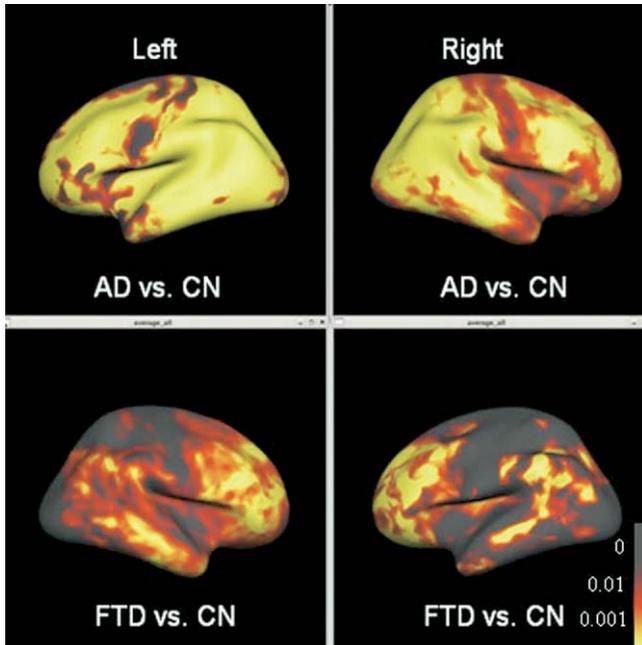


pared to FTD, AD was associated with thinner cortices in parietal regions, precuneus and posterior cingulate ($p < 0.01$). Compared to AD, FTD was associated with thinner cortices in orbitofrontal regions ($p < 0.01$). In general, cortical thickness provided no significant advantage over the corresponding volumes in classifying AD from CN and FTD. However, cortical thickness of parietal and temporal lobes (area under the ROC curve (AUC): 0.70-0.95 95% confident interval) showed a trend ($p = 0.06$) to better separate FTD from CN than the corresponding volume (AUC: 0.54-0.86). **Conclusions:** The characteristic patterns of cortical thinning in AD and FTD seen on MRI are consistent with pathological findings. Furthermore, cortical thinning may be a more prominent feature than volume loss for separating FTD from normal aging.



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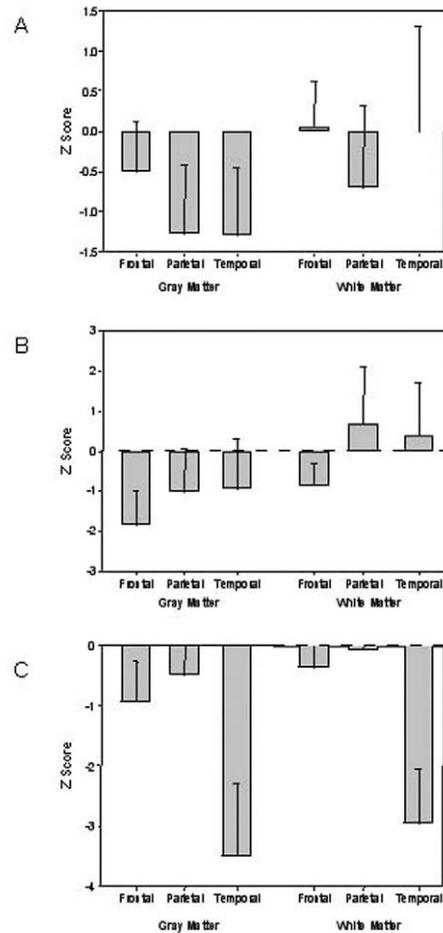
DIFFERENT PATTERNS OF GRAY AND WHITE MATTER ATROPHY IN ALZHEIMER'S DISEASE AND SUBTYPES OF FRONTOTEMPORAL LOBAR DEMENTIA

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Background: Alzheimer's disease (AD) and frontotemporal lobar degeneration (FTLD) are neurodegenerative diseases characterized by the progressive loss of cerebral tissue. Although structural magnetic resonance imaging (MRI) has been used extensively to investigate AD, and to a lesser extent FTLD, few studies have distinguished between the patterns of gray matter (GM) and white matter (WM) atrophy in these diseases. **Goals:** To identify the pattern of WM atrophy in two subtypes of FTLD—frontotemporal dementia (FTD) and semantic dementia (SD) and in AD relative to normal controls (CN). To compare the patterns of WM and GM atrophy in FTD, SD, and AD. **Methods:** Twelve FTD, 13 SD, 15 AD patients, and 24 cognitively normal subjects (CN) were studied with volumetric MRI. Regional GM and WM in each group were classified automatically from high resolution T1- and T2-weighted MRI using Expectation-Maximization Segmentation (EMS). Data were analyzed with analyses of covariance, with age and the white matter lesion volumes as covariates. **Results:** Figure 1 shows the Z-scores of GM and WM volumes by lobe for each patient group. Relative to CN, AD patients had significant GM atrophy in the parietal and temporal lobes ($p < 0.001$). Although there was a trend for

WM atrophy in the parietal lobe, there were no significant overall WM volume differences between AD and CN. FTD patients had significant GM and WM atrophy in the frontal lobe relative to CN ($p < 0.001$). There were trends of GM atrophy in the parietal and temporal lobes, but no WM atrophy in these regions. Compared to CN, SD patients had significant GM atrophy in both temporal and frontal lobes ($p < 0.001$), but significant WM atrophy only in the temporal lobe ($p < 0.0001$). **Conclusions:** These results suggest that different types of dementias are associated with different patterns of WM atrophy and that the pattern of WM atrophy differs by lobe. Whether WM atrophy in these diseases indicates general neuritic dystrophy, involving both neuron cell bodies and axons, or Wallerian degeneration remains to be explored.

Figure 1. Z scores of GM and WM volume in the different lobes for (A) AD patients, (B) FTD patients, and (C) SD patients.



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HIPPOCAMPAL ACTIVATION IN MCI PREDICTS SUBSEQUENT COGNITIVE DECLINE

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Background: We previously reported that fMRI hippocampal activation is greater in subjects with mild cognitive impairment (MCI) who subsequently demonstrate further cognitive decline. **Objective:** We investigated whether fMRI hippocampal activation predicts the degree of subsequent cognitive decline in MCI, and whether or not it predicts this degree of decline even after controlling for clinical predictors. **Methods:** Subjects included 27 older participants (ages 65-86) in a longitudinal memory and aging study for whom an average of 4.5 years (S.D. 1.1) of follow-up