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Review Article

The Invisible Enemy: Nanoparticle Exposure and Neurodegenerative Decline

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Abstract

Atmospheric air pollution, specifically the ultra-fine fraction of particulate matter ($PM_{2.5}$) known as nanoparticles (NPs), poses serious global health threats with critical neurological implications. This review summarizes evidence indicating that inhaled nanoparticles, particularly metallic nanoparticles (MNPs), can penetrate respiratory defense mechanisms and the blood-brain barrier (BBB). This enables direct translocation into the central nervous system (CNS). Once they enter the brain, these nanoparticles act as structural scaffolds, thereby accelerating the misfolding and aggregation of amyloid-beta (Aβ) peptides. This phenomenon has been identified as a pathological hallmark of Alzheimer's disease (AD). The underlying mechanisms involve chronic neuroinflammation and excessive reactive oxygen species (ROS) generation, leading to neuronal degeneration and cognitive decline. Significantly, the physicochemical properties that make nanoparticles potentially hazardous also present therapeutic possibilities. A notable example is the use of gold nanoparticles (AuNPs) to inhibit amyloid beta aggregation, which highlights the potential of nanomedicine for targeted neuroprotection.

Keywords: air pollution, particulate matter ($PM_{2.5}$), nanoparticles (NPs), amyloid beta (Aβ) protein, neurotoxicity, Alzheimer's disease (AD), reactive oxygen species (ROS)

1. Introduction

The population growth and development of industry have created various types of pollution. Among these pollutions, air pollution has become one of the main global problems around the world. The consequences of polluted air led to adverse health effects in humans and other living organisms. In addition to that, the polluted air is damaging the ecosystem and contributing to climate change. The substances that cause this air pollution are toxic chemicals such as sulfur dioxide (SO_2) or nitrogen oxides (NO and NO_2) emitted from vehicles, and particulate matter (PM) in the atmosphere [1].

Air pollution comes from both natural sources, such as volcanic eruptions, wildfires, and desert dust, and human-made sources such as biomass burning, industry smog, vehicle exhaust, power generation, and construction activity, which are a great percentage of air pollution. According to this, human activity is often associated with polluted air in many urban areas. The main difference between the two of them is that natural sources cannot be controlled, in contrast to human activities. Depending on the harmful substances, air pollution can occur inside the building, called indoor air pollution, and outside the building, called outdoor air pollution. The majority of indoor air pollution is caused by human activities. Nevertheless, both natural sources and human-made sources are outdoor pollutants [2]. The statistics published by the World Health Organization (WHO) show that 9 out of 10 people in the world are exposed to polluted air. It results in around 7 million deaths each year because of complications arising from indoor and outdoor air pollution [3].

The historical record contains numerous documented incidents of air pollution, including the Donora Smog in the United States in 1948, which led to respiratory disorders and 20 fatalities [4], and the Great Smog of London in the United Kingdom in 1952. The combination of industrial pollution and domestic coal burning (Figure 1) led to an estimated 12,000 deaths, prompting the UK government to introduce the first "Clean Air Act" (CAA) in 1956 [5]. The main goal of the act was to set new standards, take measures to decrease air pollution, and protect the environment. Therefore, the CAA implemented a decision to make the use of smokeless fuels mandatory.

Figure 1. Great Smog of London [5].

In the present age, advanced monitoring programs such as the National Air Quality Monitoring Program (NAMP) and the Air Quality Index (AQI), frequently enhanced by artificial intelligence (AI) and machine learning (ML), are employed to track and manage air quality in real time [6].

2. Particulate Matter (PM) Fractions and Their Biological Penetration

Particulate matter is a mixture of microscopic particles in the air that are composed of solids and liquids and can harm human health. It is responsible for human morbidity and mortality. Particle pollution of air can be found both indoors (induced during home activities, renovation works, and indoor workshop activities) and outdoors (traffic, industry) (Figure 2) [7]. PM accumulation affects soil and water quality, which can affect plant and animal life. It is classified based on particle size, PM_{10} (coarse particles, ≤ 10 μm) and $PM_{2.5}$ (fine particles, ≤ 2.5 μm) [8].

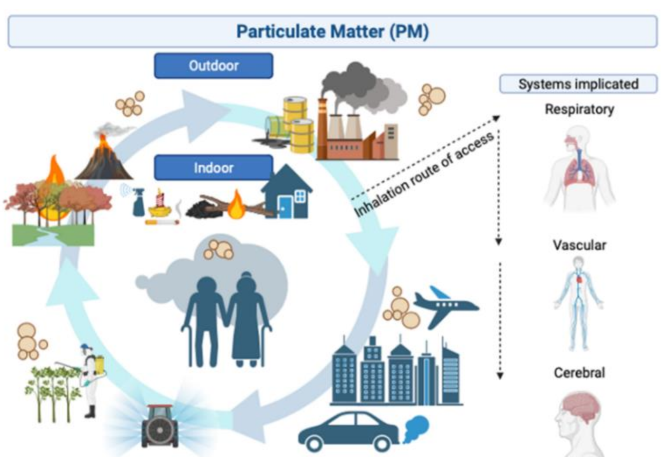

Figure 2. Graphical representation of PM in indoor and outdoor environments [7].

2.1. Biological Penetration of PM_{10}

PM_{10} is particulate matter that is 10 µm or less in diameter. However, it may still be visible to the naked eye under some circumstances as dust. Compared to $PM_{2.5}$, PM_{10} is heavier and falls to the ground more quickly, and it can cause discomfort in the eyes and nose. Over and above, it does not penetrate the lungs as deeply as $PM_{2.5}$. Respiratory and cardiovascular diseases are associated with PM_{10} exposure. The main causes of PM_{10} are transportation and industrial activities. Forests and green areas are significant as cleaning factors to prevent PM_{10} [9].

2.2. Biological Penetration of $PM_{2.5}$

$PM_{2.5}$ is particulate matter that is 2.5 µm or below in size and consists of ultra-fine particles and nanoparticles. They are not visible and come from a variety of sources, such as vehicle emissions, manufacturing facilities, power generation, and construction activities.

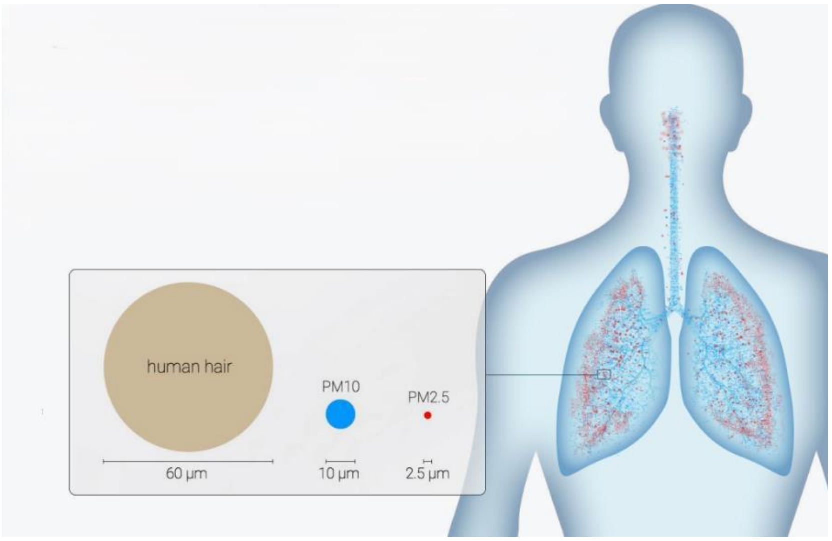

Figure 3. Graphical representation of PM_{10} and $PM_{2.5}$ [10].

Due to their microscopic size, they can be hard to prevent, may easily penetrate the body, affect its circulation and the breathing systems, and result in damage to the lungs (Figure 3) [10], heart, and brain. Table 1 illustrates disorders associated with $PM_{2.5}$ exposure [11].

Table 1. List of diseases caused by $PM_{2.5}$.

Health Effect	Associated Disorders
Respiratory diseases	Sore throat, asthma, bronchitis, tuberculosis, Chronic Obstructive Pulmonary Disease (COPD), pneumonia, lung cancer, head and neck cancer [12]
Cardiovascular diseases	Heart attacks, strokes, arrhythmias, ischemic heart disease, hypertension, atherosclerosis [13]
Neurodegenerative diseases	Dementia, Alzheimer's disease, Parkinson's disease [14]
Other health effects	Tiredness, headache, reduced intellectual ability, and premature death [15]

3. Global Monitoring of $PM_{2.5}$

The global challenge of controlling $PM_{2.5}$ underscores the necessity for international collaboration, where countries and international organizations must prioritize the mitigation of industrial emissions, the promotion of clean energy sources, and the promotion of sustainable transportation.

3.1. Regional Pollution in Asia

The presence of $PM_{2.5}$ pollution in Asia indicates a significant environmental crisis. According to the AQI 2022 ranking [16] and the 2019 World Air Quality Report [10], a considerable number of the world's most air-polluted countries are located in Asia (Figure 4).

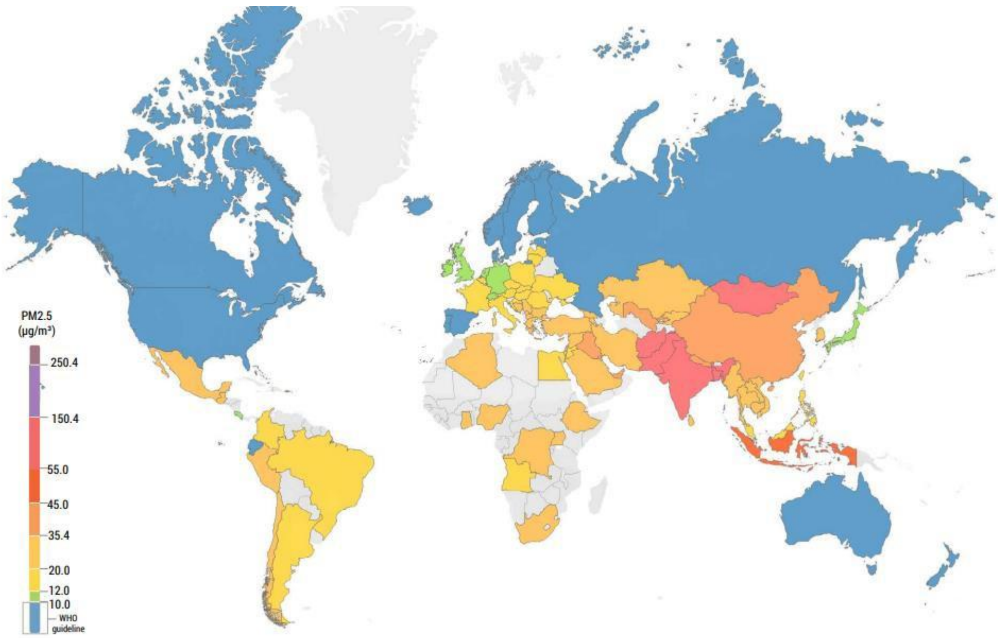

Figure 4. Global map of countries exposed by $PM_{2.5}$ in 2019 [10].

Table 2 presents the countries and their cities affected by $PM_{2.5}$ and the reasons that played the biggest role in its creation.

Table 2. List of Asian countries, the most polluted cities, and the biggest sources.

Countries	The most polluted cities	The biggest sources
Bangladesh	Dhaka	Brickmaking industry, motor vehicles, wood burning, soil, and road dust [17]
Pakistan	Punjab	Transportation, industry emissions, and power generation [18]
India	Delhi	Industryand manufacturing, vehicle emissions [19]
China	Beijing	Industrial and energy structure, vehicle exhaust, and coal combustion [20]
Thailand	Chiang Rai	Manufacturing, power plants, vehicles, and agricultural burning [21]

According to the 2018 publication by WHO, there were 543000 fatalities in children under the age of 5 and 52000 deaths in children aged 5 to 15 because of polluted air in 2016. The fact that the number of deaths in children is higher than in adults [22]. The main reason is that the absorption of contaminants is due to engaging in air activities for a long time.

3.2. Regional Pollution in Europe

Contrary to the higher air quality standards generally maintained by European countries, significant regional challenges remain, particularly in Eastern Europe. In 2015, researchers documented 449,813 premature deaths in Europe [23] that were associated with $PM_{2.5}$ from anthropogenic sources, with agricultural emissions, vehicles, and home heating recognized as primary contributors.

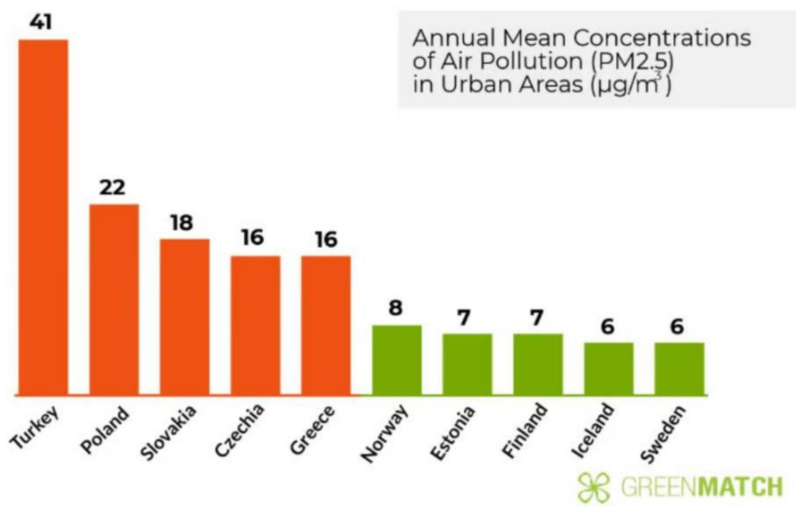

Figure 5. European countries' $PM_{2.5}$ concentrations by GreenMatch [24].

According to the "Green Match", European countries such as Poland, Slovakia, Czechia, Turkey (3% European portion), and Greece are significantly affected by air pollution $PM_{2.5}$. Figure 5 represents the annual mean concentrations of the $PM_{2.5}$ fraction in air pollution in urban areas of European countries. Turkey leads the list, followed by Poland. The main reasons are industry, transport, carbon dioxide production from other sources, and a lack of green areas. Air pollution is very low and controlled in countries like Norway, Sweden, and Finland compared to Turkey [24].

4. Nanoparticles as a Key Component of $PM_{2.5}$

The concept of "Particulate Matter" (PM), particularly the subcategory $PM_{2.5}$, serves as a comprehensive classification system for heterogeneous atmospheric pollutants. Nevertheless, the most biologically reactive and hazardous component of this mixture is the ultrafine fraction, also known as nanoparticles (NPs). According to the accepted definition, nanoparticles range from 1 to 100 nanometers (nm) and represent a high-hazard component that is unequally disproportionate to their mass concentration. These substances are derived from both naturally occurring sources and anthropogenic emissions, particularly those resulting from high-temperature combustion processes (vehicular exhaust and industrial activities) that generate metallic, metal oxide, and organic compounds [25].

The primary risk posed by nanoparticles derives from their particular physicochemical characteristics. Their large surface area relative to volume enhances chemical reactivity, promoting the generation of reactive oxygen species (ROS) and other damaging cellular molecules. Due to their ultrafine size, nanoparticles have the capacity to penetrate deeply into the respiratory tract and efficiently cross the alveolar–capillary barrier [26], a phenomenon that larger particulate matter fractions are generally unable to do. Once translocated into the blood-brain barrier (BBB), they can diffuse systemically, carrying toxic metals, organic compounds, and other adsorbent pollutants to distant organs. This systemic process converts localized air pollution exposure into a threat that affects the entire body, with particular exposure to the central nervous system (CNS). In this system, nanomaterials have been observed to induce neuroinflammation and oxidative stress [27].

5. The Potential Influence of Nanoparticles on Amyloid Beta Protein

The impact of nanoparticles (NPs) on proteins is also necessary, depending on the shape, size, and surface charge of nanoparticles. Small nanoparticles have a large surface area-to-volume ratio. It means that they can penetrate biological barriers and interact with proteins more easily. Additionally, small nanoparticles are so active based on their high surface area, and they can cause oxidative stress and inflammation, leading to greater neuronal damage and increased neurotoxicity. Large nanoparticles have a lower surface area-to-volume ratio. It means that they cannot penetrate biological barriers so easily. They can have limited protein-surface interactions. It means less oxidative stress and reduced neurotoxicity [28].

Nanoparticles in $PM_{2.5}$ have a profound effect on many proteins, including amyloid beta (Aβ) and human cystatin C. These particles can also cause oxidative stress by producing reactive oxygen species (ROS). The formation of reactive oxygen species causes cellular, protein, and DNA damage, which creates mutations. In the end, these proteins change their structure and denature or lose their functionality. Many diseases, such as cancer, cardiovascular, respiratory, and neurodegenerative disorders, occur as a consequence of the deterioration of these proteins. Studies in mice have concluded that zinc, iron, and copper are directly related to the development of amyloid plaques in neurodegenerative disorders and cause increased oxidative stress and neuronal dysfunction [29]. Moreover, other studies have provided strong evidence that copper, zinc, and iron contribute to the development of Alzheimer's disease in terms of the aggregation of amyloid beta proteins and the generation of reactive oxygen species (Figure 6) [30, 31].

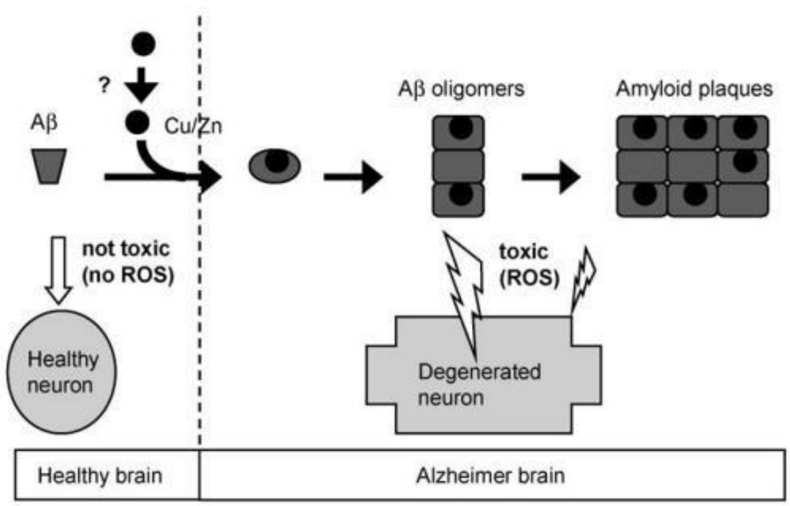

Figure 6. An illustration of neural defects [31].

6. Amyloid Beta Protein as a Key Component of Alzheimer's Disease

Amyloid beta (Aβ) peptide is derived from the amyloid precursor protein (APP), which is a large protein in the central nervous system (CNS) and is linked to Alzheimer's disease (AD). Amyloid beta exists in a random coil shape or α-helical and typically consists of approximately 35-43 amino acids [32]. As a result of the environmental impact (air pollution), genetic mutations (age, neuronal death), and lifestyle factors (lack of exercise and poor diet), protein abnormalities appear. In this case, amyloid beta can misfold into an aberrant shape spontaneously. The aggregation of these abnormal proteins leads to the formation of small oligomers, which harmfully influence the cells and speed up the development of disorders. Finally, the aggregation of these oligomers creates long fibrils (Figure 7) [33] that are distinguished by their cross-beta sheet structure and are extremely stable. Briefly, the amyloid beta peptide is the key point of oligomerization and fibrillogenesis that contributes to the development of Alzheimer's disease [34].

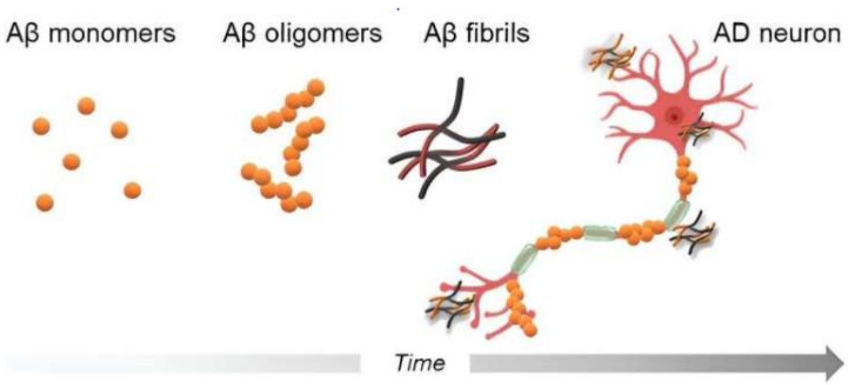

Figure 7. Alzheimer's Disease Mechanism [33].

Alzheimer's disease is a neurodegenerative disorder associated with the amyloidogenesis process. This process (according to the amyloid hypothesis) is linked to the degradation and death of brain cells, leading to Alzheimer's disease. Firstly, it begins with dementia, which means poor memory and declined cognition. Based on research in London, it is suggested that living near the main roads in the city center increases the risk of dementia [35]. Air pollution caused by traffic, which contains particulate matter ($PM_{2.5}$), has a strong effect on cognitive decline [36], particularly in children.

Plaques and tangles are the abnormal protein deposits in the brain that provoke Alzheimer's disease. These deposits are composed of two main proteins known as amyloid beta, which is found around the neurons, and tau, which is found inside the neurons. Normally, amyloid beta and tau are present in healthy brains; however, their functions are deviant in Alzheimer's disease. Neurofibrillary tangles are composed of tau protein, and amyloid plaques are composed of amyloid beta protein, which is part of the amyloid precursor protein. Tangles and plaques destroy many nerve cells, causing cell death and, consequently, the brain begins to shrink. The key symptoms of Alzheimer's disease are memory loss, cognitive disabilities, and behavior problems [37].

7. Dual Roles of Nanoparticles

Despite their hazardous association with particulate matter (PM) and protein aggregation, some nanoparticles are being studied for therapeutic applications. Their distinctive characteristics, including precise size control and surface chemistry, qualify them as optimal candidates for drug targeting and delivery. For instance, studies have examined the potential of certain gold nanoparticles to regulate the aggregation of amyloid beta peptides, providing a promising perspective for future Alzheimer's disease therapeutic approaches. Similarly, carbon-based nanomaterials, including graphene quantum dots and polymeric nanoparticles, have exhibited inhibitory effects on amyloid beta (Aβ) fibrillation and enhanced blood-brain barrier transport in earlier research studies. [38, 39].

8. Conclusion and Future Directions

The findings of this study provide substantial evidence that ultrafine particulate matter and the metallic nanoparticles it harbors are not merely respiratory irritants but rather systemic toxicants capable of inducing significant neurological and cellular damage. The precise mechanism by which these nanoparticles stimulate the misfolding and aggregation of amyloid beta is a critical area of research. It is crucial to comprehend the dual nature of nanoparticles, which serve both as environmental contaminants and as prospective therapeutic agents. This understanding is fundamental to the development of effective public health policies aimed at reducing exposure, as well as the creation of innovative nanomedicines for the treatment of neurodegenerative disorders.

Author Contributions

The authors confirm responsibility for the conception, drafting, critical revision, and final approval of the manuscript.

Conflict of Interest

The authors declare no conflicts of interest.

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Data Availability Statement

The authors declare that the data supporting the findings of this study are available within the article.

Abbreviations

Sulfur Dioxide (SO_2), Nitrogen Oxides (NO and NO_2), World Health Organization (WHO), Clean Air Act (CAA), National Air Quality Monitoring Program (NAMP), Air Quality Index (AQI), Artificial Intelligence (AI), Machine Learning (ML), Particulate Matter (PM), Nanometers (nm), Chronic Obstructive Pulmonary Disease (COPD), Nanoparticles (NPs), Metallic Nanoparticles (MNPs), Central Nervous System (CNS), Reactive Oxygen Species (ROS), Blood-Brain Barrier (BBB), Amyloid Beta (Aβ), Amyloid Precursor Protein (APP), Alzheimer's Disease (AD).

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