review of research investigating the effects of air pollution on various levels of the auditory system, including peripheral and central components, as well as related cognitive processing and associated disorders.

Recent Findings: Air pollution includes a diverse and complex mixture of chemicals, particulate matter or biological substances in ambient air. Air pollution can be harmful to humans and other living things. It has become a major environmental concern in recent decades, especially in developing and industrialized countries. There is a lot of evidence showing that air pollution can harm different parts of the body, like the lungs, heart, brain, and ears. Hearing loss is the second most common non-infectious disorder, and its effects can lead to serious issues in communication, speech, language, education, social interactions, finances, and mental well-being.

Conclusion: This study examines various research studies that link air pollution to hearing problems, highlighting how air pollutants can harm different aspects of the auditory system.

Keywords: Air pollution; auditory system; central auditory nervous system; hearing loss; otitis media; sudden sensory neural hearing loss

Introduction

The term "air pollution" refers to the presence of foreign substances in the air that alter the usual composition of its components. The things that cause air pollution are called air pollutants. Air pollution usually means there's a mix of Particulate Matter (PM), chemicals, and sometimes living things in the air around us that can be harmful or unpleasant for people and animals. These pollutants come from both natural events like volcanic eruptions and human activities like industry [1]. Over the past decade, air pollution has become a significant environmental issue, especially in both developed and developing nations. Studies have shown clear connections between the amount of air pollution and things like car exhaust, factory emissions, population density, burning fossil fuels, farming, and power plant operations [2].

Because millions of people are constantly exposed to air pollutants at levels higher than what's considered legally safe [1], air pollution continues to be a major public health problem worldwide, as it is making people sick and causing deaths. It's estimated that air pollution accounts for about 2.5% of all deaths in developed countries, making it the 8th biggest risk factor for mortality [3]. The World Health Organization estimates that air pollution causes over 3 million premature deaths every year [4]. Studies have found a clear link in observational and epidemiological research between being exposed to outdoor air pollution and negative health effects like increased deaths and hospitalization [5]. Research has shown that exposure to air pollution is linked to a higher risk of cardiovascular problems like reduced blood flow to the heart, heart failure, and irregular heartbeats, as well as respiratory issues like lung cancer and asthma, and an increased chance of death [4]. The Central Nervous System (CNS) is another part of the body that could be harmed by air pollution. Recent research, including observational, epidemiological, experimental, and clinical studies, suggests a possible connection between air pollution and neurological conditions like Alzheimer's Disease (AD) [6, 7], Parkinson's Disease (PD) [8], and stroke [9]. Some scholars also argue that air pollution increases the risk of Sudden Sensorineural Hearing Loss (SSNHL) [10], Conductive Hearing Loss (CHL) [11], Sensorineural Hearing Loss (SNHL) [12], and central hearing loss [13].

The phenomena of urbanization and industrialization are increasing air pollution and its consequences worldwide [1]. On the other hand, the communication issues that people with auditory disorder face cause irreversible harm to their personal, professional, educational, economic, social, and psychological well-being [14-18]. This manuscript reviews the findings of available epidemiological, observational, clinical, and experimental studies on the link between air pollution and the auditory system to improve our understanding of the adverse impacts of air pollutants on the auditory system.

Methods

We conducted a thorough search across multiple databases, including Medline (PubMed), Google Scholar, Science Direct, and the Cochrane Library, without any date restrictions. The keywords used in the search were: “air pollution”, “central auditory processing”, “conductive hearing loss”, “otitis media”, “sudden sensorineural hearing loss”, and “sensorineural hearing loss”. In addition to the database searches, we reviewed the references of the selected papers to ensure that all relevant studies were included. All papers identified through this process were reviewed, and a subset was selected based on the following inclusion criteria: 1) Papers with an accessible abstract or full text; 2) Papers related to peripheral and central auditory processing disorder; 3) Papers written in English.

Results

Initially, we selected 84 relevant papers. From this collection, we purposefully sampled 73 papers that were most pertinent to the topic of air pollution and the auditory system, ensuring they met our inclusion criteria. This thorough search and selection process enabled us to compile a strong body of literature, enhancing our understanding of how air pollution and various airborne particles impact the health of both the peripheral and central auditory systems, as well as related cognitive processing and associated disorders.

Discussion

Air pollutants

Air pollution is a diverse mixture of materials such as gases, e.g. ground-level ozone, Carbon Monoxide (CO), Sulfur Oxides (SO_x), and Nitrogen Oxides (NO_x), MP, organic compounds (e.g. bacterial endotoxins and polycyclic aromatic hydrocarbons), and toxic metals (e.g. vanadium, copper, manganese, nickel, and lead) [3]. It should be noted that the most prevalent hazardous air pollutants are PM and ground-level ozone, which are mostly made up of NO_x and Volatile Organic Compounds (VOCs) [19].

Particulate matter

PM is characterized by its size and aerodynamic properties, which are closely linked to its biological consequences. For example, larger particles typically get trapped in the upper airways, and only particles smaller than 10 μm can be inhaled deeply into the lungs. PM is generally divided into three categories including coarse particles with an aerodynamic diameter of 2.5 to 10 μm (PM_{10}), fine particles with an aerodynamic diameter less than 2.5 μm ($PM_{2.5}$), and Ultrafine Particles (UFPs) or Nanoparticles (NP) with an aerodynamic diameter less than 0.1 μm. [1]. The main sources of PM_{10} include construction and demolition activities, tire wear emissions, wood combustion products, mining operations, and transportation and agricultural dust. Power plants, metal processing facilities, oil refineries, tailpipe emissions, brake wear emissions, residential fuel burning, and wild fires are the primary sources of $PM_{2.5}$ [3]. These particles consist of condensed high-temperature vapors and gases that are formed during the burning of fuel and other industrial processes. Sulfates, nitrates, hydrogen ions, carbon, ammonium, Lipopolysaccharide (LPS), water, and metals are examples of the organic and inorganic compounds that can make up $PM_{2.5}$ [1]. Power plants, internal combustion engines, incinerators, and other thermodegradation sources can produce UFPs. UFPs, which are mainly combustion-derived NPs, can carry oxidized transition metals, Dissolved Organic Matter (DOM), and Polycyclic Aromatic Hydrocarbons (PAHs) on their surface [20]. There is a direct relationship between mortality and ambient UFP concentrations. The distinct properties of these particles (e.g. inhibition of phagocytosis) make them more dangerous than other PMs [21]. UFPs pose a serious threat to human health due to their ability to pass through the nose and bronchioles without being filtered out. This allows the particles to enter the bloodstream, penetrate deep into the lungs, and disseminate throughout the body [1].

Gases

The planet is surrounded by a thin shell of gases, particles, and clouds called the atmosphere. Each year, billions of tons of pollutants are disposed of by humans into this thin shell. Fossil fuel combustion for the production of power, transportation, and cooking, and forest and savannah fires (wildfires) are the primary causes of air pollution. Carbon Dioxide (CO_2), a colorless gas, is the ultimate byproduct of all combustion processes. On the other hand, incomplete combustion products (e.g., CO and NO_x) can react with other gases in the atmosphere to form ozone, another greenhouse gas. In addition, the formation of primary aerosols and aerosol precursor gases, such as Sulfur Dioxide (SO_2), in the atmosphere is another consequence of energy consumption, which has direct adverse effects on both the ecosystem and human health [22].

Organic compounds

Incomplete burning of organic matter in the environment produces PAHs. These substances can be found in large quantities in the atmosphere as particulate, volatile, and semi-volatile pollutants. Major sources of these pollutants include domestic sources (e.g. household coal or wood combustion and environmental tobacco smoke), regional sources (e.g. forest fires and burning of agricultural residues), mobile sources (e.g. gasoline and diesel engine exhausts), and stationary sources (e.g. gas-fired and coal-fired power plants) [23]. Numerous PAHs and their epoxides are extremely mutagenic, toxic, and/or carcinogenic to microorganisms and higher systems such as humans. Multiple physicochemical techniques have been employed to eliminate these substances from the environment; however, their scope of use is quite limited. Though long assumed to be recalcitrant, xenobiotic-degrading microorganisms offer great promise for bioremediation; nonetheless, novel modifications are needed to make such microorganisms successful in eliminating these substances [24].

Toxic metals

Basic metal elements including nickel, lead, mercury, silver, cadmium, chromium, manganese, and vanadium are examples of heavy metals. These metals, which are natural components of the Earth's crust, cannot be degraded or eliminated. In addition, heavy metals can contaminate human food and water through the air. Additionally, these substances are released into the environment by sewage discharge, combustion, and manufacturing

facilities, etc. While the human body needs a small amount of these compounds to function normally, at higher (albeit still relatively low) concentrations, they can become poisonous [25]. The majority of heavy metals are regarded as dangerous because they have a tendency to bioaccumulate in the human body. The term "bioaccumulation" describes the gradual rise in a chemical substance's concentration within a biological organism relative to that of the environment. In other words, bioaccumulation occurs when chemicals are absorbed and stored more quickly than they are excreted [26].

Auditory system

The most prevalent sensory impairment in humans is hearing loss, which results in partial or complete loss of hearing in one or both ears. Factors such as the site and severity of the lesion/disease, the audiogram's shape, and the hearing loss's onset and progression are used to categorize different types of hearing loss. In normal hearing, sound vibrations are transmitted from the outer ear to the inner ear through the middle ear. In CHL, vibrations cannot go from the outer ear to the inner ear. SNHL occurs when there is a dysfunction in the inner ear (the cochlea and/or the auditory nerve). Finally, Mixed Hearing Loss (MHL) is a combination of CHL and SNHL [27]. Damages to regions above the inner ear and the auditory nerve (including the brainstem, midbrain, and the auditory cortex) result in central hearing loss. Hearing loss can occur suddenly or develop gradually over time. In addition, it may be mild or severe, transient or permanent, depending on the underlying reason. Furthermore, hearing loss can be unilateral or bilateral; it can also fluctuate, getting better sometimes and getting worse at other times. In other cases, hearing loss is constant and does not alter over time [28]. The impacts of air pollution on the different kinds of hearing loss are discussed in the following paragraphs. A schematic picture of the relationship between air pollution and the auditory system has been shown in Figure 1.

The relationship between air pollution and middle ear infection (otitis media)

One of the most prevalent illnesses in children, particularly during the first three years of life, is middle ear infection or Otitis Media (OM). By the age of 3, three out of four children experience OM [29, 30].

A massive body of evidence points to the link between high levels of air pollution exposure and an increased risk of upper respiratory infections and lower respiratory illnesses, which in turn elevates the risk of ear infections. Research also shows that exposure to tobacco smoke is among the main environmental risk factors for developing OM. Accordingly, exposure to SO_2 was found to impair ciliary function and mucus secretion in the middle ear of guinea pigs. On the other hand, little is known about how other ambient air pollutants affect OM. Children who are exposed to ambient air pollution have a higher risk of developing OM, according to some studies; however, risk levels vary in different studies [11]. In a study carried out in South Korea between 2011 and 2012, Park et al. observed a significant relationship between exposure to ambient air pollutants and the incidence of OM in children under the age of 15. PM_{10}, NO_2, and Ozone (O_3) had the highest negative impacts on children. The risk of developing OM was higher in higher concentrations of CO and SO_2 than in lower values. Most notably, there was a strong correlation between the incidence of OM and exposure to PM_{10}. They also found that exposure to PM_{10}, NO_2, O_3, SO_2, and CO increased the incidence of OM both immediately and one to four weeks later [31]. In a cross-sectional study in Brazil, a positive relationship was found between SO_2 levels and the prevalence of OM in children between the ages of 11 and 13 [32]. Research findings show a correlation between early life exposure to air pollution and the development of OM in children. This is particularly true for $PM_{2.5}$ during the second trimester of pregnancy and PM_{10} during the early postpartum period (especially the first year of life) [33]. In a systematic review, Vithayathil et al. observed that air pollution raises the risk of OM in both infants and children. It was unclear to what degree exposure to higher levels of NO_2, PM, SO_2, PAHs, and wood smoke contributes to the increased prevalence of OM infections. Furthermore, it was concluded that exposure to PM_{10}, NO_2, O_3, SO_2, and CO increases the risk of developing OM both immediately and 1 to 4 weeks later [34]. Although the impact of air pollution on OM has been highlighted in many studies, not all of the relevant studies have found associations between different air pollutants and OM. For instance, researchers found no connection between the incidence of OM and air pollution in studies carried out in Canada [23] and China [24].

The relationship between air pollution and sensorineural hearing loss

Several factors (e.g. viral infections, genetic mutations, accidents, ototoxicity, loud noises, and autoimmune diseases) can cause SNHL. In addition, there is a correlation between the incidence of SNHL and a number of underlying conditions such as Diabetes Mellitus (DM), Chronic Kidney Disease (CKD), Hypertension (HT), stroke, Ischemic Heart Disease (IHD), Chronic Obstructive Pulmonary Disease (COPD), nicotine dependence,

alcoholism, Rheumatoid Arthritis (RA), and asthma [12]. Some of these diseases can result in the incidence of hypoxia by impairing the vascular system and the cochlear microcirculation. It has been found that hypoxia harms inner ear neurons and Hair Cells (HCs) [35]. Chung et al. examined the effects of Nitrogen Dioxide (NO_2) and CO, the two main air pollutants linked to transportation, on human hearing. Their results indicated that both NO_2 and CO can potentially lead to SNHL. A long-term exposure to the highest quartile of NO_2 raised the risk of SNHL by 1.63 times, whilst exposure to the highest quartile of CO increased the risk by 1.45 times. Moreover, a moderate amount of NO_2 (but not CO) exposure increased the incidence of SNHL by 1.40 times. It was also found that NO_2 and CO damage cells via several mechanisms. NO_2 is among Reactive Nitrogen Species (RNS). Both Reactive Oxygen Species (ROS) and RNS cause cell apoptosis. Conversely, CO damages organisms irreversibly by impeding the efficient flow of oxygen, particularly to vital organs like the heart and brain [12].
Oxidative stress and inflammation are two of the primary risk factors for SNHL development. NO_2, as one of the most important traffic-related air pollutants, can be hydrolyzed to nitric acid and nitrous acid, both of which can induce inflammation through lipid peroxidation and oxidative stress. Intratympanic gentamicin use may lead to a hearing threshold shift after two days. This hearing impairment is linked to increased NO_2 concentrations (a stable oxidation product of NO_2) in the lateral wall, indicating that NO_2 may play a role in the gentamicin-induced hearing impairment [36]. Furthermore, factory workers who are simultaneously exposed to noise and organic solvents or heavy metals have an extremely increased chance of developing SNHL [37]. The risk of developing SNHL is higher when people are simultaneously exposed to noise and heavy metals or toxic substances than when they are exposed to each pollutant separately [38]. It is worth mentioning that both air pollution and noise pollution may damage the cochlear HCs through oxidative stress [39]. and hypoxia-induced ROS [40].

The relationship between air pollution and sudden sensorineural hearing loss

Sudden Sensorineural Hearing Loss (SSNHL) is defined as sensorineural hearing loss of at least 30 dB in three consecutive frequencies. SSNHL is considered as an emergency that occurs within 72 hours. Although many hypotheses have been proposed to explain the etiology of SSNHL (e.g., viral, vascular, and autoimmune pathologies), the etiopathogenesis of SSNHL is still unknown [41].
Considering that Upper Respiratory Infection system (URIs) are regarded as major etiological triggers of Idiopathic SSNHL (ISSNHL), meteorological conditions may have an impact on the onset and prognosis of ISSNHL patients. It has been found that climatic changes suppress the immune system, and thereby contribute to the pathogenesis of a number of illnesses such as SSNHL [42]. Studies have produced conflicting results regarding the impact of meteorological conditions on the incidence of ISSNHL [43-45].
A weak correlation was found by Lee et al. in a study conducted in Busan, South Korea, between the average daily PM_{10} and $PM_{2.5}$ concentrations and the number of hospitalization days experienced by SSNHL patients [45]. In a study in South Korea, Choi et al. investigated the effects of air pollution parameters on the onset of SSNHL. They observed an association between NO_2 concentrations and development of SSNHL [10]. Their findings revealed that with an Odds Ratio (OR) of 3.12, SSNHL was associated with a lag effect of the 14-day NO_2 concentration (0.1 ppm).
The impact of NO_2 on intracochlear Nitric Oxide (NO) concentrations alters intracochlear neurotransmission and neuromodulation. NO plays an essential role as a signaling molecule in blood vessels, gap junctions, and the synaptic region of the cochlea. As a result, increased NO concentrations raise the risk of developing SSNHL by impairing hearing [10]. It should be noted, though, that NO_2 can potentially cause SSNHL, as it is considered a major risk factor for developing URIs [46].
In a Taiwanese study, the risk of developing SSNHL was higher in individuals who were exposed to moderate or high levels of $PM_{2.5}$ over a long period of time than in those who experienced a low level of exposure to $PM_{2.5}$. In addition, long-term exposure to elevated levels of NO_2, NO, and CO increased the risk of developing SSNHL in participants [47]. In a retrospective study, Karali and Gunes examined the impacts of meteorological factors and air pollution parameters on the prognosis of 40 ISSNHL patients who had clinic treatment between 2015 and 2018. Researchers analyzed meteorological data from 10 days before the emergence of the disease and 14 days after the treatment. The data included relative humidity, maximum and minimum temperature (T_{max} and T_{min}), mean temperature (T_{mean}), and air pollution parameters (SO2 and PM_{10}). Their findings indicated that the prognosis of ISSNHL patients may be influenced by T_{mean}, T_{max}, T_{min}, and relative humidity. Moreover, no connection was found between ISSNHL recovery rates and exposure to various SO_2 and PM_{10} concentrations [42].

In a study in Taipei, Cheng et al. examined the relationship between exposure to air pollution and the development of SSNHL. They found correlations between the frequency of hospitalizations per day for SSNHL and the lag effects of exposure to O_3, NO_2, and $PM_{2.5}$ [46].

Air pollution leads to the development of SSNHL through several mechanisms such as oxidative stress, toxicity, and upregulation of inflammatory pathways. The following are potential mechanisms by which air pollutants can cause hearing impairment. Inhaled air pollutants disrupt the redox signal, cause neuroinflammation and oxidative stress with an imbalance between oxidants and antioxidants [48]. ROS impairs cochlear blood flow, while endothelial dysfunction lowers perfusion by reducing nitric oxide and raising nuclear factor activity and interleukin expression. According Capaccio et al. study, oxidative stress causes endothelial dysfunction in the inner ear microcirculation, which in turn leads to the development of SSNHL [49]. Research also suggests that URIs caused by the lag effects of NO_2, O_3, and $PM_{2.5}$ may increase the risk of developing SSNHL. Neuroinflammation and oxidative stress can destroy neurons by influencing the apoptotic pathway. In a recent study, the risk of cerebral ischemia was raised by oxidative stress and inflammatory responses following the inhalation of air pollutants. Cochlear ischemia increases the risk of developing SSNHL [46]. SSNHL development is also linked to cardiovascular diseases and exposure to air pollution raises the risk of cardiovascular diseases [50]. Additionally, studies reveal that NO_2 is a more important risk factor for myocardial infarction than PM_{10} and O_3 [46].

The relationship between air pollution and the central auditory system

Chemicals, metals, and environmental pollutants have negative and occasionally irreversible effects on the central nervous system development. The neurological function of today's children is affected throughout their lives by air pollution exposure, which starts in the uterus [51]. A study conducted on children who had grown up in areas with severe urban air pollution revealed that changes in the auditory pathways of these individuals are likely linked to neuropathological lesions or even metabolic abnormalities. These lesions may involve a build-up of structurally abnormal proteins, neuronal and axonal damages, and vascular, glial, and neuropil damages. The examination of the auditory brainstem potentials of children showed significant delays in waves III and V, and greater delays in interwave intervals I-III, III-V, and I-V, indicating a notable delay in the central conduction time of neural transmission through the brainstem. While transmission through children's cochlea and auditory nerve was normal, transmission through their cochlear nucleus (wave III), superior olivary complex (wave IV), and lateral lemniscus pathways (wave V) was abnormal. This suggested the involvement of many brainstem nuclei and pathways in the facilitation of auditory processing. In the studied children, factors including abnormal brainstem auditory evoked potentials, gait deviation, positive Romberg test, and dysmetria manifested the diffuse pathology of the brainstem, which involves the vestibular and cochlear nuclei as well as cerebellar connections (vestibular nucleus afferents) [13].

The relationship between air pollution and neurological disorders related to the central auditory system

About ten years ago, CNS was recognized as a target organ for the harmful impacts of airborne pollutants. Data from recent clinical, experimental, epidemiological, and observational study shows strong correlations between air pollution and some neurological diseases such as PD, AD, and stroke [1]. Examining the impact of air pollution on neurological diseases such as stroke, PD, and AD is of particular importance, because these diseases are linked to hearing and auditory processing disorders, and auditory impairments may predict the incidence of these neurological disease [52].

According to recent epidemiological research, there is a relationship between air pollution exposure and the incidence of PD. Direct neurotoxicity and induction of systemic inflammation (which causes inflammation of the CNS) are among the potential ways by which air pollution may raise the risk of developing PD [53].

Research findings introduce air pollution as a new risk factor for stroke. Air pollution levels have been steadily rising over the past few decades, and according to current estimates, air pollution accounts for 14% of all stroke-related deaths [54]. Given the strong temporal relationships of gaseous and particulate air pollutants with stroke-related hospitalization, enhancing air quality is believed to lessen the burden of stroke [55].

Strong interactions between unfavorable environmental factors and systemic, neurological, immunological, metabolic, and inflammatory responses most likely lead to the development of AD [56]. Research shows a strong correlation between exposure to $PM_{2.5}$ and a 138% rise in the risk of AD for every 4.34 $\mu g/m^3$ increase above the United States Environmental Protection Agency (USEPA) $PM_{2.5}$ standards (an annual average of 12.0 $\mu g/m^3$ over 3 years). In another study, AD and dementia were found to be substantially correlated with average annual $PM_{2.5}$

concentrations from fossil fuel combustion and transportation [57]. According to toxicological and epidemiological studies, exposure to elevated levels of SO_2 or CO raises the risk of developing dementia, suggesting that SO_2 or CO may trigger the development of AD [58]. In addition, recent findings suggest that young individuals who are exposed to combustion-generated fine particles and nanoparticles have a higher risk of developing AD [59]. Pathological AD markers, including processing areas and auditory nuclei (e.g., the vestibular nuclei, the Dorsal Cochlear Nucleus (DCN), and the Medial Superior Olive (MSO), were found in 99.5% of cases in a study of 203 consecutive autopsies of individuals between the ages of 1 and 40 (mean age: 25.36±9.23 years) who were chronically exposed to PM [60]. The summary of studies regarding the effect of air pollution on the auditory system has been shown in Table 1.

Conclusion

The current study showed that air pollution has a very big impact on the hearing system and increases the risk of developing peripheral and central hearing problems. In this review study, all the evidence related to the effect of air pollution on the incidence of middle ear infection, cochlear and nervous disorders including sudden hearing loss and central auditory system problems were presented. It was also found that air pollution increases the risk of neurological problems related to hearing, including alzheimer's disease, Parkinson's disease, and stroke. Because hearing loss has irreparable effects on the daily life of affected people and significant health costs, and on the other hand, air pollution is a global issue that has increased due to industrial processes in developing countries. The positive relationship between hearing loss and air pollution should be considered in prevention, early diagnosis and early intervention.

Ethical Considerations

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Authors' contributions

NG: Conceptualization, Study design, Data curation, formal analysis, investigation, validation and drafting the manuscript; ZHD: Study design, data curation, formal analysis, investigation, validation and drafting; AM and SFE: Data curation, investigation, and drafting the manuscript.

Conflict of interest

The authors declare no competing interests.

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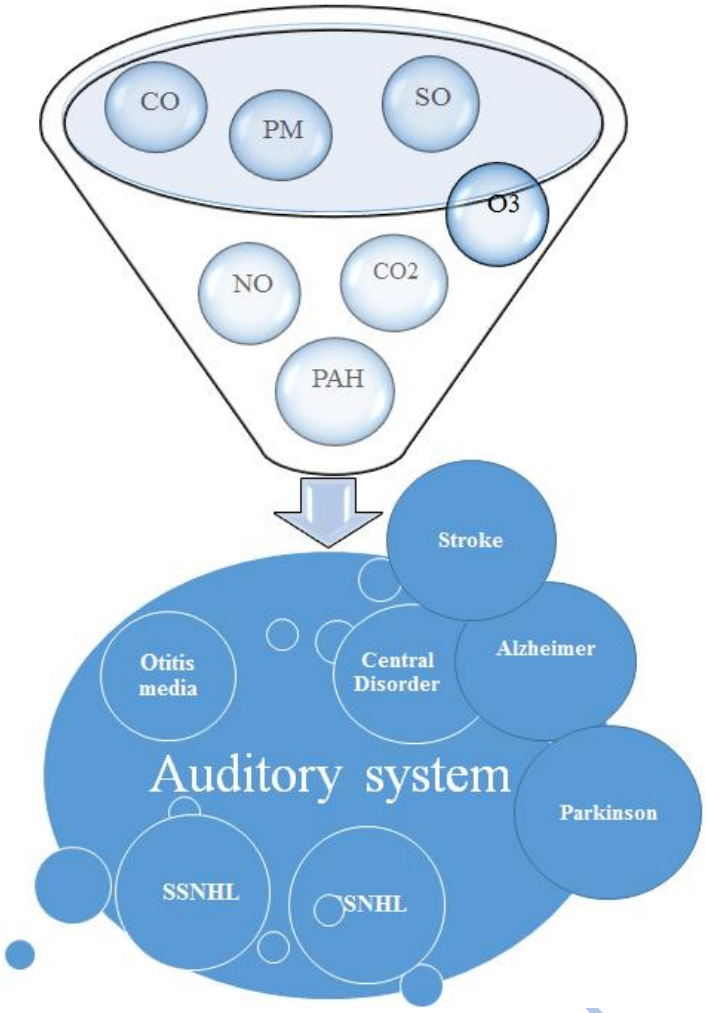

Figure 1. Schematic illustration of the relationship between air pollution and the auditory system. CO; carbon monoxide, PM; particulate matter, SO; sulfur oxide, O3; ozone, CO2; carbon dioxide, NO; nitrogen oxide, PAH; polycyclic aromatic hydrocarbon, SSNHL; sudden sensorineural hearing loss, SNHL; sensorineural hearing loss

Table 1. Summary of studies on the effect of air pollution on the auditory system

Number	Source	Year	Source of air pollution	Population	Population sample size and age range	Main findings
1	Calderón-Garciduenas et al. [13]	2011	O3, PM	Children	N=57, 96.3±8.5 m	There is a relationship between exposure to severe air pollution and neurological dysfunction of the auditory pathway, which can lead to hearing disorders and vestibular dysfunction.
2	Chang et al. [12]	2020	NO2, CO	Adult	N=75767, 54.1 y	NO2 and CO could have potential adverse effects on SNHL.
3	Zemek et al. [23]	2010	CO, NO2, O3, SO2, PM	Toddler	N=14527, 1–3 y	Increasing CO and NO2 levels can increase the prevalence of otitis media
4	Park et al. [31]	2018	PM10, NO2, O3, SO2, CO	Children	N=160875, age<15 y	Incidence of OM is associated with ambient air pollution.
5	Lu et al. [33]	2023	PM2.5, SO2, NO2, PM10	Preschooler	N=8689, 3–6 y	Early life exposure to air pollution is associated with childhood OM.
6	Lee et al. [45]	2019	SO2, CO, O3, NO2, PM2.5, PM10	Adult	N=817, 51.0 ± 16.5 y	There is a weak significant relationship between SSNHL and meteorological data.
7	Cheng et al. [46]	2022	PM2.5, NO2, O3, SO2, CO	Adult	N=850, 49.69±16.6 y	Exposure to air pollution can increase the prevalence of SS NHL.
8	Tsai et al. [47]	2021	PM2.5, SO2, CO, NO, NO2	Adult	N=64321, >20 y	An increased risk of SSNHL is associated with long-term exposure to air pollution.
9	Lin et al. [58]	2021	CO, NO2, PM10, SO2	Older adult	1. n=584, 79.23±7.84 y 2. n=120,78.20±6.69y	Air pollution, which is an environment-related risk factor that can be controlled for, is associated with cognitive deterioration of AD.

O3; ozone, PM; particulate matter, NO2; nitrogen oxide, CO; carbon monoxide, SNHL; sensorineural hearing loss, SO2; sulfur dioxide, OM; otitis media, SSNHL sudden sensorineural hearing loss