

Cognition Colloquium

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Targeting Dysfunction in Episodic Memory Circuitry in Alzheimer's Disease

Alzheimer's disease causes pathology to spread along neural circuits that serve specific cognitive processes, such as pattern separation and pattern completion and familiarity recognition. I will discuss how amyloid and tau pathology impact on synaptic function in these circuits and how brain dysfunction can be integrated in a multi-modal disease progression model of Alzheimer's disease. These data have implications on the question whether synaptic function can partially recover after amyloid removal with disease modifying anti-amyloid treatments. Building on these observations, I will discuss how non-pharmacological interventions can be used to unlock reserve mechanisms in conjunction with amyloid-removal. Our data indicate that cognitive reserve can be associated with the ability to activate upstream visual areas and midline cortical areas of the episodic memory circuitry. Individuals who maintain high levels of encoding related activation in these areas can cope with hippocampal atrophy, amyloid and tau-pathology, such that they show slower cognitive decline in memory over several years of follow-up. I will integrate these data in with observations from Super Agers and into a framework called the Circuit Utilization Framework, aimed to optimize the treatment of patients with Alzheimer's disease, also in conjunction with amyloid antibody treatments.



Join online:

<https://zoom.us/j/93526030034?pwd=ZkJnYlFVOEthU2lDeE5nVmV6TlZLZz09>

Meeting ID: 935 2603 0034

Passcode: 250171