Neuroscience data talk

Transcriptional Control Of Calmodulin By CAMTA Regulates Neural Excitability

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Host: Lora Sweeney

" Calmodulin (CaM) is the major calcium ion (Ca2+) sensor in many biological processes, regulating for example the CaM kinases, calcineurin, and many ion channels. CaM levels are limiting in cells compared to its myriad targets, but how CaM levels are controlled is poorly understood. We find that CaM abundance in the C. elegans and Drosophila nervous systems is controlled by the CaM-binding transcription activator, CAMTA. C. elegans CAMTA (CAMT-1), like its fly and mammalian orthologues, is expressed widely in the nervous system. camt-1 mutants display pleiotropic behavioural defects and altered Ca2+ signaling in neurons. Using FACS-RNA Seq we profile multiple neural types in camt-1 mutants and find all exhibit reduced CaM mRNA compared to controls. Supplementing CaM levels using a transgene rescues camt-1 mutant phenotypes. Chromatin immunoprecipitation sequencing (ChIP-Seq) data show that CAMT-1 binds several sites in the calmodulin promoter. CRISPR-mediated deletion of these sites shows they redundantly regulate calmodulin expression. We also find that CaM can feed back to inhibit its own expression by a mechanism that depends on CaM binding sites on CAMT-1. This work uncovers a mechanism that can both activate and inhibit CaM expression in the nervous system, and controls Ca2+ signalling, neuronal excitability and behavior."

Tuesday, November 3, 2020 01:30pm - 02:00pm
online

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