Title

Rates of Cognitive Decline and Alzheimer's Disease (AD) Neuropathology in Oldest-Old

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T1524. Rates of Cognitive Decline and Alzheimer's Disease (AD) Neuropathology in Oldest-Old

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Objective: To examine if rates of cognitive decline vary by level of AD neuropathology in oldest-old.

Methods: Participants were 68 autopsied individuals from ***The 90+ Autopsy Study***, a population-based longitudinal study of people aged 90 and older. Participants were non-demented at baseline and had 3 or more visits. Global cognition was assessed using the Mini-Mental State Exam (MMSE). Participants were categorized as having low or high AD neuropathology based on plaques (CERAD staging: Low = 0-A, High = B-C) and tangles (Braak staging: Low = 0-III, High = IV-VI). Random effects models were used to estimate rates of cognitive decline in people with low or high AD neuropathology.

Results: Rates of cognitive decline did not differ by level of AD neuropathology. Individuals with low plaques declined 0.63 points/year on the MMSE, whereas individuals with high plaques declined 0.50 points/year ($p = 0.43$). Individuals with low tangles declined 0.66 points/year on the MMSE, whereas individuals with high tangles declined 0.48 points/year ($p = 0.26$).

Conclusion: AD neuropathology is not associated with differing rates of cognitive decline in the oldest-old. Other factors such as health, lifestyle or other brain pathology may contribute to rates of cognitive decline in the oldest-old.

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T1525. Cortical Thickness on MR Imaging: Relation to Cognitive Reserve in Normal Aging and Mild Cognitive Impairment

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Resistance to cognitive decline from neuropathology is postulated to occur due to cognitive reserve (CR). We examined whether baseline regional cortical thickness and rate of regional atrophy are structural markers for CR. We hypothesized that higher education, a proxy for CR, would be related to greater cortical thickness in areas related to literacy or intellectual ability in healthy controls and individuals with mild cognitive impairment (MCI) from the Alzheimer's Disease Neuroimaging Initiative. Cortical thickness in these regions was compared between high (>18 yrs) and low education (<13 yrs) subgroups among controls and MCI individuals separately. Unexpectedly, high education was related to thinner cortices at baseline for controls in lateral occipital and temporal regions and for MCI in the left inferior parietal region after controlling for age and sex. In MCI, the difference in cortical thickness persisted after controlling for disease severity. Pre-existing thinner cortex in the high education group was not associated with a higher atrophy rate than that observed in the low education group. Cortical thickness in areas related to intellectual ability or literacy therefore may not be a persistent marker of CR.

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