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function, including a loss of memory, which is a major health issue in old age. In this proposal we aim to determine which signaling pathway is critical for the agerelated learning and memory impairment, particularly during the period of learning. We have found that levels of secreted BDNF are decreased in the hippocampus during learning in aged rats. This decrease is correlated with a decrease in the activity of glycogen synthase kinase 3 (GSK-3), a kinase that phosphorylates transcription factors, including CREB. Importantly, treatment with a GSK-3 inhibitor during learning completely blocks the age-related decline in memory formation. We will use molecular, pharmacologic and genetic approaches to test the central hypothesis that the loss of BDNF signaling in the hippocampus during learning contributes to the age-related cognitive decline. In addition, we will determine whether agerelated changes in GSK-3 activity are the result of a decline in brain function or a

compensatory response to a decline in signaling in the aging brain. Finally