The link between air pollution and Alzheimer’s disease

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Alzheimer’s disease (AD), as the most common age-related neurodegenerative disease, affects 2% of general elderly populations. Amyloid plaques and neurofibrillary tangles are two main hallmarks of AD that are usually associated with cerebral amyloid angiopathy. Aβ aggregates inside and outside of neurons and intra-neuronal hyper-phosphorylated tau induce dendritic spine collapse and synaptic degeneration, which finally leads to memory loss in AD patients (1). More recently, the potential deleterious effects of air pollution (AP) inhalation on the central nervous system was also investigated and mounting evidence supports a link between AP exposure and neurodegenerative pathologies, especially AD (2). Mice exposed to severe AP exhibited chronic inflammation and acceleration of Alzheimer’s-like pathology, suggesting that the brain is adversely affected by pollutants (3). Exposure to AP can lead to chronic oxidative stress (OS), which is involved in the pathogenesis of AD (4). To analyze the effect of chronic ozone exposure on changes in the production and accumulation of the Aβ1-42 and Aβ1-40 amyloid fibrils in the mitochondria of hippocampal neurons of rats exposed to ozone, we examined the mitochondrial expression levels of Presenilins 1 and 2 and ADAM10 to detect changes related to the OS caused by low doses of ozone (0.5 ppm). The results revealed significant accumulations of Aβ1-42 amyloid fibrils in the mitochondrial fractions on days 60 and 90 of ozone exposure along with reduction in Aβ1-40 amyloid fibril accumulation, significant over-expression of Pres2 and significant reduction in ADAM10 expression. These results indicate that the time of exposure to ozone and the accumulation of Aβ1-42 amyloid fibrils in the mitochondria of the hippocampal cells of rats were correlated (5).

References:

1) Shiler Khaledi, Shamseddin Ahmadi. Amyloid Beta and Tau: from Physiology to Pathology in Alzheimer’s Disease. 26 Mar 2016