2020 IMPACT CIRCLE

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Investigating Synapse Decline in Alzheimer's disease

Trillions of synapses in the brain form the connections between neurons that enable cognitive processes such as memory. In fact, the dynamic modulation of synapse strength is critical for the encoding of new memories. Long-term potentiation (LTP) is the enhancement of synapse strength that occurs during memory formation and it requires the orchestration of many synaptic proteins and signaling pathways. Previous research supports that LTP is inhibited by the toxic tau protein that accumulates in the brain in Alzheimer's disease and related dementias. However, how toxic tau blocks the LTP from occurring at synapses is unknown.

We hypothesize that toxic tau disrupts a subset of proteins at synapses which blocks LTP and promotes memory loss in Alzheimer's disease. Furthermore, we hypothesize that by manipulating the activity of these dysregulated synaptic proteins during LTP we may be able to alleviate the toxicity caused by tau and restore memory.