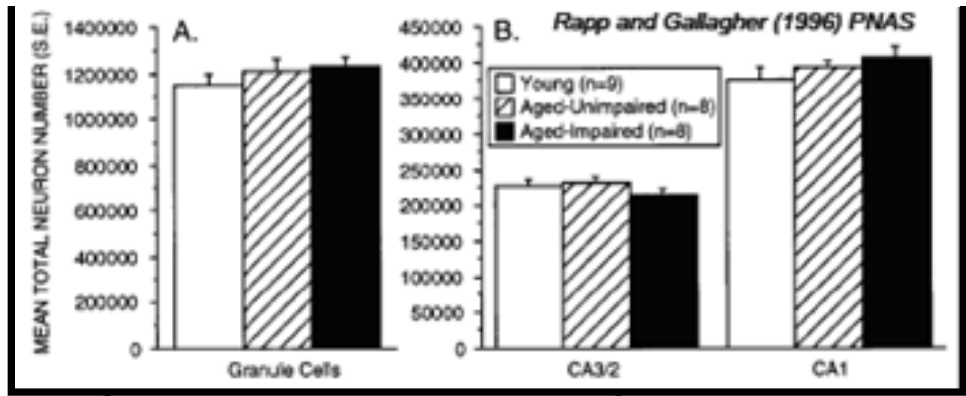
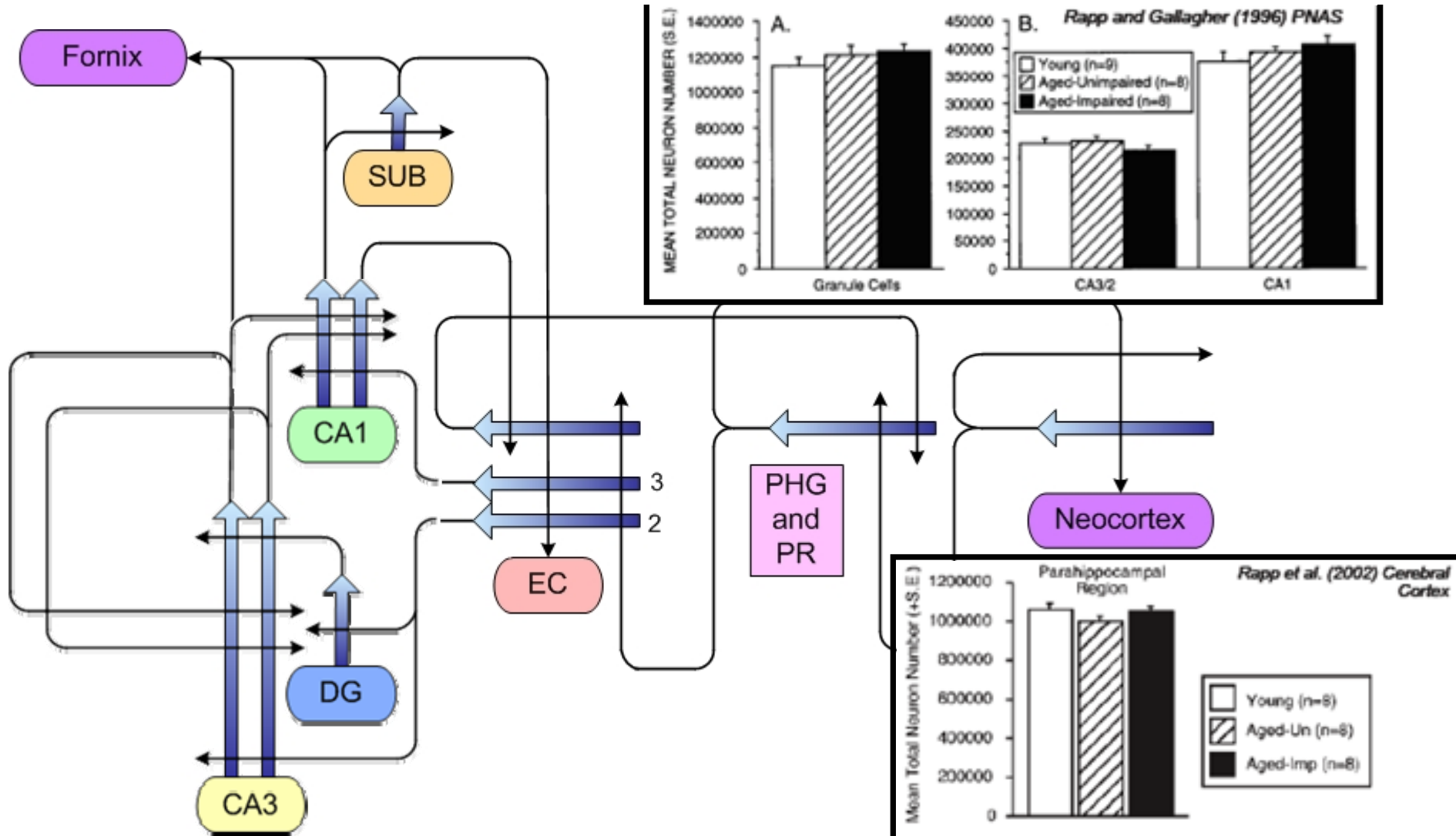


Neurocognitive aging and plasticity

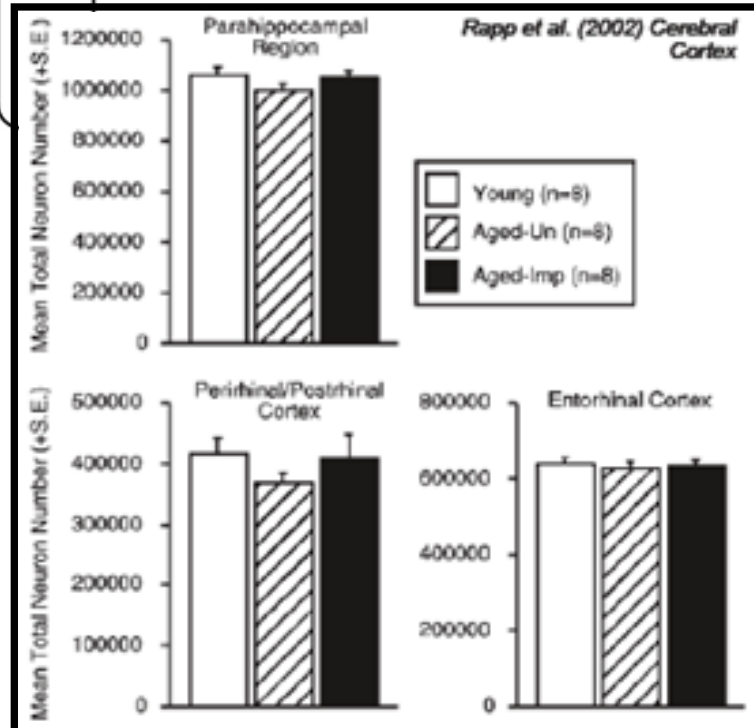
Michela Gallagher

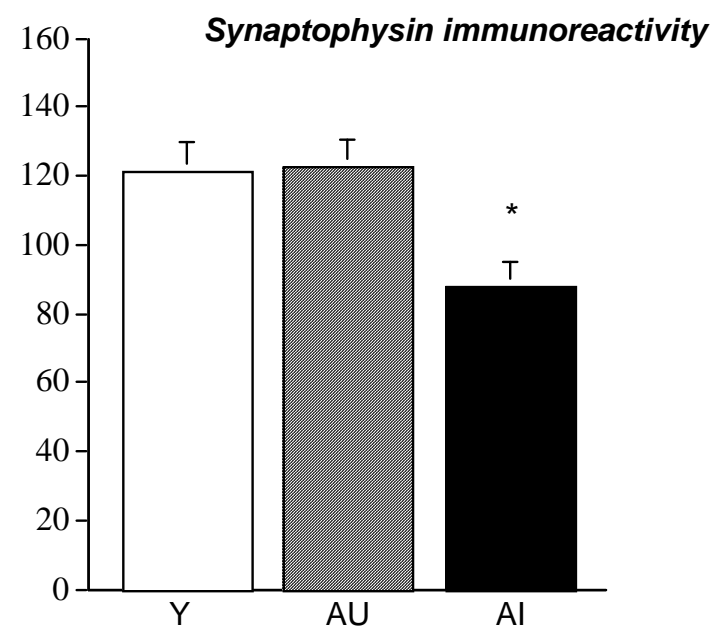
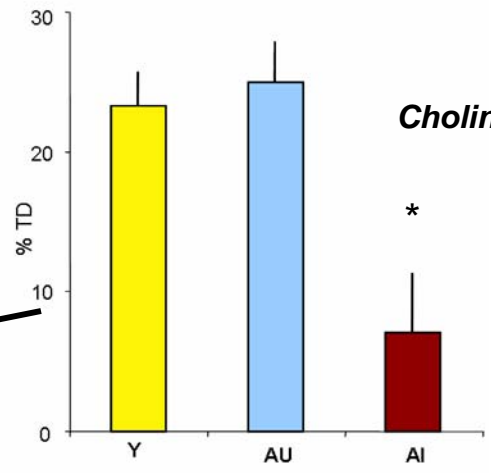
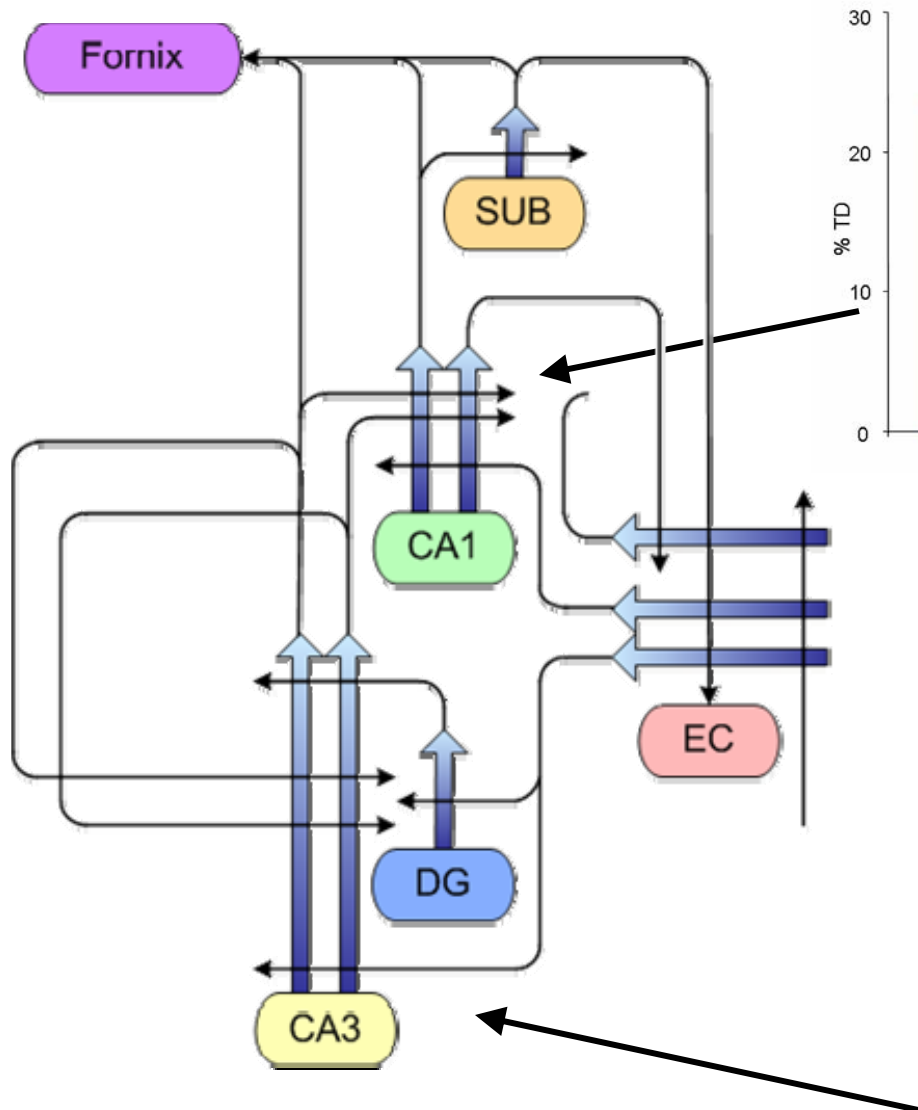
JOHNS HOPKINS
UNIVERSITY



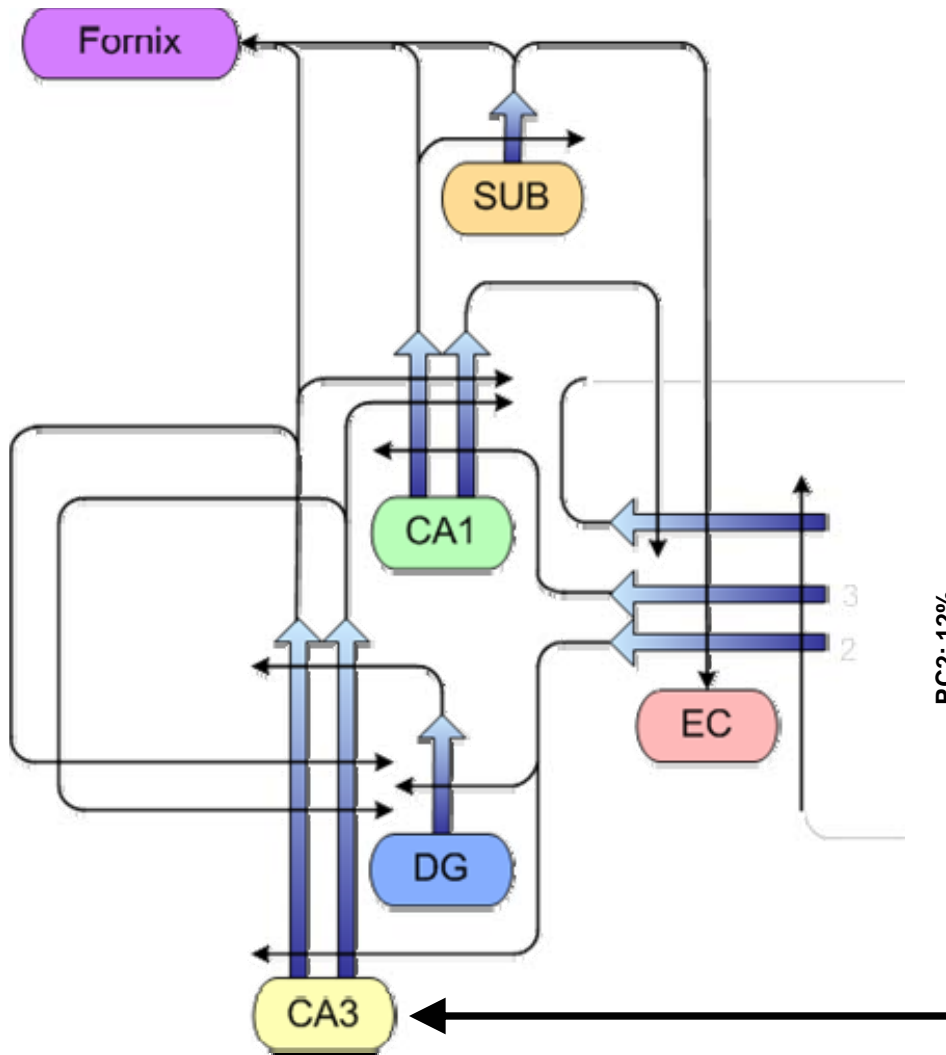


No difference in numbers of neurons

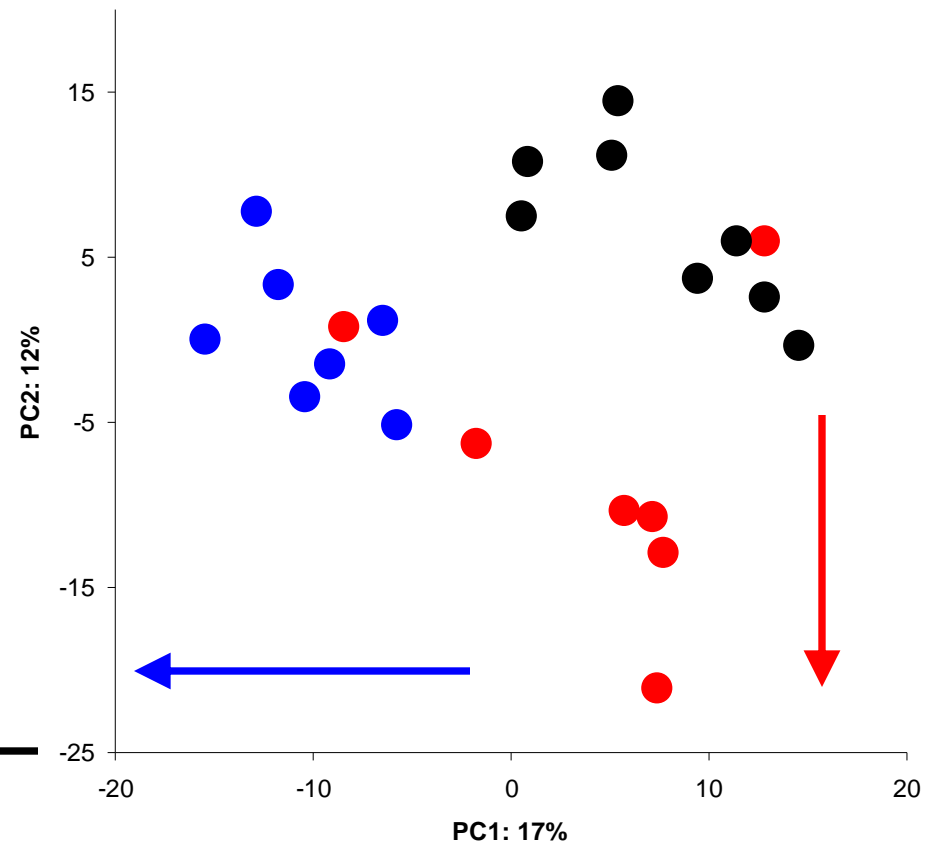




Synaptic basis of neurocognitive failure



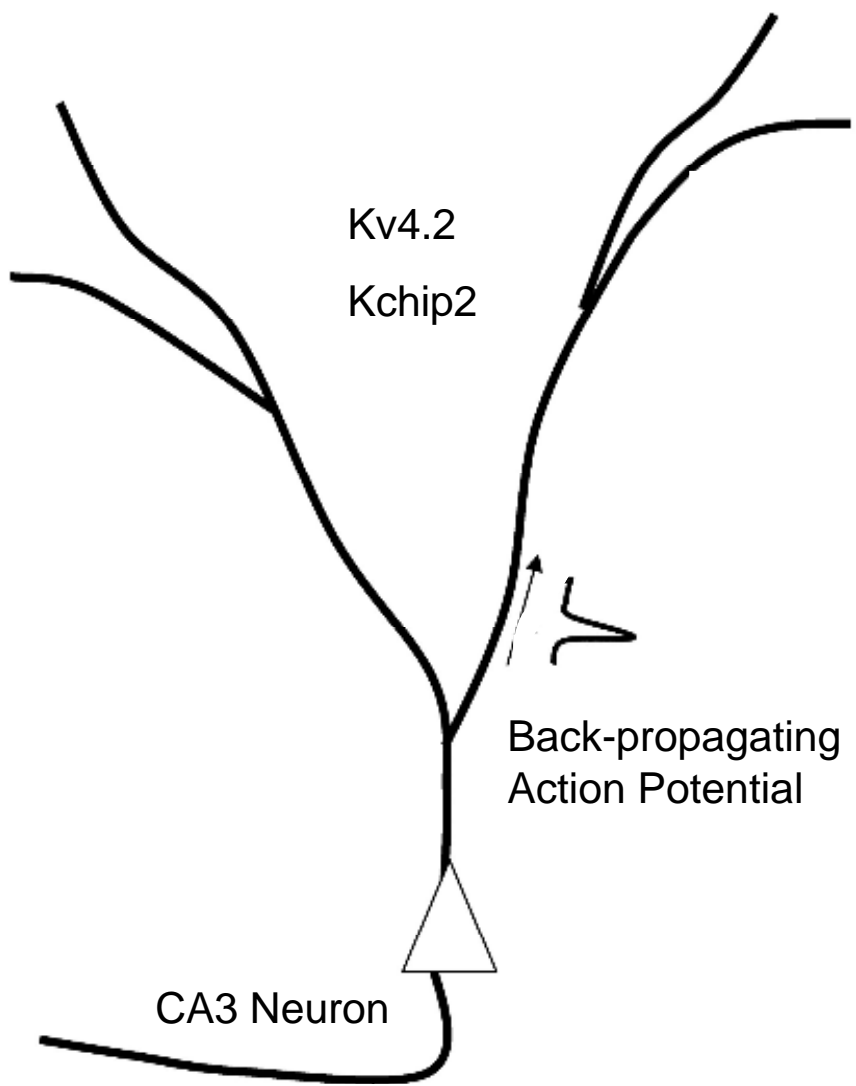
Principal Component Analysis(PCA) of CA3



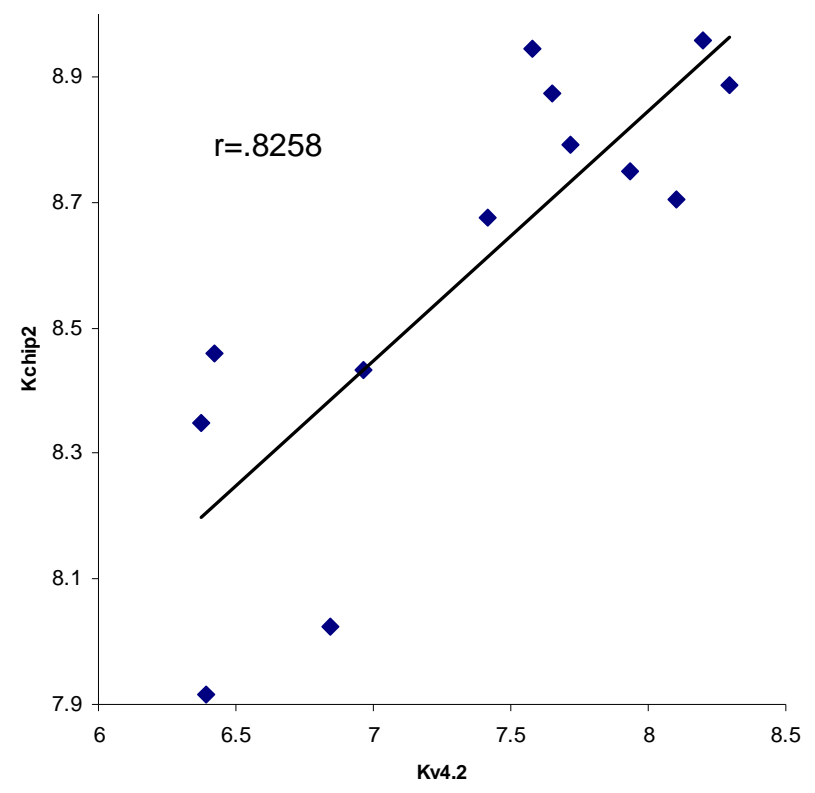
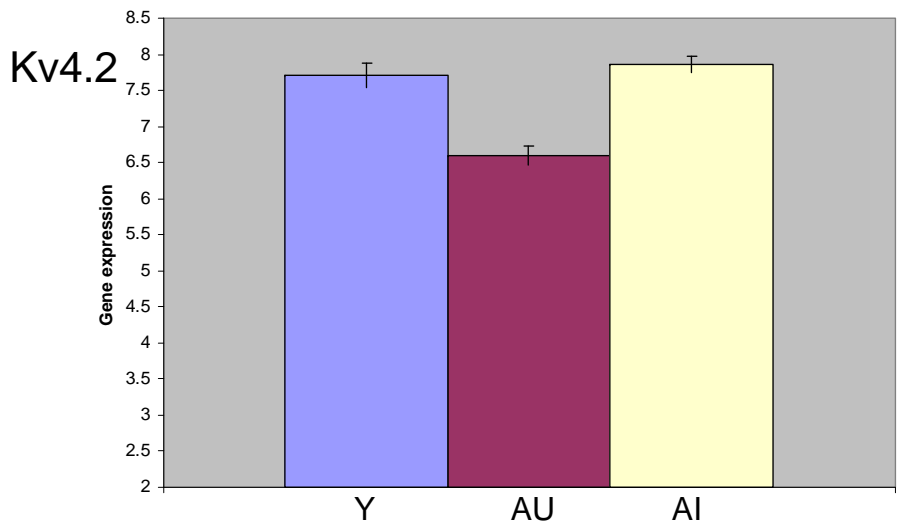
Basis of neurocognitive preservation

A broad molecular profile (>20,000 genes) indicates distinctive changes (PC1 on bottom axis and PC2 on Horizontal axis) in the memory system network in Aged impaired (blue dots) and Aged unimpaired (red dots), both separating from young (black dots).

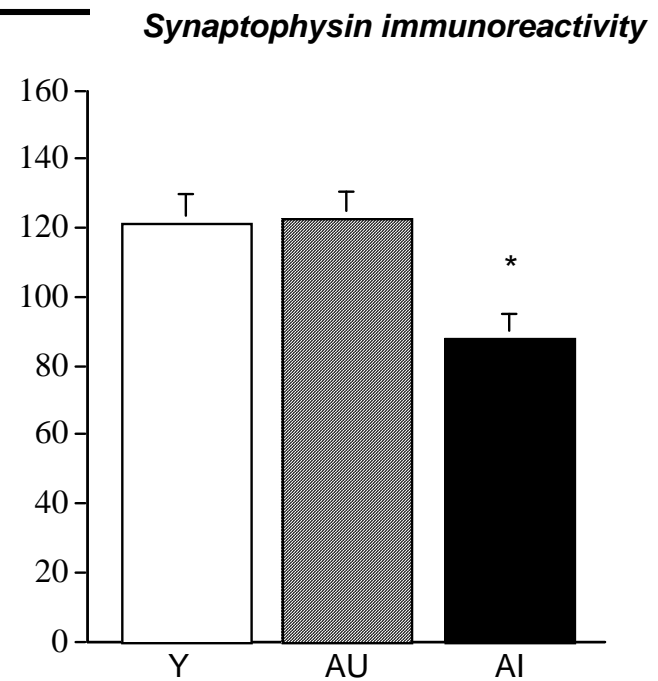
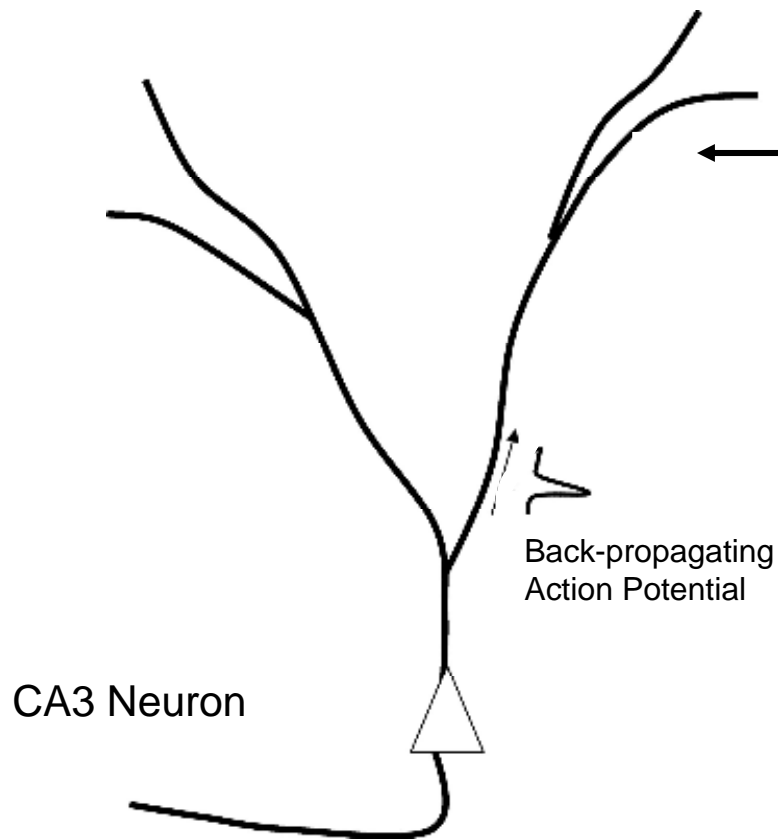
Data illustrate a change in aged unimpaired gene expression for a functional unit that could boost information storage



Adapted from Birnbaum, S. G. et al. *Physiol. Rev.* 84: 803-833 2004



Neurocognitive preservation is 'robust aging'
compensatory, adaptive **AND** protective



with
Peter Rapp
Howard Eichenbaum
Heikke Tanila
Ian Wilson
Jennifer Bizon
Rebecca Haberman
Carlo Colantuoni
Alfredo Kirkwood
Hey-Koung Lee
Ming-Teng Koh

National Institute on Aging
Bristol-Myers Squibb Foundation

JOHNS HOPKINS
UNIVERSITY

