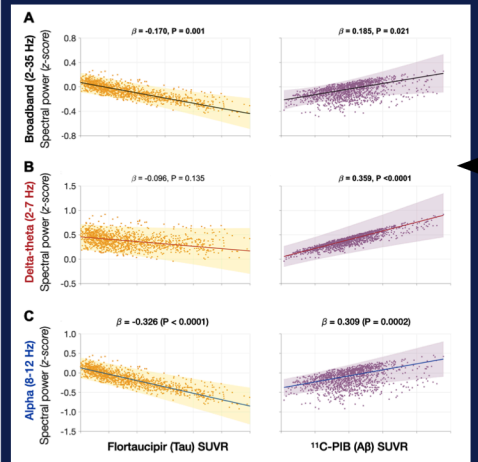
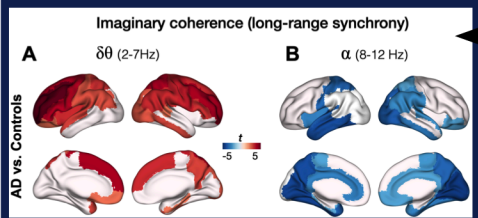
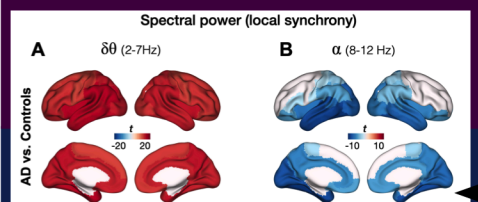
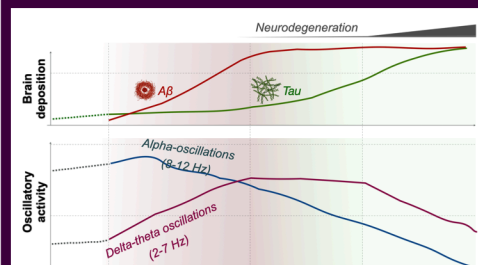
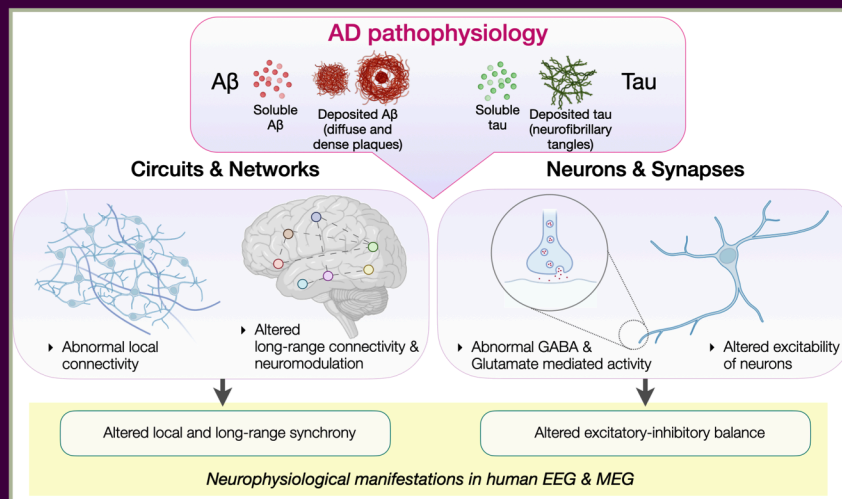




Neurophysiological Changes Associated With Pathological Processes of A β & tau in Patients with Alzheimer's Disease

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Frequency-specific local and long-range synchrony manifestations: reduced alpha and increased delta-theta oscillatory activity constitute signature changes in AD.

Distinct associations between neuronal synchrony deficits and tau and A β .

- Amyloid consistently increased oscillatory activity while tau consistently reduced oscillatory activity.

Neurophysiological changes in AD include quantifiable oscillatory alterations and excitatory-inhibitory imbalances. These abnormalities scale from synapses and neurons to circuits and networks. Our findings suggest that A β and tau contribute through distinct mechanisms. A clearer mechanistic understanding of these functional abnormalities is therefore essential for developing effective therapies.

Neural excitability and neural fragility capture distinct mechanisms of regional E/I imbalance, reflecting local intrinsic synaptic processes and long-range synaptic integration, respectively.

A β and tau are associated with distinct mechanisms of E/I imbalance, with the AD-epileptic phenotype showing heightened long-range synaptic vulnerability.

