



Gemma Casadesus Smith, PhD

*Associate Professor of Biological Sciences
Kent State University*

Luteinizing hormone: Evidence for direct action on cognition and neuroplasticity

Age-related changes in reproductive hormone levels are a well-known risk factor for the development of cognitive dysfunction and AD. While the role of gonadal steroids in memory loss has been extensively studied, the contribution of gonadotropins in this process is less known. We and others have shown that downregulation of serum gonadotropin levels rescues cognitive function in AD and menopause models, however, the exact mechanism through which gonadotropins regulate cognitive function and AD pathogenic markers is unknown. Here we show that downregulation of serum gonadotropins, which leads to cognitive improvement and increases in spine density, correlates with increases brain-produced LH. Brain-LH is localized to neurons in cognitive-associated areas and is reduced after ovariectomy in the absence of changes in LHR expression. ICV administration of an LHR agonist improves ovariectomy-related cognitive loss and increases spine density similarly to downregulating peripheral LH. In vitro, our data demonstrates that ligand binding to the LHR in primary neuronal cultures leads to dose-dependent activation of known LHR pathways that are involved in neuroplasticity and result in neurite outgrowth in hippocampal cultures. Taken together these findings demonstrate a novel mechanism through which gonadotropins can alter synaptic plasticity and ultimately regulation of learning and memory.

Date: Thursday, 1 June 2017

Hour: 13:00

Place: Sala de Graus, Facultat de Biologia, Campus Diagonal



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