Mapping Dynamic Changes in Ventricular Volume onto Baseline Cortical Surfaces in Normal Aging, MCI, and Alzheimer's Disease

S.K. Madsen, B.A. Gutman, S.H. Joshi, A.W. Toga, C.R. Jack, Jr., M.W. Weiner & P.M. Thompson for the Alzheimer's Disease Neuroimaging Initiative (ADNI)

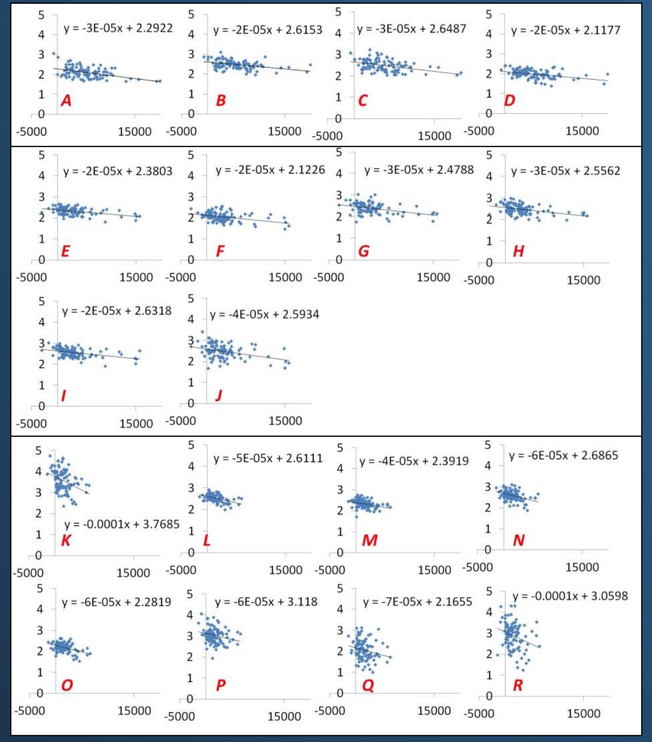
Introduction

Ventricular volume (VV) is a powerful global indicator of brain tissue loss on MRI in normal aging and dementia. VV is used by radiologists in clinical practice and has one of the highest obtainable effect sizes for tracking brain change in clinical trials, but it is crucial to relate VV to structural alterations underlying clinical symptoms.

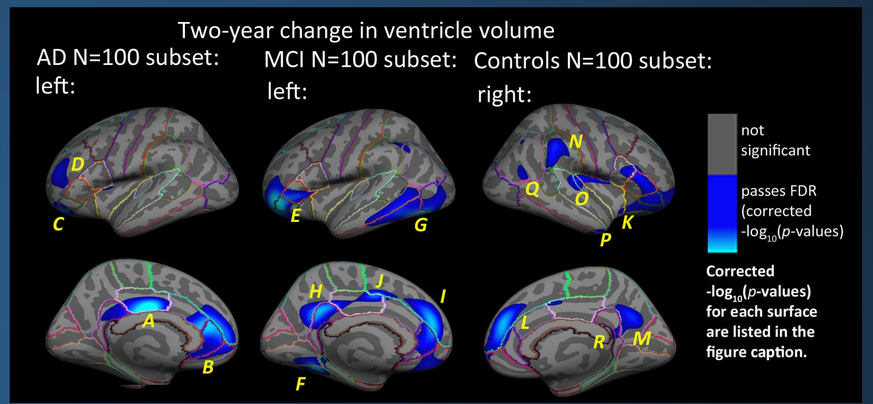
Here we identify patterns of thinner cortical gray matter (GM) associated with dynamic changes in lateral VV at 1-year (N=677) and 2-year (N=536) intervals in the ADNI cohort.

Methods

We plotted 2-year VV change against mean baseline GM thickness (*x*-axis: raw 2-year VV change in mm^3, *y*-axis: mean baseline GM thickness for statistically significant regions in mm). Each data point represents one subject within the matched N=100 subsets for AD, MCI, and healthy elderly control groups (AD: first row, MCI: second and third rows, Controls: last two rows). Each plot represents a distinct and continuous cortical region that passed correction with FDR in the surface GLM maps shown in the top panel of this figure.



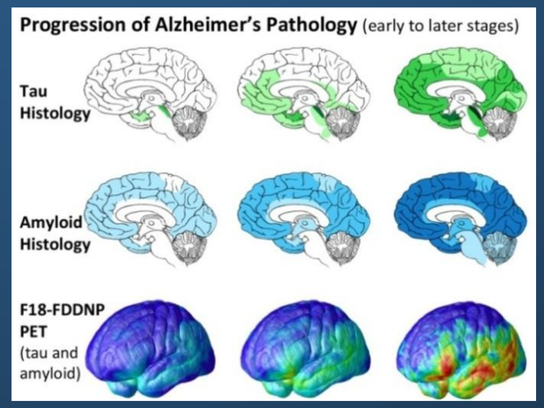
Results



Hemispheric 3D maps of significant negative associations between 2-year change in VV and cortical GM thickness in matched N=100 sub-samples for AD, MCI, and healthy elderly individuals, after controlling for age and sex.

Discussion

People with faster VV loss had thinner baseline cortical GM in temporal, inferior frontal, inferior parietal, and occipital regions (controlling for age, sex, diagnosis). These findings show the patterns of relative cortical atrophy that predicts later ventricular enlargement, further validating the use of ventricular segmentations as biomarkers. We may also infer specific patterns of regional cortical degeneration (and perhaps functional changes) that relate to VV expansion.



Canonical progression of AD pathology has been mapped previously in non-overlapping elderly samples. These patterns agree well with those seen in our cortical mapping of changes in VV, with significant associations in areas known to be susceptible to AD pathology and no detected relationship in areas that do not have a significant disease burden (primary sensorimotor cortex).

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