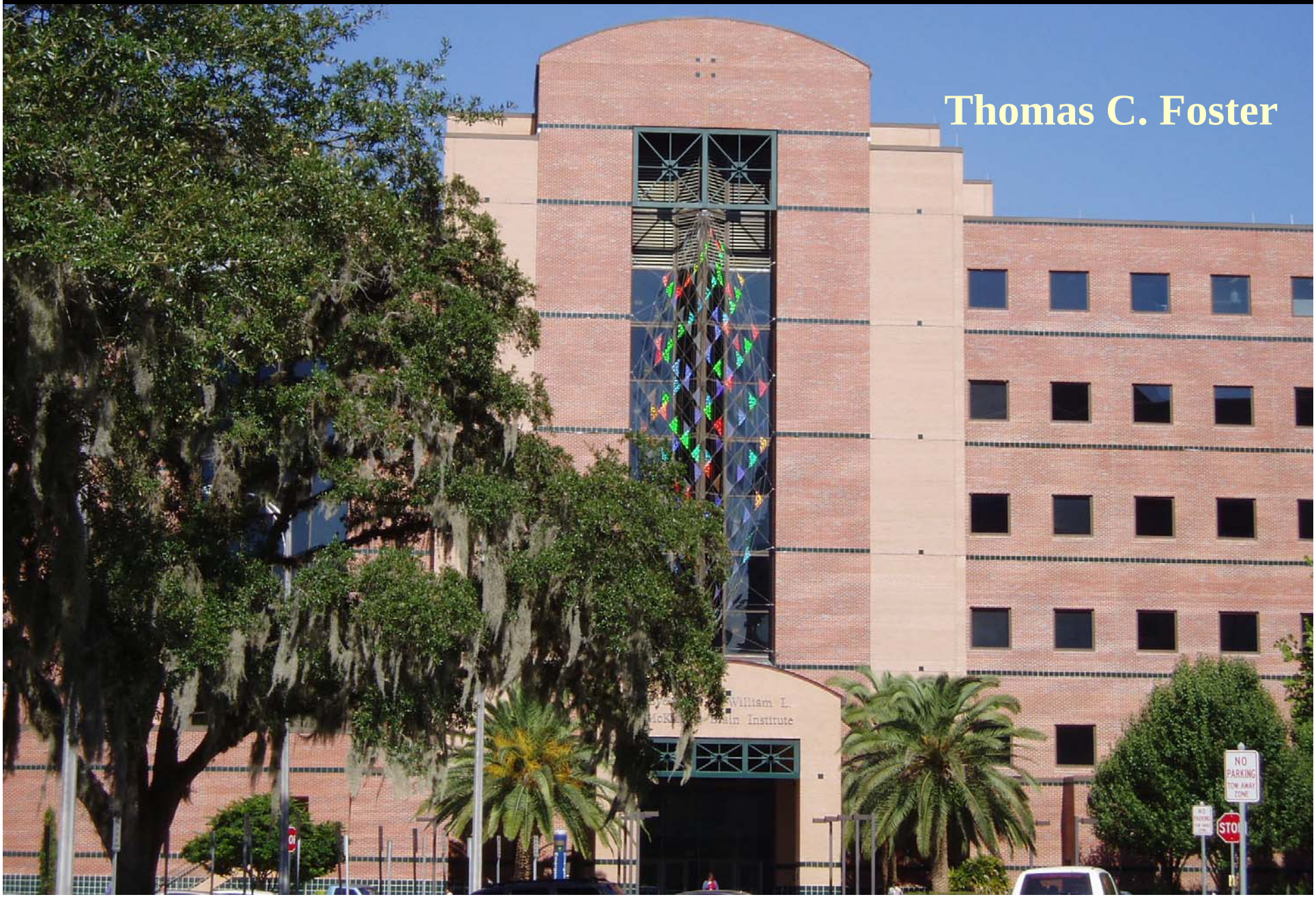
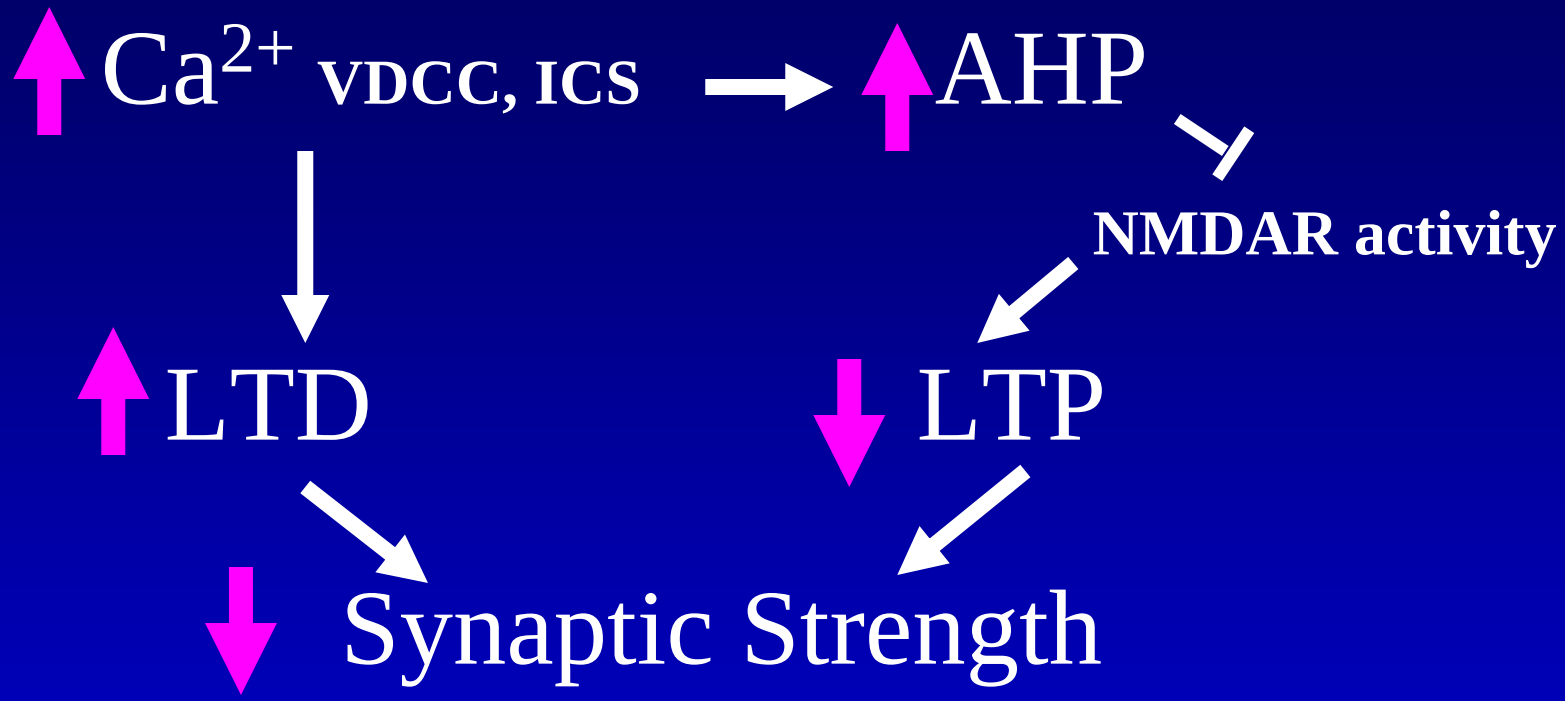
Synaptic plasticity and the aging brain

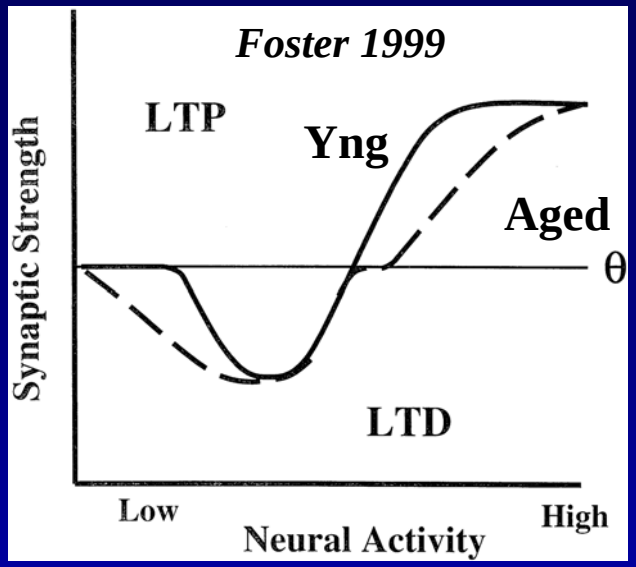


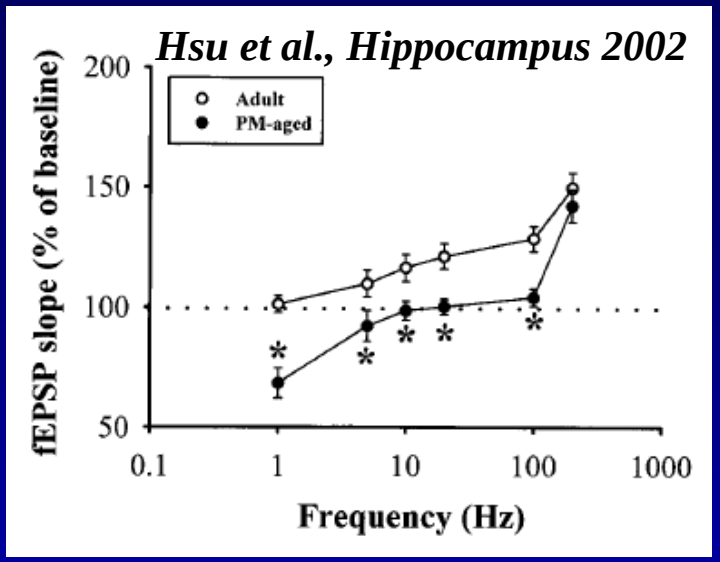
Altered Ca^{2+} homeostasis & disruption of synaptic plasticity



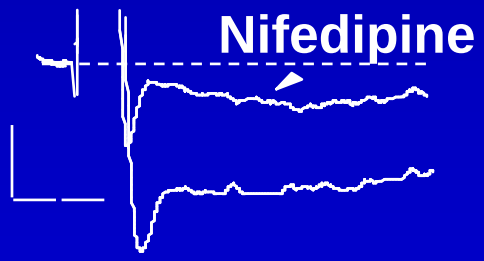
The shift in the balance of LTP/LTD, favoring LTD, may contribute to a decrease in synaptic strength and memory impairments.

An age related shift in the threshold for synaptic plasticity

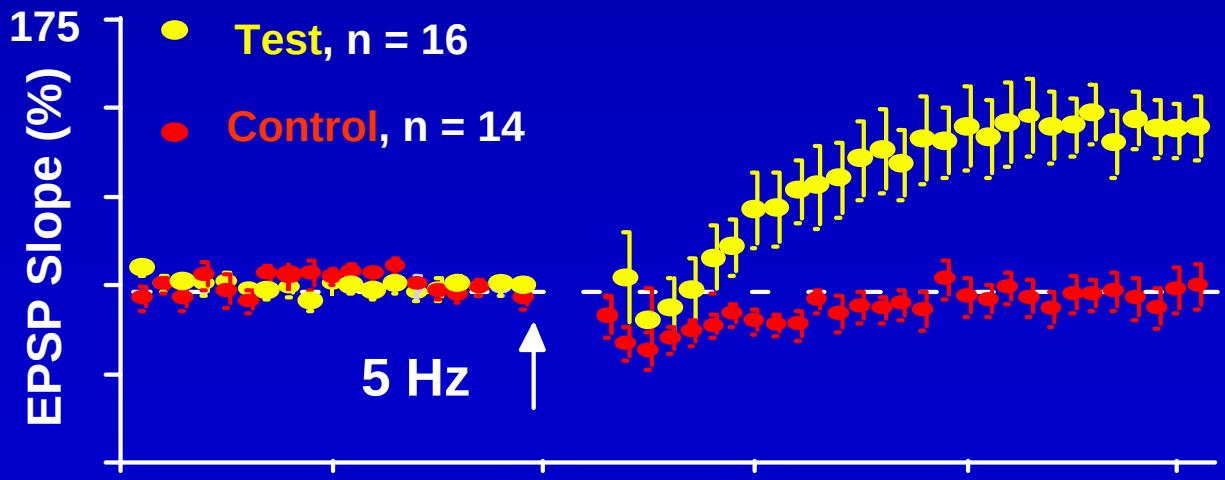




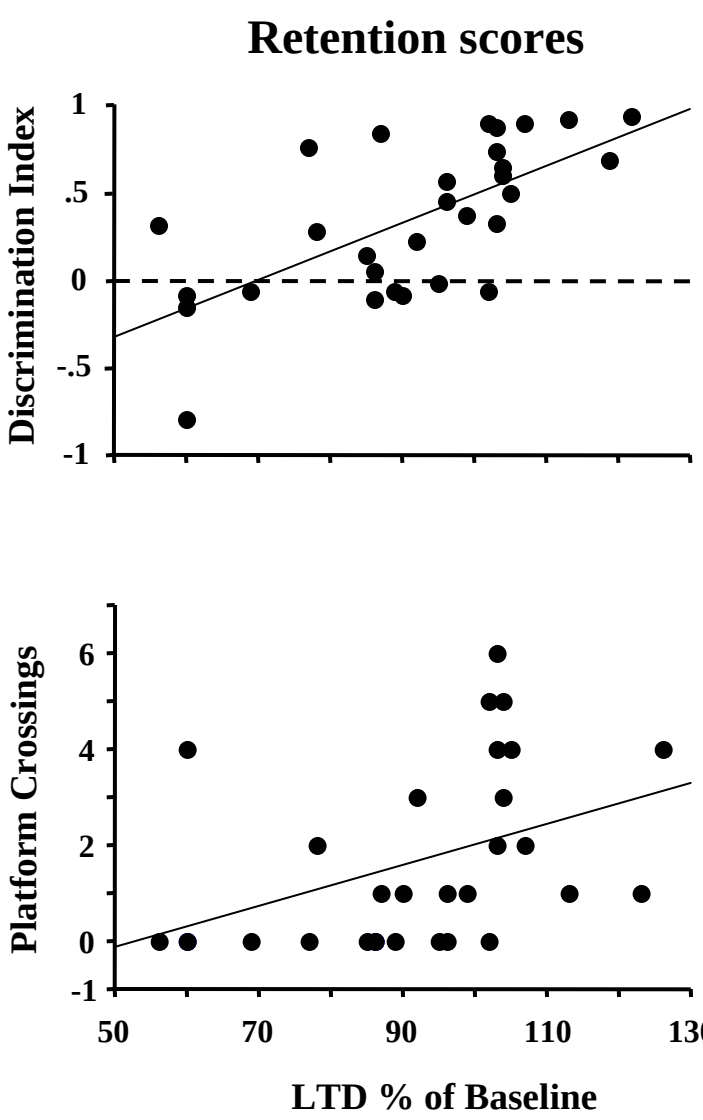
L-channel blockade permits LTP in aged animals

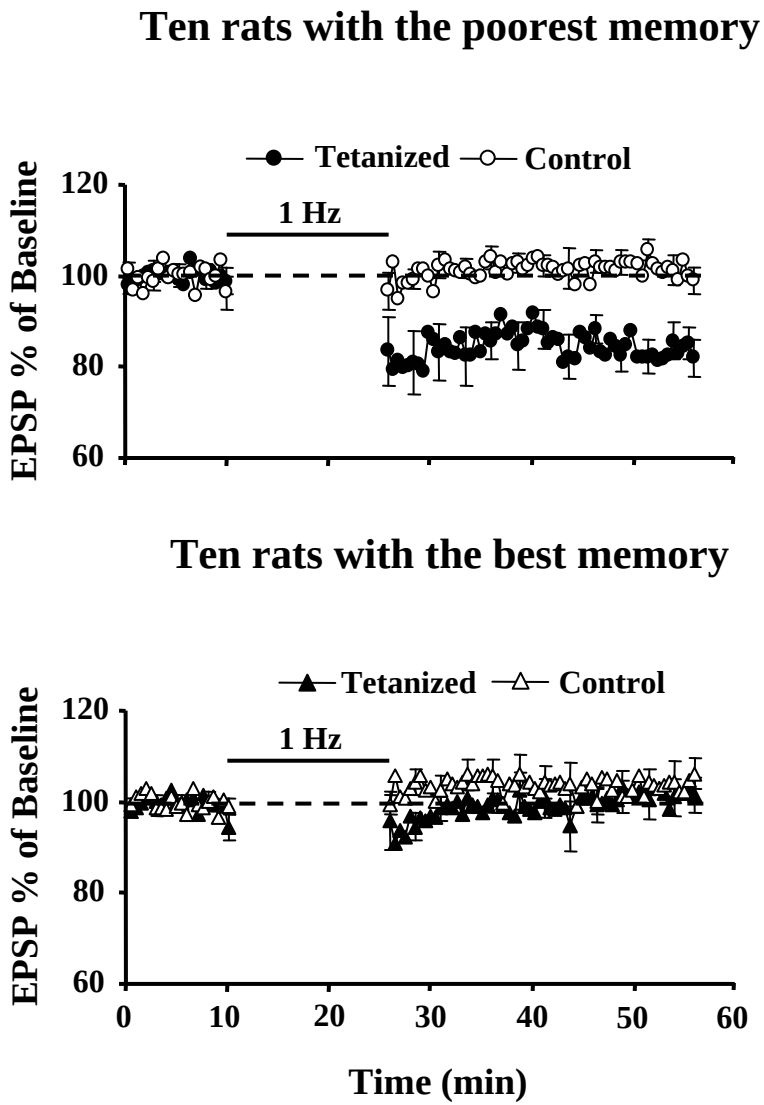


Norris 1998

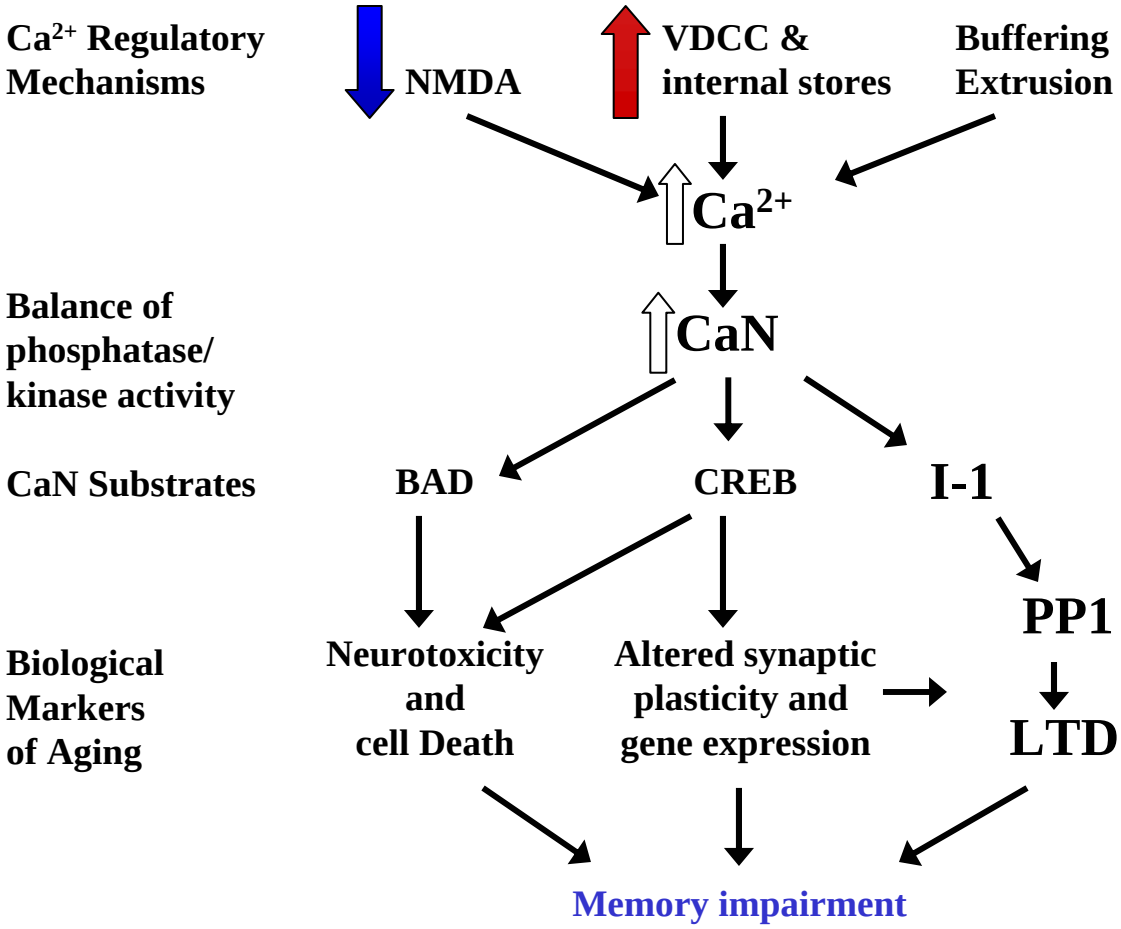


Impaired memory is correlated with age-related changes in synaptic plasticity

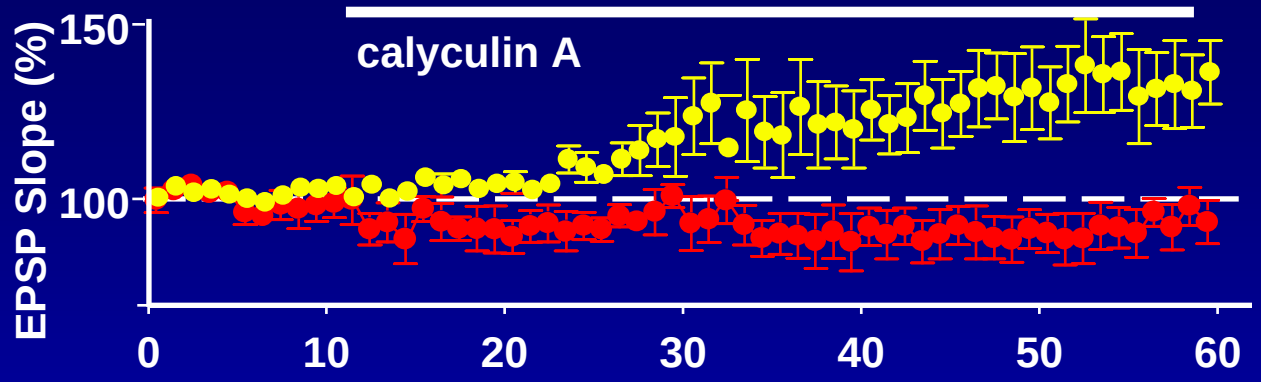


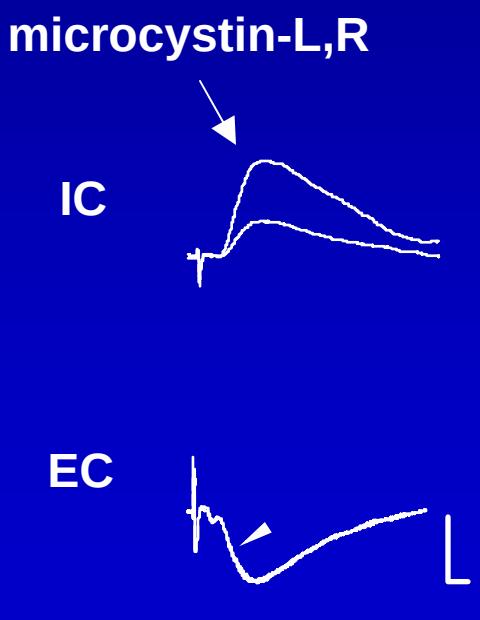


Biochemical model linking altered Ca^{2+} homeostasis with memory impairment

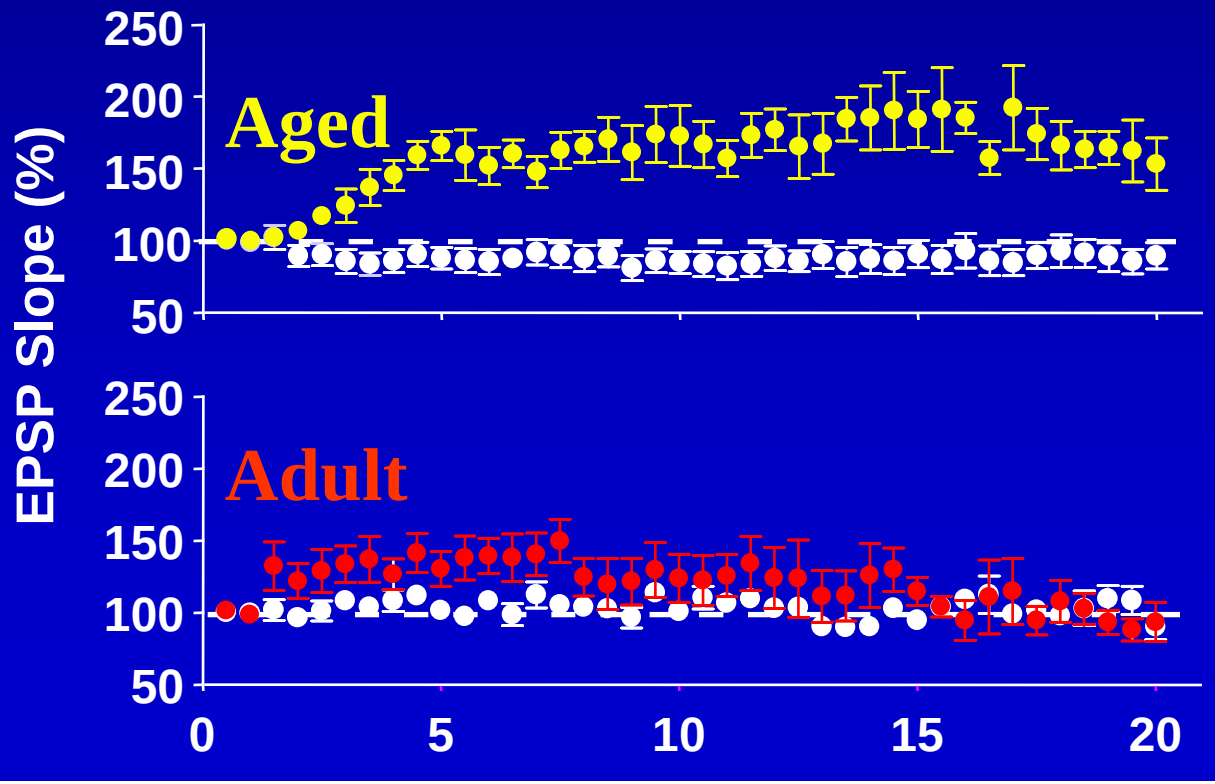


Decreased synaptic strength during aging is due, in part, to increased phosphatase activity

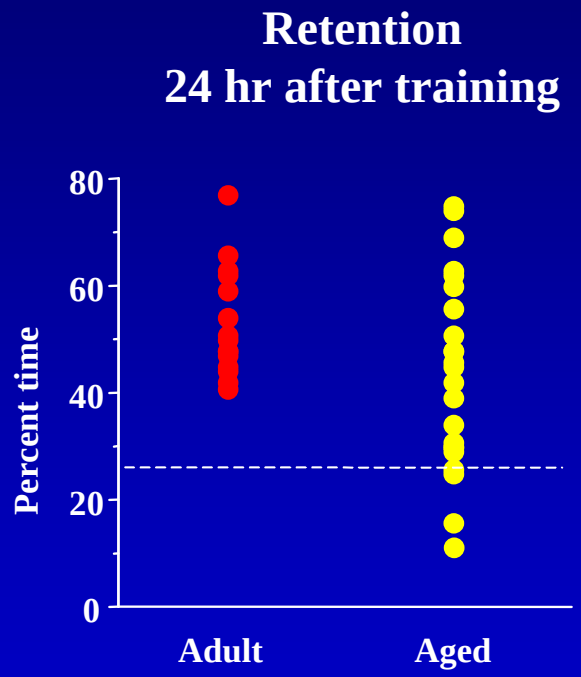


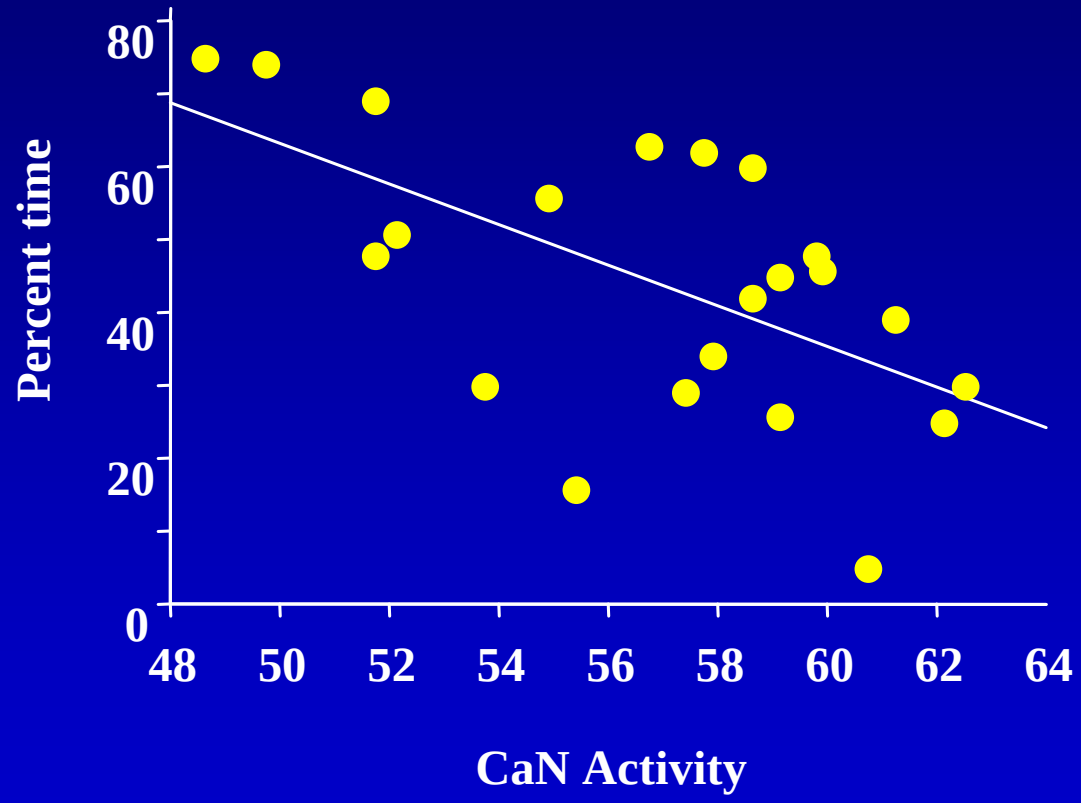


Norris et al., 1998



Phosphatase activity linked to impaired memory





Foster et al, 2001

Protein phosphatase 1 is a molecular constraint on learning and memory

David Genoux*, Ursula Haditsch*, Marlen Knobloch*, Aubin Michalon*, Daniel Storm† & Isabelle M. Mansuy*

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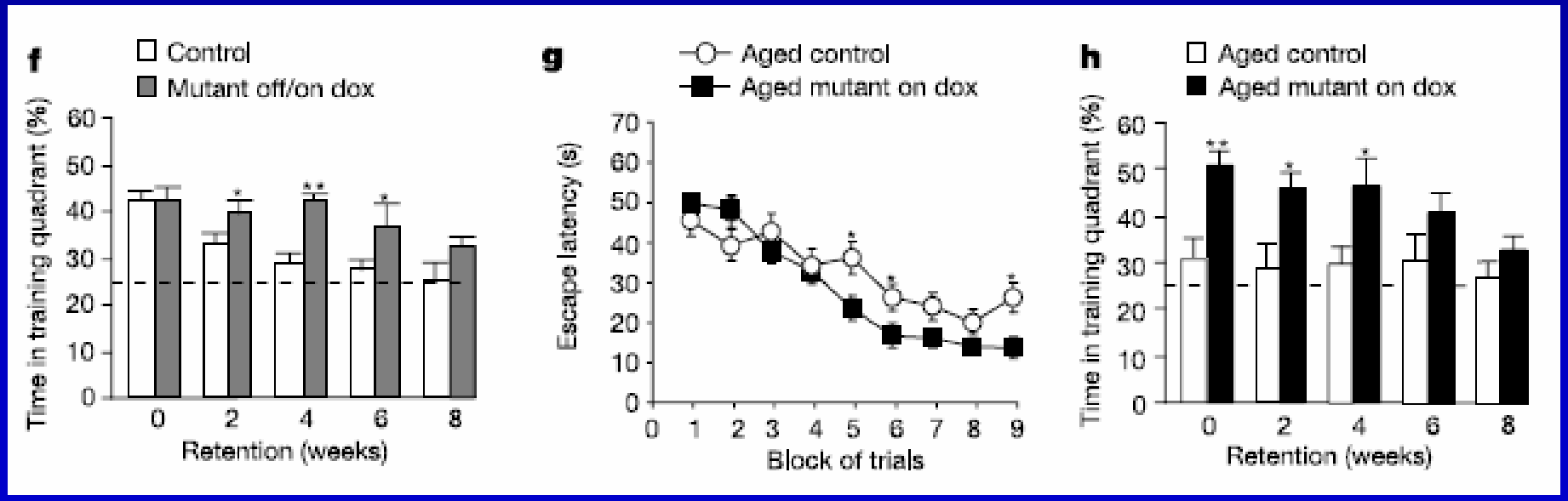
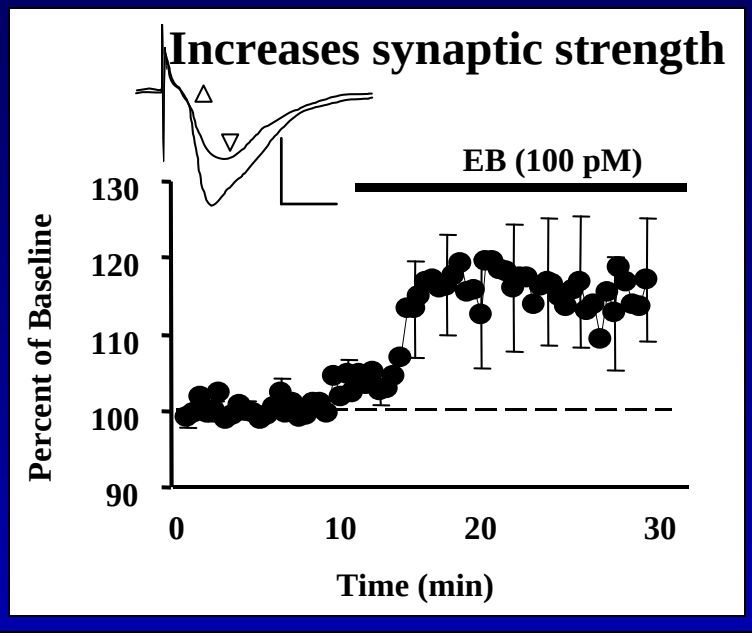
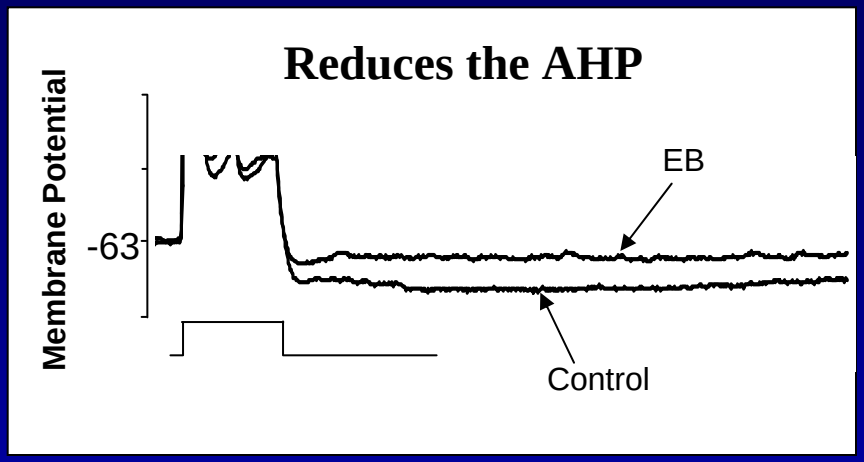
Post training genetic inhibition of PP1 activity prolongs memory in young mice

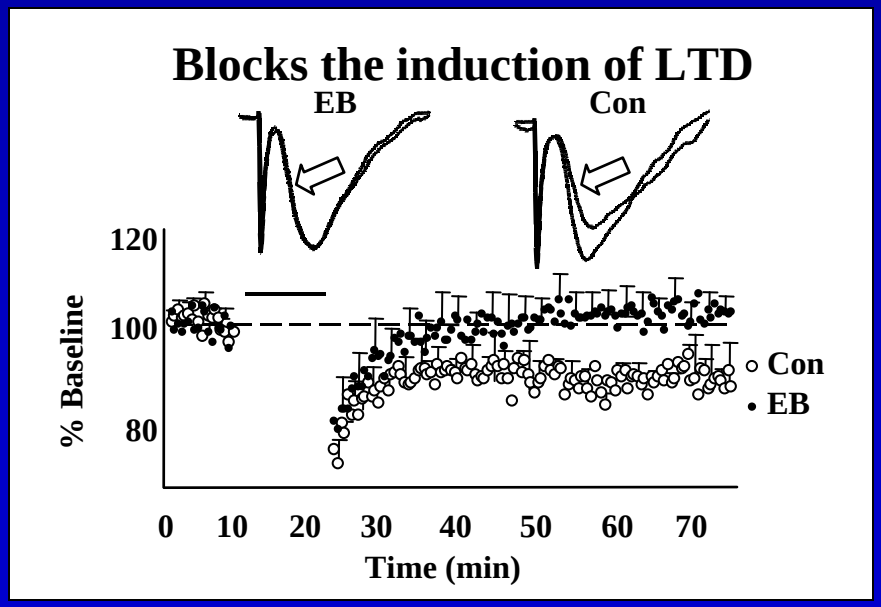
Genetic inhibition of PP1 activity prolongs memory in aged mice

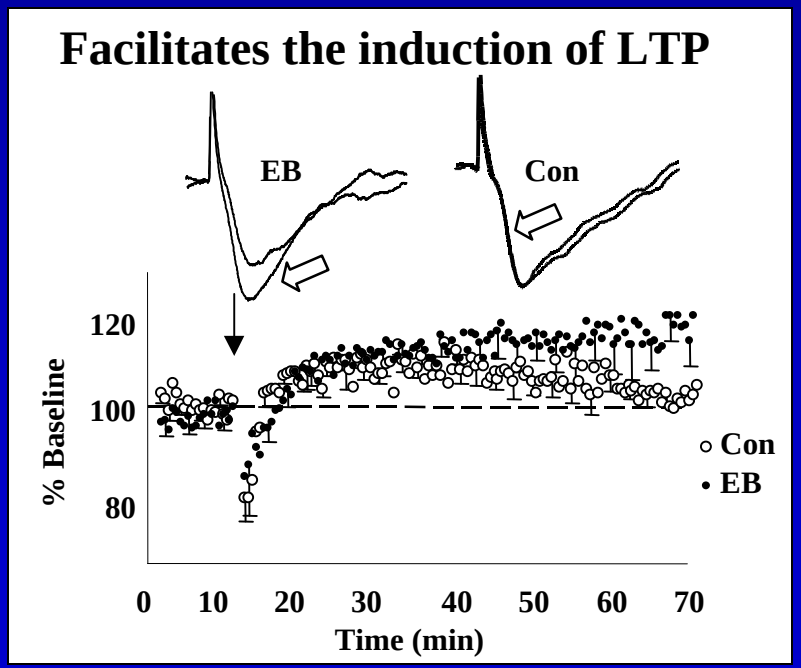


Rapid estrogen effects are diametrically opposite that observed during aging

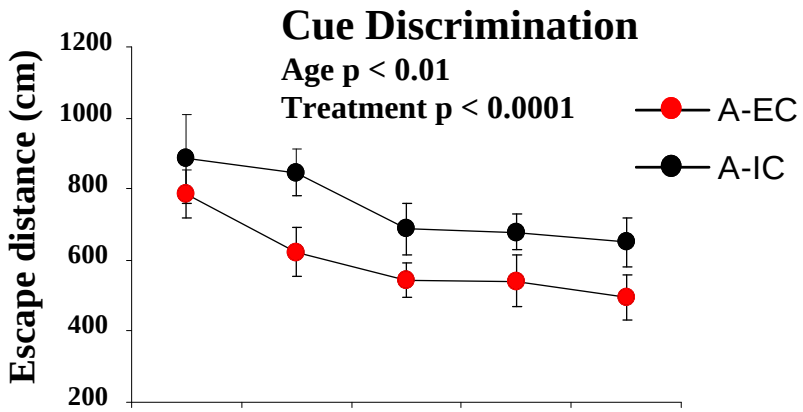


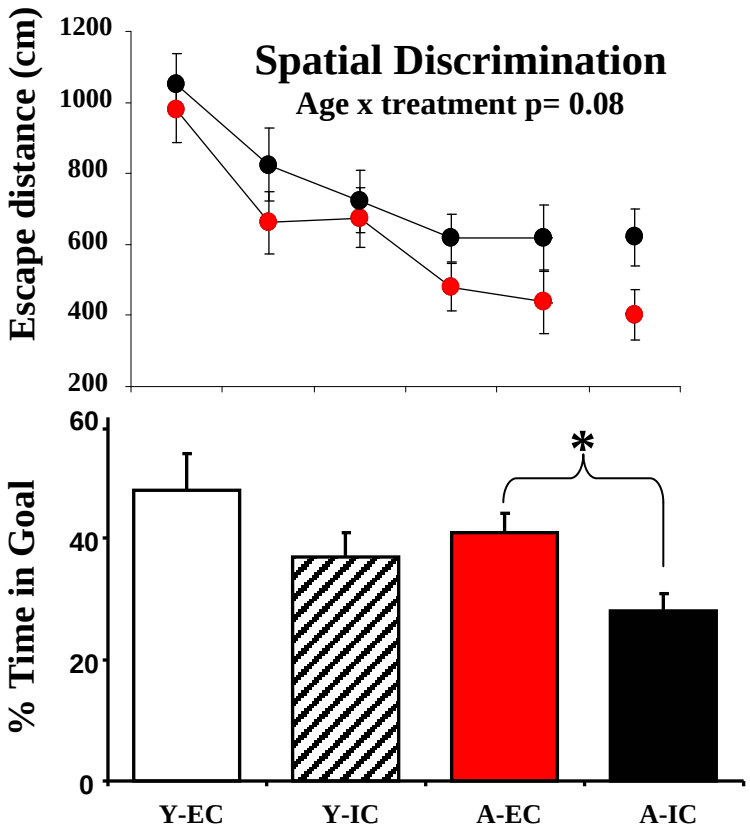




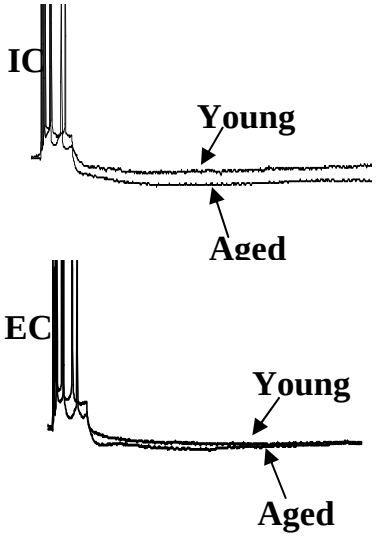


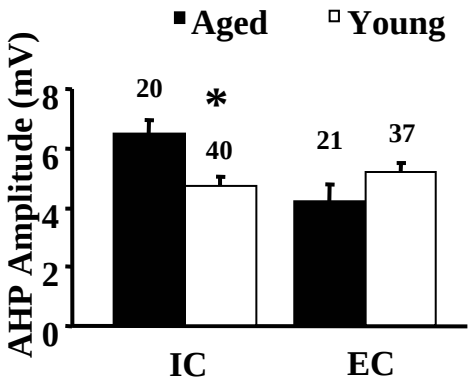
Behavior after 4 wks of enrichment

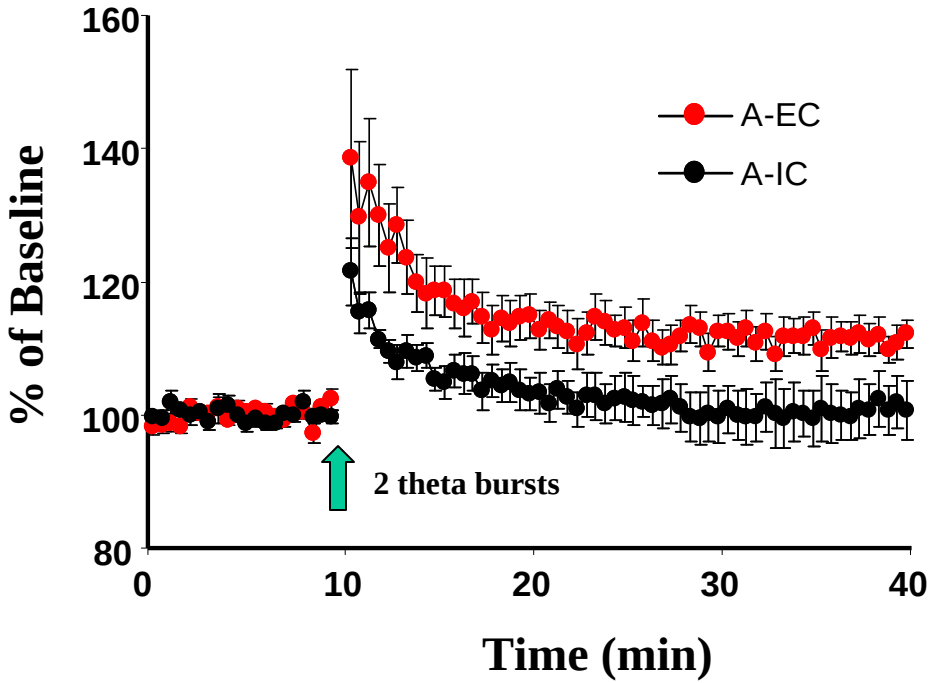




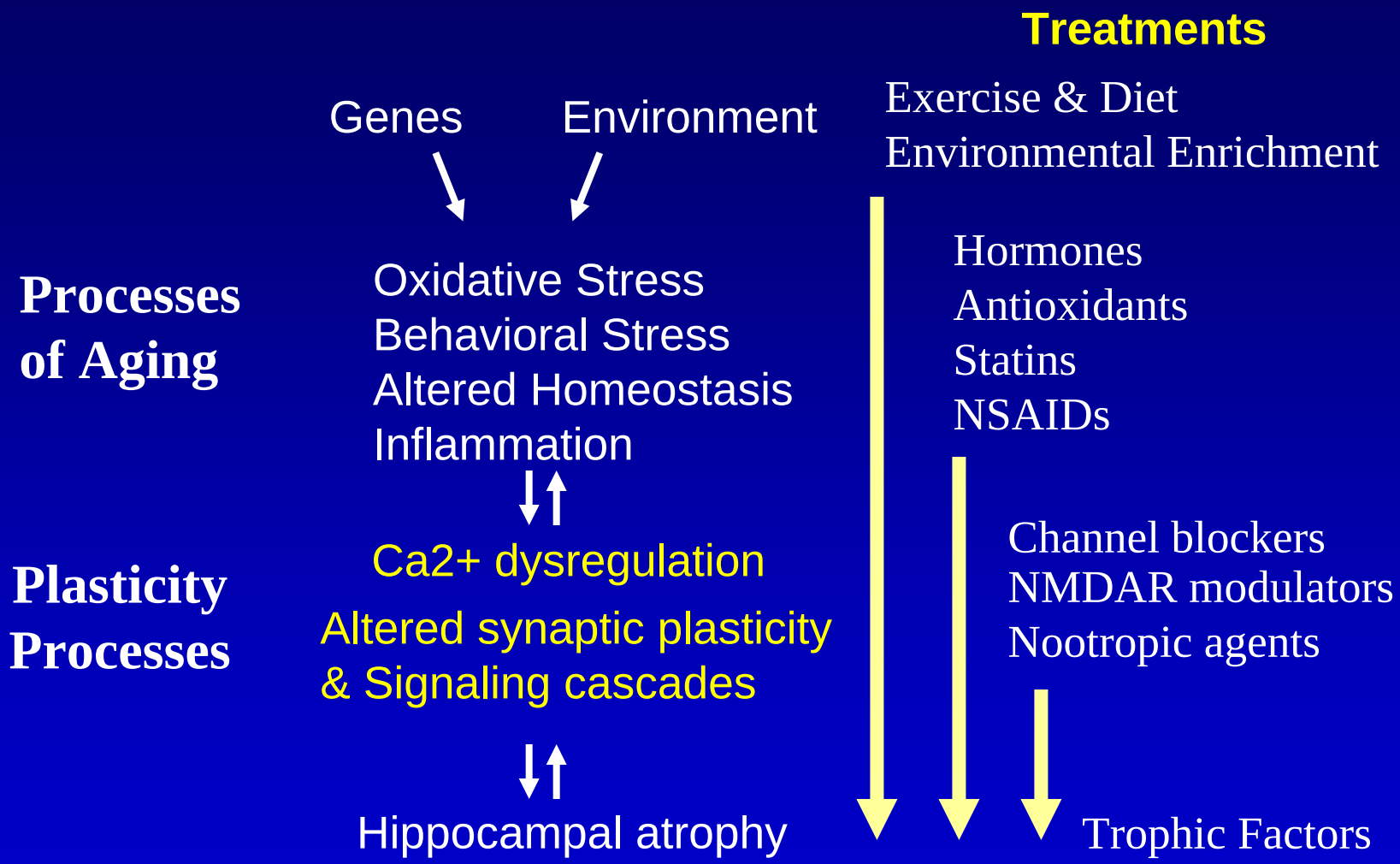
E-phys after more 4 wks of enrichment







Cascade of events leading to cellular and molecular senescence



Which processes of aging underlie senescent physiology?