Air pollution in Mexico City associated with development of Alzheimer disease

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A new study by researchers at the Universities of Montana, Valle de México, Boise State, Universidad Veracruzana, Instituto Nacional de Pediatría and Paul-Flechsig-Institute for Brain Research heightens together with German company Analytik Jena concerns over the evolving and relentless Alzheimer's pathology observed in young Metropolitan Mexico City (MMC) urbanites. These findings are published in the *Journal of Alzheimer's Disease*.

Mexico City children have lifetime exposures to concentrations of air pollutants above the current USA standards, including fine particulate matter (PM 2.5). Metropolitan Mexico City is an example of extreme urban growth and serious environmental pollution and millions of children are involuntarily exposed to harmful concentrations of PM 2.5 every day since conception.

This study focused on studying 507 CSF normal samples from children, teens and young adults average age 12.8±6.7 years from MMC and control cities with low levels of air pollutants, using a high affinity monoclonal non-phosphorylated tau antibody (Non-P-Tau) as a potential biomarker of AD and axonal damage. In 81 samples, researchers also measured total tau (T-Tau), tau phosphorylated at threonine 181 (P-Tau), amyloid-β 1-42, brain-derived neurotrophic factor (BDNF), insulin, leptin and inflammatory mediators. Authors documented by transmission electron microscopy (TEM) myelinated axonal size, and the pathology associated with combustion-derived nanoparticles-iron rich, highly oxidant CDNPs- in anterior cingulate cortex (ACC) white matter in 6

young residents (4 MMC, 2 controls). Non-P-Tau showed a strong increase with age significantly faster among MMC versus controls. Anterior cingulate cortex showed significant decrease in the average axonal size and CDNPs were associated with organelle pathology in MMC residents. Non-P-Tau exhibited significant increases with age, an important finding in a young population where axonal changes are present and AD hallmarks are evolving steadily in the first two decades of life. Non-P-Tau is potentially an early biomarker of axonal damage and AD axonal pathology in highly exposed young populations. Drs.Lilian Calderón-Garcidueñas and Lachmann commented air pollution is a serious public health issue and exposures to concentrations of air pollutants at or above the current standards have been linked to neuroinflammation and high risk of Alzheimer's disease. Jung et al., 2015 found a 138% risk of increase of AD per increase of 4.34 μg/m3 in PM 2.5 suggesting long-term exposure to PM 2.5, as well as ozone above the current US EPA standards are associated with increased risk of Alzheimer's disease. In the USA alone, 200 million people live in areas where pollutants such as ozone and fine particulate matter exceed the standards.

The international team of researchers stated efforts should be aimed to identify and mitigate environmental factors influencing the development of Alzheimer's disease and neuroprotection of children and young adults ought to be a public health priority to halt the development of Alzheimer in the first two decades of life.

More information: Lilian Calderón-Garcidueñas et al, Non-Phosphorylated Tau in Cerebrospinal Fluid is a Marker of Alzheimer's Disease Continuum in Young Urbanites Exposed to Air Pollution, *Journal of Alzheimer's Disease* (2018). DOI: 10.3233/JAD-180853

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