Etiology and Route of Access of Particulate Matter

Subjects: Toxicology

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The primary origin of particulate matter (PM) is from a combination of industrial activity, internal combustion engines, and geographic and meteorological conditions linked to the increased forest fires.

particulate matter air quality elderly cognitive function neurodegeneration

1. Introduction

The primary origin of particulate matter (PM) is from a combination of industrial activity, internal combustion engines, and geographic and meteorological conditions linked to the increased forest fires [1]. Specifically, smoke originating from forest fires is primarily responsible for ultrafine particles (UFPM ≤ 100 nm in diameter) [1]. Inspiring such particles increases the risk of developing respiratory diseases, such as tonsillitis, asthma, bronchitis, lung cancer, and cystic fibrosis pneumonia later in life [2].

2. Etiology of Particulate Matter

Air quality is determined by estimating the number of solid matter particles in the air [3]. These particles contribute to the air pollution level and are referred to as PM [4]. PM are partly formed in the atmosphere through chemical reactions that produce inorganic nitrates and sulfates, as well as organic compounds [3]. Inhalable particles are divided into four groups by diameter highlighting three of them: larger than 10 μm (μm), smaller than 10 μm (PM_{10}), and smaller than 2.5 μm ($PM_{2.5}$) [5]. In addition, a fourth group exists, referred to as ultrafine PM (UFPM or $PM_{0.1}$, with diameter < 100 nm) [6]. $PM_{2.5}$ are not visible by the naked eye, and levels of such particles (measured in $\mu g/m^3$) are not distinguished without specialized equipment when kept at concentrations below 35 $\mu g/m^3$ [7]. The US EPA, the European Environment Agency (EEA), and the WHO set a $PM_{2.5}$ safety standard under 12 $\mu g/m^3$ (annual average) and a 24 h average of 35 $\mu g/m^3$. Nonetheless, this value is routinely exceeded worldwide (e.g., in large cities in India, China, or Mexico) [6]. Therefore, many countries have failed to keep urban PM pollution levels within WHO guidelines.

PM is mainly derived from road transport, agriculture, power plants, industry, and homes. Outdoor particles are primarily produced from traffic-related activities [6]. $PM_{2.5}$ is a mixture of harmful chemicals, such as sulfur dioxide, nitrogen dioxide, organic phosphates, and chlorine, which are utilized in agriculture, bisphenol, etc., along with combustion particles, carbon particles, heavy metals, and pollutants from industrial sites and factories. In contrast,

PM_{10} consists mainly of pollen, mold, and dust particles [8][9]. Hence, despite being less visible to the public, <$PM_{2.5}$ poses much greater health risks than larger particles, and due to their size, with limited efficacy of protective gear, such as masks or respirators, in preventing exposure [3]. UFPM are mostly derived from wildfire smoke [1] (see **Table 1**). Indoor environmental particles are derived from combustion activities such as cooking, as well as heating with coal, wood or dung, candles, incense, kerosene lamps, and tobacco smoking. In addition, indoor pollution originates from non-combustion sources and volatile organic compounds such as cleaning and insecticide products, electric devices, and printers [3].

It is noteworthy that PM pollution also has important economic ramifications since it increases medical expenses, reduces worker productivity, and damages soil, crops, forests, lakes, and rivers [3].

3. Route of Access and Biological Systems of Particulate Matter

PM can reach and accumulate throughout tissues and organs in humans, such as the gastrointestinal tract, skin, mucosae, placenta, and brain [1].

The cardiovascular system is affected by PM exposure [6][10]. The probability of heart attack due to coronary ischemia, coronary revascularization, ischemic heart disease, thrombosis, or stroke is also positively correlated with air pollution [11][12], especially among the older population [13][14][15], with evidence indicating a possible biological sex bias, where women are affected to a greater extent than men [14][15]. Notably, non-industrial fine airborne particles (e.g., dust particles from dry desert areas) contribute to the probability of developing cardiovascular diseases as much as anthropogenic $PM_{2.5}$, e.g., emitted from industrial (e.g., metals) and agricultural (organic chemicals) sites [16]. While the impact of air pollution on respiratory health can manifest even after brief exposure to elevated levels of $PM_{2.5}$, the effects on the cardiovascular system are best observed through longitudinal studies comparing populations exposed to long-term high levels of $PM_{2.5}$ to those living in areas with $PM_{2.5}$ levels within the safety limit of 10–12 $\mu g/m^3$ [17].

The brain is especially vulnerable to air pollution. Inhalation is the main route of PM exposure [6][18][19]. Costa and collaborators characterized PM absorption into the brain [6]. PM enters the central nervous system (CNS) through the nose but follows different paths depending on the size and solubility. While PM_{10} is deposited in the upper airways, $PM_{2.5}$ is deposited in the lungs and is also subject to olfactory transport and deposition in the olfactory cortex and other brain regions [6][19][20]. Furthermore, UFPM can be deposited in the cerebral cortex and cerebellum secondary to transport via the olfactory nerves [18]. Once reaching the lung, PM may also travel through the blood into the brain [6][20] (See **Table 1**). Understanding these pathways and how PM is capable of reaching the CNS underscores the urgent need for an in-depth analysis of its impact on cognitive function. In the brain, PM accumulation contributes to CNS diseases [2][21][22]. The impact on older people is such that there is even evidence of a greater accumulation of amyloid beta (a characteristic of Alzheimer's disease, AD) in individuals exposed to higher levels of air pollution [21].

Overall, the decrease in $PM_{2.5}$ concentration correlates with the reduction of hospitalization and early death rates [23], decreasing the burden on economic sectors and the health care system. Altogether, PM exposure affects the respiratory, cardiovascular, and autoimmune systems, potentially accelerating its adverse effects on cognitive functions (**Figure 1**).

Table 1. Summaries of etiology of particulate matter and pathways implicated until reaching the CNS in the brain.

PM by Diameter	[1] Main Etiology	Deposited in:	References
$PM_{0.1}$ or UFPM ≤100 nm	Smoke from wildfire	Cerebral cortex and cerebellum secondary to transport via the olfactory nerves	[1][18]
$PM_{2.5}$ ≤2.5 µm	Traffic-related activities, industrial sites, factories, and agriculture	[2] Lungs and is also subject to olfactory transport and deposition in the olfactory cortex and other brain regions	[6][8][9][20]
PM_{10} ≤10 µm	Pollen, mold, and dust particles	Filtered out by the nose and upper airways	[6][8][9][19][20]

UFPM: ultrafine particles; CNS: central nervous system. Notes: [1] Outdoor environmental particles are primarily produced [1][6] onmental particles or dung, candles, mpounds such as c once the lungs are [2] 2 [6][20]

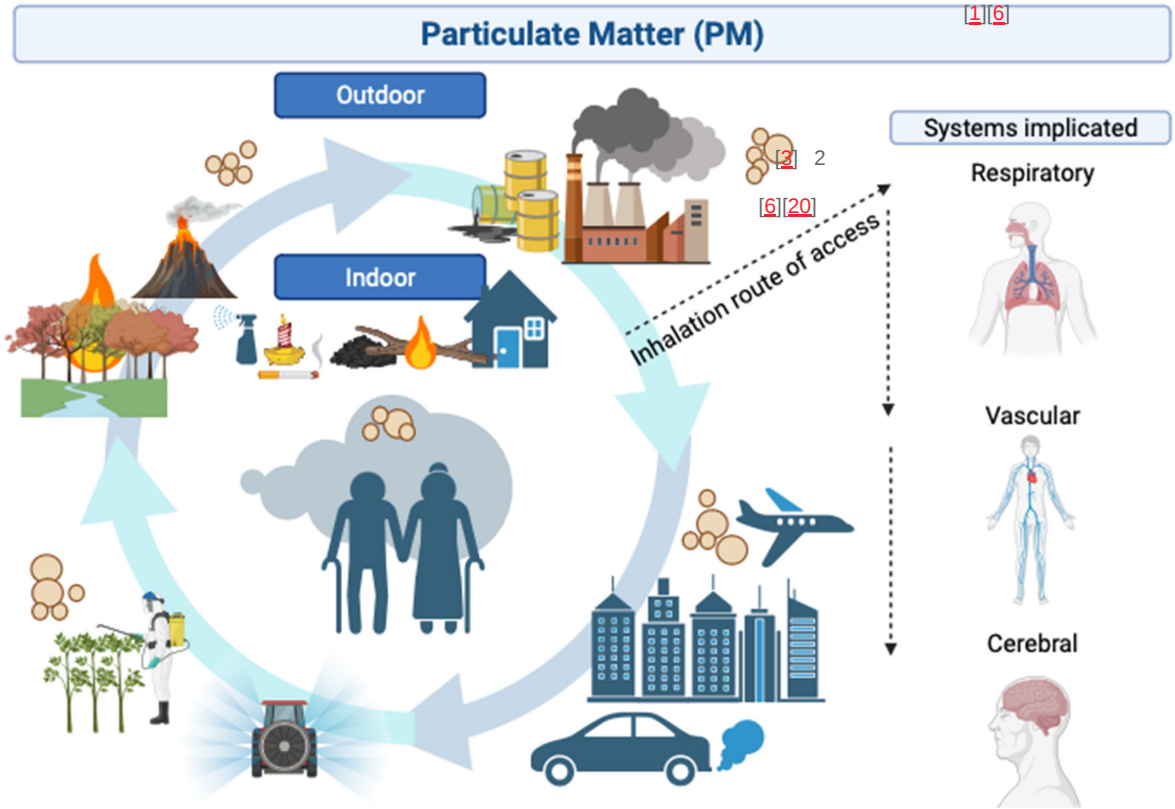

Figure 1. Overview of indoor and outdoor air pollution sources and their consequences to the human body. The main origin of outdoor PM is from a combination of industrial activity, internal combustion engines, traffic-related and geographic and meteorological conditions linked to increased forest fires and practices derived from

agricultural activities. On the other hand, indoor environment particles originate from combustion, encompassing activities such as cooking and heating with coal, wood or dung, candles, incense, kerosene lamps, tobacco smoking, and non-combustion sources, such as cleaning and insecticide products, electric devices, and printers. PM enters the body via inhalation through the nose, increasing the risk of respiratory, cardiovascular, and cerebrovascular disease, therefore affecting the CNS. Figure created with BioRender.com (accessed on 10 October 2023).

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