



P102 Large Artery Stiffness is Associated with Lower Brain pH and Memory Performance in Middle-aged and Older Adults

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ABSTRACT

Background/Objectives: Large artery stiffening is a novel risk factor for cognitive impairment including Alzheimer's disease. Loss of impedance mismatch between central and cerebral arteries promotes cerebrovascular dysfunction via chronic transmission of excessive pulsatile pressure and flow to the brain. Cerebrovascular dysfunction uncouples cerebral blood flow supply from metabolic demand, contributing to hypoperfusion and acidosis. Conversely, greater impedance mismatch may protect against cerebrovasculature dysfunction, brain acidosis and cognitive impairment. Values for brain T1rho, a novel pH-sensitive MRI biomarker, are higher (more acidic) in patients with cognitive impairment compared with healthy controls. However, relations of T1rho with 1) large artery stiffness and central pulsatile hemodynamics and 2) memory performance are unknown.

Methods/Results: Middle-aged/older adults ($n = 31, 68 \pm 2$ years) underwent vascular, global T1rho MRI and memory testing. In a subset (n = 17), common carotid artery (CCA) intima-medial thickness (IMT) and pulsatile pressure/flow hemodynamics were measured (applanation tonometry, Doppler ultrasonography). In the entire cohort, higher T1rho was associated with greater aortic stiffness (cfPWV; r = 0.36, p = 0.054, covariate: MAP) and lower memory performance (r = -0.43, p = 0.03, education). In the subset, greater CCA IMT was correlated with higher cfPWV (r = 0.45, p = 0.08, MAP), suggesting that elevated aortic stiffness may promote concentric CCA remodeling. Higher CCA characteristic impedance was associated with lower T1rho (r = -0.57, p = 0.02) and higher memory performance (r = 0.46, p = 0.08). T1rho was not correlated with CCA reflection coefficient or pulsatile flow parameters (p > 0.05).

Conclusion: These preliminary data suggest that compensatory remodeling of the CCA artery associated with elevated aortic stiffness may be protective against alterations in brain pH and cognitive performance.

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