

Distinguished Lecture Series in Physiology

Samuel Young, Ph.D.

Associate Professor
Department of Anatomy and Cell Biology
University of Iowa

“Elucidating the molecular mechanisms of Presynaptic Cav2 Calcium channel subtype abundance, organization, and how they regulate of neurotransmitter release”

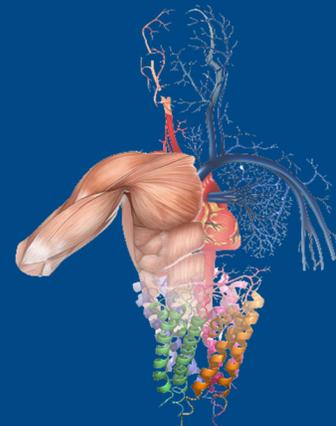
The diversity of information encoding by neuronal circuits is regulated by the magnitude and location of Ca²⁺ entry through voltage-gated Ca²⁺ channels (CaV). In the mammalian central nervous system, the CaV2.1 channel is the critical subtype for CNS function since it is the most efficient CaV2 subtype at triggering synaptic vesicle (SV) release. At the majority of synapses, CaV2.1 is present at higher levels and in closest proximity to SVs. During development synapses become progressively more dependent on CaV2.1 due to selective reduction of CaV2.2 and CaV2.3. Neurons that signal with rapid and temporally precise action-potentials use Cav2.1 exclusive synapses that have fast SV release kinetics. Additionally, CaV2.1 is the dominant CaV2 isoform associated with human CaV2 channelopathies that manifest in migraine, epilepsy, and ataxia. Consistent with these findings, dysregulation of SV release is a cause of these and several other neurological disorders. Despite the importance of CaV2.1 in CNS function, we know little about the molecular mechanisms that regulate these CaV2.1 functions at the synapse. To define these molecular mechanisms, we are using transgenic mouse models and novel viral vectors to manipulate CaV2 subtypes at the calyx during different developmental stages. With these tools and proposed experiments, we are beginning to elucidate how the CaV2 α_1 subunit regulates CaV2 subtype levels, organization and proximity to SVs thereby controlling synaptic transmission and neuronal circuit output. Results of our recent findings will be presented.

Monday, April 4, 2022
GBSF Auditorium and
Zoom 12:00 p.m.

April
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Host: Theanne Griffith
tgriffith@ucdavis.edu