Control Energy of Temporal Regions Relates to Tau Burden and Longitudinal Memory Decline in Preclinical Alzheimer's Disease

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INTRODUCTION

Tau deposition is common in specific regions of the temporal lobe in early Alzheimer's disease (AD cortical signature)[1]. Tau accumulation in the AD cortical signature is linked with functional disconnection of temporal regions, tau-related disruption of memory systems, and memory loss[2]. Control energy is a network neuroscience metric that quantifies the theoretical energy required to drive brain activity changes in downstream target regions[3].

Hypotheses

I. Tau accumulation increases the energy needed to control the activity of AD cortical signature regions from the rest of the brain

II. Higher control energy of AD cortical signature temporal regions is associated with worse memory scores

METHODS

Study sample

Cognitively Normal Amyloid-positive
Mean age: 72 ± 5 yrs.
n = 15

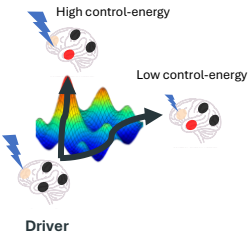
Memory score

RAVLT forgetting (higher = worse)

Regions of interest

AD cortical signature

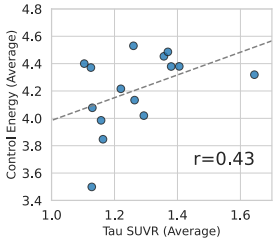
Control energy



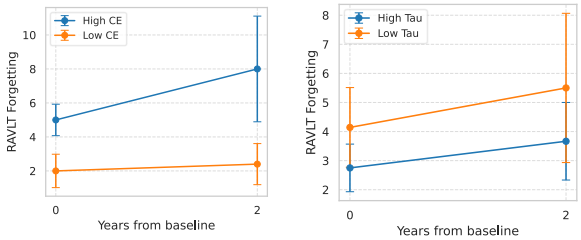
Theoretical input to shift brain activity in target region
Interpretation: Lower energy = transition more easily supported by network

RESULTS

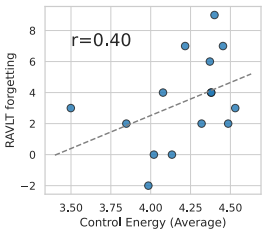
1 Higher tau → Higher control energy



3 Higher baseline control energy → Worse memory over time



2 Higher control energy → Worse memory



CONCLUSIONS

Control energy may serve as a biomarker to identify cognitively unimpaired amyloid-beta positive individuals at risk for accelerated decline, warranting validation in larger, longitudinal cohorts.

References

1. Jack C, Wiste HJ, Weigand SD, et al. Different definitions of neurodegeneration produce similar amyloid/neurodegeneration biomarker group findings. Brain. 2015;138(Pt 12):3747-3759.
2. Harrison T, Maas A, Adams JN, et al. Tau deposition is associated with functional isolation of the hippocampus in aging. Nat Comms, (2019)10:4900, https://doi.org/10.1038/s41467-019-12921-z.
3. Manjunatha KKH, Baron G, Benozzo D, et al. Controlling target brain regions by optimal selection of input nodes. PLOS Computational Biology. 2024 gen;20(1):e1011274.

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