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DND Dementia and Neurocognitive Disorder

Editorial

Air Pollution and Dementia

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Air pollution is a current and growing global problem. It also has a major adverse effect on our health. Air pollution exposure, a powerful health risk factor, is more likely to cause disease than the commonly known modifiable disease risk factor, and impacts health life reduction and labor productivity degradation in addition to disease outbreaks and early deaths.[1,2] The association between air pollution and cardiovascular or respiratory problems has been well documented. Conversely, there has been an insufficient number of studies on the relationship between air pollution and cognitive functions.[3,4] In Korea, since the late 1990s, the impact of air pollution on health has been recognized, and epidemiological studies on public health have been actively conducted. However, the relationship between cognitive function and air pollution is insufficient. In this paper, we reviewed the relationship between particulate matter (PM), which inflicts the greatest harm to humans among air pollutants, and cognitive functions and dementia and introduced the plans of the Korean Dementia Association (KDA) for this study on the relationship between PM and dementia.

Air pollution is contamination of the atmosphere by any chemical, physical or biological agent that is artificially discharged. Home heating and equipment use, motor vehicles, industrial facilities, and forest fires are common sources of air pollution. Pollutants of major public health concern include PM, carbon monoxide, ozone, nitrogen dioxide, and sulfur dioxide. Among them, much attention is focused on the adverse effects of PM on health. PM is not only one material, but a complex mixture of fine solids and liquid particles of organic and inorganic components dispersed in the atmosphere. PM can be classified into primary PM and secondary PM according to the production process. The primary form refers to the direct discharge of soil dust and pollen from nature, generated by burning fossil fuels, automobile exhaust pipes, and the secondary form is produced by chemical reaction of pollutants already discharged in the atmosphere.[3] Smaller particles are more harmful to our health. PM can be further classified into the particle diameter is smaller than 10 μm (PM_{10}) and the particle diameter is smaller than 2.5 μm ($PM_{2.5}$). If the concentration and the content of PM are the same, the smaller the particle, the more harmful the $PM_{2.5}$ is to the PM_{10}, because the surface is wider than the PM_{10}. And the smaller the particle, the easier it is also to move from the bronchus to the other human organs. PM directly impacts the skin and eyes, the primary shields of the human body, and the nasal or throat mucosa, causing physical tangent and local inflammatory responses. Additionally, PM is small, so it can penetrate the respiratory tract, which can trigger various health problems. The mechanism of tissue and cytotoxicity by PM is most representative of the increase of oxidative stress, and inflammation. The endotoxin impact and DNA damage are known causes of PM.[5,6]

Inhaled PM travels directly to the brain tissue through the body circulation or the olfactory bulb, and intrudes the nasal epithelium in the nasal cavity causing inflammation.[7,8] Other pathways are not removed by mucocillary clearance among the inhaled PM, but contaminants achieving alveolar ejaculation cause inflammation and produce inflammatory cytokines. This inflammatory cytokine is transmitted to the brain tissue through systemic circulation, spreading neuroinflammation.[8] In the elderly with high risk of dementia, the overall physiological function gradually decreases as age increases. Thus, compared to young adults, the ability to expel PM, once inhaled, and to alleviate or eliminate toxicity, is diminished. Additionally, the elderly are often suffering from cardiovascular or respiratory diseases and have low resistance to the biological toxicity of PM because of their diminished immunity. Thus, the elderly are more vulnerable to the influence of PM.[9,10]

Experimental and animal studies observed that inhalation of PM enhanced reactive oxygen species and inflammatory responses in the brain, associated with precipitating amyloid beta protein (Aβ) peptides, disruption of the blood-brain barrier, and microglial activation.[11-13] Recently, a small number of epidemiologic studies have reported that exposure to air pollution was associated with cognitive decline, and pathological changes in the brain. People living in areas with high air pollution accumulate more Aβ and in neurons and astrocytes than people living in areas with low air pollution.[14] In a large-scale cohort study of elderly females, overall cognitive function, language memory, working memory, and attention were tracked. In the long-term PM exposure group, cognitive function diminished more rapidly than in the short-term PM exposure group. It was estimated that the impact of cognitive aging was approximately two years when PM concentration increased by 10 μg/m^3.[15] In another large-scale cohort study, followed up for 10 year with elderly people older than 65, the risk of developing Alzheimer's disease (AD) increased by 138% with increasing $PM_{2.5}$ concentration by 4.34 μg/m^3.[16] Also, living near major roadways was associated with higher incidence of dementia.[17] In the Framingham Offspring Study, $PM_{2.5}$ exposure degree, total brain volume, and white matter degeneration were examined in elderly people older than 60 without dementia and stroke. The brain volume diminished by 0.32% as the concentration of $PM_{2.5}$ increased by 2 μg/m^3.[18] In the Women's Health Initiative Memory Study (WHIMS), the white matter volume and total brain volume of the exposed persons also diminished relative to the high concentration of $PM_{2.5}$.[19] The ε4 allele, a variant of the apolipoprotein E (APOE) gene, is the most prominent genetic risk factor for sporadic non-familial AD. It has been postulated that APOE ε4allele carriers could have a higher risk of developing AD if they live in a polluted environment.[14] However, further study is needed to clarify the role of APOE ε4 alleles with air pollution exposure.

Dementia or cognitive impairment are difficult to study regarding the relationship with PM, because both occur because of long-term degenerative changes in the brain. It is difficult to distinguish the impact of various risk factors and PM that cause dementia, and it is difficult as well to accurately measure the degree of cognitive function impairment and PM exposure. Especially in Korea, the social interest in air pollution and PM has not increased for a long time, so there is a lack of research on PM and dementia. However, in Korea, it is a relatively ideal situation in which to conduct relevant research compared to other countries in that abundant big data can be retrieved from the National Health Insurance Service (NHIS) enabling access to medical and residence data on dementia patients and infrastructures such as dementia safety centers in each region. The KDA would actively conduct research on the relationship between PM and dementia reflecting the increasing social interest and we are actively participating in PM research relevant to the Korean Academy of Medical Sciences

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and Global Alliance for Clean Air. Relative to the big data research with NHIS, we will discuss the fine dust threat as a crucial topic. Based on these results, we will contribute to the preparation of the national PM policy in the health field. I would like to invite the interest and participation of many members in the efforts of the KDA.

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