pay special attention to older adults in immigrant families, with a particular focus on promoting emotional closeness with adult children, friend networks, and children’s filial piety.

SESSION 2075 (SYMPOSIUM)

AIR POLLUTION IN BRAIN AGING AND DEMENTIA

Chair: C.E. Finch, University of Southern California, Los Angeles, California
Co-Chair: J. Chen, University of Southern California, Los Angeles, California
Discussant: G.M. Martin, University of Washington

Over 75% of older Americans are living in metropolitan areas, and this urban-dwelling aging population will continue to grow in the coming decades. Ambient air pollution, a ubiquitous exposure in urban environments, has emerged as a new environmental factor in brain aging and dementia. Over the last few years, accumulating epidemiologic and neurotoxicological data have shown the aging brain is vulnerable to neurotoxic effects of ambient air pollutants. For instance, elevated levels of fine particulate matter (PM$_{2.5}$; PM with aerodynamic diameters<2.5µm) are associated with several years of faster cognitive aging and loss of white matter volume. Rodent brain models with inhaled PM exposure suggest the neurodegenerative mechanisms may involve increased neuroinflammation and soluble amyloid, and attrition of glutamate receptors. Assembling four presentations with new findings, this symposium aims to better define the individual risk, heterogeneity, and pathobiological mechanisms linking ambient air pollutants with brain aging and dementia. Epidemiological studies show that both PM$_{2.5}$ and O$_3$ exposures may increase the risk for dementia in older women (Chen). Epidemiologic studies also show that PM$_{2.5}$ -associated adverse effects on aging brain may be strengthened in populations with APOE4 alleles (Chen) and in populations of low educational attainment (Ailshire). Rodent models with PM exposures from traffic emissions document the role of the lung-brain axis in microglial activation (Block) and illustrate ApoE4 interaction with exogenous factors (Cacciottolo). Together these findings show that environmental factors contribute to accelerated brain aging in synergy with the ApoE4 allele risk factor for Alzheimer disease.

NEUROTOXIC EFFECTS OF AMBIENT AIR POLLUTION ON BRAIN STRUCTURE AND DEMENTIA RISK IN OLDER WOMEN

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Ambient air pollution is a novel determinant of cognitive aging. Using a Bayesian Maximum Entropy method to estimate outdoor concentrations of PM$_{2.5}$ (particulate matter < 2.5µm; 1996–2010) and O$_3$ (1996–2010) for the prospective cohort (n=7479) from Women's Health Initiative Memory Study, we examined whether long-term exposures affect the aging brain structure and dementia risk. Residing in places with high PM$_{2.5}$ (>12 µg/m³) increased the relative risk (by 81–92%) for global cognitive decline and all-cause dementia, with greater adverse effects in APOE ε4/4. In both region-of-interest and voxel-based morphometry (VBM) analyses of brain MRI (n=1300), increased PM$_{2.5}$ predicted smaller white matter volumes in multi-modal association regions (frontal; temporal). In VBM, PM$_{2.5}$ exposure also predicted smaller volumes of prefrontal cortex, but not the hippocampal-amygdalar complex (HAC). Preliminary results suggested long-term O$_3$ exposure was associated with smaller grey matter (bilateral HACs/temporal poles; left entorhinal/perirhinal cortices) and increased the dementia risk in older women.

PM2.5 AIR POLLUTION, EDUCATIONAL ATTAINMENT, AND COGNITIVE FUNCTION AMONG OLDER U.S. ADULTS

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This study examines whether the association between air pollution and cognitive function among older adults differs by educational attainment. Data on 13,389 adults over age 50 are from the 2004 Health and Retirement Study and were linked to 2004 annual average concentrations of ambient fine particulate matter air pollution (PM$_{2.5}$). Multilevel linear regression models were used to examine the association of PM$_{2.5}$ concentrations and individual-level education on cognitive function. Higher concentrations of PM$_{2.5}$ were associated with lower scores on cognitive function, but the association was stronger among individuals with lower educational attainment. These findings were robust to the inclusion of a variety of individual demographic, socioeconomic, and health factors as well as neighborhood-level education and income. The findings suggest the cognitive harms of pollution exposure may be offset by educational attainment, and that older adults with low educational attainment may be particularly vulnerable to air pollution.

AIR POLLUTION, MICROGLIA, AND THE LUNG-BRAIN AXIS

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Urban air pollution exposure has recently been linked to increased risk of several central nervous system (CNS) diseases and conditions, including cognitive decline and Alzheimer’s disease (AD). The mechanisms mediating these effects are poorly understood. Recent findings indicate that the brain’s innate immune cells, microglia, detect and respond to inhaled pollutants, where pulmonary damage may signal to the brain through circulating factors (The Lung-Brain Axis). Here, we will reveal the role of damage associated molecular patterns (DAMPs) in the microglial response to diesel exhaust particles, discuss the effects of these circulating factors in the 3x-TG murine AD model, and explore how aging may impact this process. These findings provide insight into the mechanisms underlying how air pollution may activate microglia and deleteriously impact central nervous system health.