UC DAVIS HEALTH | SCHOOL OF MEDICINE

Department of
Physiology and Membrane Biology

Distinguished Lecture Series in Physiology

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"Dynamic Regulation of Voltage-Gated Ca2+ Channels by Membrane PIP2"

Voltage-gated calcium (CaV) channels serve as critical transducers that convert electrical signals into intracellular chemical signaling, governing diverse physiological processes. However, their activity is not autonomous; it relies heavily on the dynamic membrane environment, particularly phosphatidylinositol 4,5-bisphosphate (PIP2). In this seminar, I will present a comprehensive mechanistic framework describing the dual nature of PIP2 regulation: its constitutive requirement for maintaining channel stability and its hydrolysis-mediated depletion that leads to voltage-independent inhibition.
A key focus of the talk will be the structural interplay between membrane lipids and auxiliary CaV β subunits. We identified a multi-site binding model involving the S4II domain and the intracellular I-II loop of the α1 subunit. Our findings reveal that the subcellular localization of the β subunit acts as a determinant factor. Membrane-anchored β subunits (e.g., β2a) structurally hinder PIP2 access to the I-II loop, reducing sensitivity to lipid depletion while facilitating voltage-dependent Gβγ inhibition. Conversely, cytosolic β subunits leave the I-II loop exposed, rendering the channel highly sensitive to PIP2 depletion. Furthermore, I will discuss the dynamic nature of these interactions, highlighting how specific isoforms reversibly associate with membrane phospholipids. Collectively, these studies propose a model where the CaV β subunit functions as a molecular switch, dictating the dominant signaling pathway, lipid metabolism versus G-protein modulation, to precisely control neuronal excitability.

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GBSF and Zoom
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February
26

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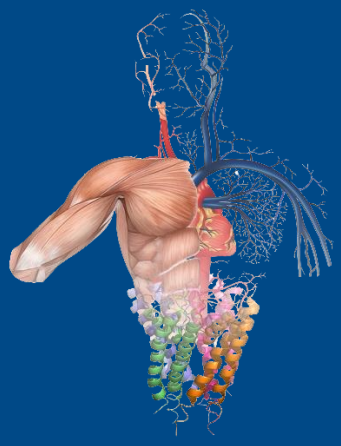

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