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Molecular Underpinnings of Postsynaptic Calmodulin-dependent Calcium Signaling

Abstract:

Calcium (Ca^{2+}) signaling is a dynamic system where Ca^{2+} concentration fluctuates in range of 0.1-10μM with time (4). These short transient Ca^{2+} around the entry sites activate Ca^{2+}-binding proteins such as calmodulin (CaM). The prototypical pathway describes CaM as encoding a Ca^{2+} signal by selectively activating downstream CaM-dependent proteins through molecular binding. However, CaM's intrinsic Ca^{2+}-binding properties alone appear insufficient to decode rapidly fluctuating Ca^{2+} signals. It has been proposed that the temporally varying mechanism for producing target selectivity requires CaM-target interactions that directly tune the Ca^{2+}-binding properties of CaM through reciprocal interactions. In this presentation, I will focus on the binding mechanism of CaM and its target, which requires mutually and conformationally-induced changes in both participants Then, I will focus on two unique and distinct CaM binding targets, neurogranin (Ng) and CaM-dependent kinase II (CaMKII), which are abundant in postsynaptic neuronal cells and are biochemically known to tune CaM's affinity for Ca^{2+} in opposite directions. My group has employed an integrative approach of quantum mechanical calculations, all-atomistic molecular dynamics, and coarse-grained molecular simulations to investigate the molecular mechanisms of CaM's reciprocal interaction between target binding and Ca^{2+}binding. The research of my group has been driven and tested in close collaboration with experimentalists. I will also discuss CaM binding and target selection in the context of evolution and in a crowded environment.

Keck Seminar

Friday, Sept 7, 4pm

BioScience Research Collaborative

Room 280 (2nd Floor)

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