

Particulate Matter and Associated Metals

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Particulate air pollution (PM) is a mixture of heterogenous components from natural and anthropogenic sources and contributes to a variety of serious illnesses, including neurological and behavioral effects, as well as millions of premature deaths. Ultrafine (PM_{0.1}) and fine-size ambient particles (PM_{2.5}) can enter the circulatory system and cross the blood–brain barrier or enter through the optic nerve, and then upregulate inflammatory markers and increase reactive oxygen species (ROS) in the brain. Toxic and neurotoxic metals such as manganese (Mn), zinc (Zn), lead (Pb), copper (Cu), nickel (Ni), and barium (Ba) can adsorb to the PM surface and potentially contribute to the neurotoxic effects associated with PM exposure. Epidemiological studies have shown a negative relationship between exposure to PM-associated Mn and neurodevelopment amongst children, as well as impaired dexterity in the elderly. Inhaled PM-associated Cu has also been shown to impair motor performance and alter basal ganglia in schoolchildren.

particulate matter

air pollution

particulate air pollution

metal inhalation

ambient metals

particulate matter associated metals

neurotoxicity

mental health

1. Introduction

Air pollution continues to pose a global environmental health risk that affects both the development and worsening of many health issues including cardiovascular disease, pulmonary illnesses, cancer, and central nervous system disorders ^[1]. According to the 2017 Global Burden of Disease Study, 4.9 million deaths and 1.4 billion disability-adjusted life years (DALYs) in 2017 were attributed to air pollution ^{[1][2]}. Previous mechanistic studies have reported that inhalation of air pollutants can provoke neuro-inflammation, oxidative stress, and dopaminergic neurotoxicity ^[2]. Particulate air pollution, composed mainly of organic and elemental carbon, metals, polycyclic aromatic hydrocarbons, inorganic compounds, nitrates, sulfates, and other organic materials (like polychlorinated biphenyls from industrial manufacturing), is a major component of air pollution and is of great research interest due to its well-documented associations with serious short- and long-term adverse health effects ^{[1][3][4][5][6][7]}. Particulate matter (PM) can be denoted by size and ranges from 10 µm to less than 0.1 µm in size. Fine PM particles (PM_{2.5}) are between 2.5 and 0.1 µm in diameter, while ultrafine particles (UFP) are less than 0.1 µm in diameter (PM_{0.1}) ^{[6][7][8]} ^[9]. Major sources of PM include combustion of fossil fuels, traffic, and industrial and agricultural processes ^{[1][4]}.

2. PM Distribution to the Brain

Particulate matter can enter the body through multiple routes of exposure including inhalation and ingestion [10]; the smaller the particle, the longer it can remain in the lungs [11]. Ultrafine particles in the lung act like gas molecules and can reach the circulatory system and potentially cross the blood–brain barrier [8][9][11][12][13]. Previous research studies also suggest that inhaled PM_{2.5} reaches the brain [11]. Fine and ultrafine PM can reach the brain through multiple pathways once it enters into the circulatory system [1][12]. Ultrafine particles (PM_{0.1}) can pass through the blood–brain barrier, reach the vagal nerves, and enter the brain [1]. Alternatively, PM can bypass the blood–brain barrier by entering the brain via the olfactory bulb and fifth cranial nerve from direct inhalation through the nose [1][12]. The fact that fine and ultrafine PM can be transported directly through the olfactory pathway to the brain has been previously confirmed by the presence of particles in olfactory neurons and intracellular erythrocytes in the frontal lobe of the brain [12]. The presence of PM_{2.5} in the brain has been shown to cause cell cycle arrest, apoptosis of neurons, neuro-inflammation, induce oxidative stress and genetic damage leading to neurodegenerative changes and dopaminergic neurotoxicity, as well as structural damage to the myelin sheath [2][6][12][14][15][16].

3. PM Constituents

Particulate matter is a mixture of chemical compounds that can include inorganic and organic materials, as well as nitrates and sulfates [1][3][4][6]. The surface of ambient PM, particularly PM_{2.5}, can carry viruses, bacteria, volatile organic compounds (VOCs), and heavy metals by adsorbing to the surface of the particle [6][7][10][13]. A number of studies have demonstrated high levels of adsorbed toxic heavy metals coming from both natural and anthropogenic sources that can adversely affect human health [3][10][11]. The types and concentrations of metals adsorbed to PM varies depending on the source and environment, but can range in concentration from 30 to 35 µg/m³ [17].

PM-associated heavy metals, including manganese (Mn), zinc (Zn), iron (Fe), cadmium (Cd), copper (Cu), arsenic (As), barium (Ba), lead (Pb), aluminum (Al), and nickel (Ni), are often significant metal components of PM_{2.5} [10][18][19]; previous studies have reported Mn, Ba, Ni, Fe, and Cu triggering inflammatory responses after exposure [4][18]. Maternal exposure during pregnancy to Mn, Ni, Pb, or Fe has previously been associated with increased risk of childhood autism, and exposure of pregnant mice to Mn has been linked with an acute inflammatory response resulting in neurotoxicity during fetal neurodevelopment [4][6][16][20]. Developmental Pb exposure has also been reported to be linked with schizophrenia due to antagonism of N-methyl-D-aspartate (NMDA) subtype of glutamate receptors (NMDAR); antagonism of NMDAR receptors can lead to decreased function, which could also play a role in the pathophysiology of schizophrenia [4]. Recent studies reported that the presence of PM and associated metals, Fe and Al, in the corpus callosum is associated with structural myelin sheath damage, which indicates that exposure to PM and associated heavy metals can disturb myelinogenesis [6][13][15][16].

4. PM Sources

Traffic emissions are a major source of heavy metal pollution in the ambient environment due to increased vehicle use and traffic congestion [9][19]. For example, automobile emissions release a number of heavy metals into the ambient environment including Cu, Zn, Cd, As, mercury (Hg), Mn, cobalt (Co), and Fe [10][21]. In addition, both Cu and Zn can be released into the air from tire abrasion, lubricants, and corrosion of vehicular parts, while Cd contamination arises from aging automobile tires, gasoline use, and car body and brake lining wear [19][21]. Industrial activity also contributes to particle-bound heavy metal air pollution, along with power plants, mining, metal smelting, and chemical plants [10][19]. Other sources of PM-associated heavy metal pollution originate from construction activity through building demolition and renovation, the spraying of pesticides and fungicides, as well as residential and commercial heating [10][19].

5. Air Pollution and Mental Health

Recently, studies have emerged that demonstrate the adverse effects of air pollution, either gases, particles, or a combination of both, on mental health [2]. The risk of psychosis has been linked to both genetic and environmental factors with increasing evidence that the environment can play a large part in influencing genetic effects through gene-environment interactions and epigenetic mechanisms [1]. Epidemiological studies reported that both short- and long-term exposure to PM_{2.5} were associated with greater odds of depression; the same relationship was not observed with inhalation of larger size particulate matter (PM₁₀) [2]. There is also a reported relationship between urbanicity of birthplace/upbringing and a higher incidence of schizophrenia and other non-affective psychoses [1]. One of the speculated risk factors that could partially explain this association is exposure to particulate/gaseous air pollutants, which represents an underlying urbanicity risk factor [1]. Inhalation exposure to PM_{2.5} can also influence central nervous system development, resulting in an increased risk of later life depression, as well as influence cognitive disorders and create abnormalities in the architecture of brain white matter during childhood [7][12][20].

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