Brain inflammation and Alzheimer's-like pathology in individuals exposed to severe air pollution.


Abstract

Air pollution is a complex mixture of gases (e.g., ozone), particulate matter, and organic compounds present in outdoor and indoor air. Dogs exposed to severe air pollution exhibit chronic inflammation and acceleration of Alzheimer's-like pathology, suggesting that the brain is adversely affected by pollutants. We investigated whether residency in cities with high levels of air pollution is associated with human brain inflammation. Expression of cyclooxygenase-2 (COX2), an inflammatory mediator, and accumulation of the 42-amino acid form of beta-amyloid (Abeta42), a cause of neuronal dysfunction, were measured in autopsy brain tissues of cognitively and neurologically intact lifelong residents of cities having low (n:9) or high (n:10) levels of air pollution. Genomic DNA apurinic/apyrimidinic sites, nuclear factor-kappaB activation and apolipoprotein E genotype were also evaluated. Residents of cities with severe air pollution had significantly higher COX2 expression in frontal cortex and hippocampus and greater neuronal and astrocytic accumulation of Abeta42 compared to residents in low air pollution cities. Increased COX2 expression and Abeta42 accumulation were also observed in the olfactory bulb. These findings suggest that exposure to severe air pollution is associated with brain inflammation and Abeta42 accumulation, two causes of neuronal dysfunction that precede the appearance of neuritic plaques and neurofibrillary tangles, hallmarks of Alzheimer's disease.

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