

Calcium/calmodulin-dependent protein kinase II

Calcium/calmodulin-dependent protein kinases (CaMKs) are key regulators of calcium signaling in health and disease. CaMKII is the most abundant isoform in the heart; although classically described as a regulator of excitation-contraction coupling, recent studies show that it can also mediate inflammation in cardiovascular diseases (CVDs). Among CVDs, cardiorenal syndrome (CRS) represents a pressing issue to be addressed, considering the growing incidence of kidney diseases worldwide.

Choy et al. developed an optogenetic kindling model through repeated [stimulation](#) of ventral hippocampal [CaMKII neurons](#) in [adult rats](#). They then combined [fMRI](#) with [electrophysiology](#) to track brain-wide circuit dynamics resulting from non-afterdischarge (AD)-generating stimulations and individual convulsive [seizures](#). [Kindling](#) induced widespread increases in non-AD-generating stimulation response and ipsilateral [functional connectivity](#) and elevated [anxiety](#). Individual seizures in kindled animals showed more significant increases in brain-wide activity and bilateral functional [connectivity](#). Onset time quantification provided evidence for kindled seizure propagation from the ipsilateral to the contralateral [hemisphere](#). Furthermore, a core of slow-migrating hippocampal activity was identified in both non-kindled and kindled seizures, revealing a novel mechanism of seizure sustainment and propagation ¹⁾.

¹⁾

Choy M, Dadgar-Kiani E, Cron GO, Duffy BA, Schmid F, Edelman BJ, Asaad M, Chan RW, Vahdat S, Lee JH. Repeated [hippocampal seizures](#) lead to brain-wide reorganization of circuits and [seizure](#) propagation pathways. *Neuron*. 2021 Oct 23;S0896-6273(21)00778-9. doi: 10.1016/j.neuron.2021.10.010. Epub ahead of print. PMID: 34706219.

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