Double-Nanodomain Coupling of P/Q-type Ca^{2+} Channels, Ryanodine Receptors, and BK Channels Controls the Generation of Burst Firing

Tomohiko Irie

Div. Pharm., National Institute of Health Sciences

Action potentials clustered into high-frequency bursts play distinct roles in neural computations. However, little is known about ionic currents that control the duration and probability of these bursts. We found that, in cartwheel inhibitory interneurons of the dorsal cochlear nucleus, the likelihood of bursts and the interval between their spikelets were controlled by Ca^{2+} acting across two Ca^{2+} nanodomains, one between plasma membrane P/Q-type Ca^{2+} channels and endoplasmic reticulum (ER) ryanodine receptors, and another between ryanodine receptors and large conductance, voltage- and Ca^{2+}-activated K^{+} (BK) channels. Each spike triggered Ca^{2+}-induced Ca^{2+} release from the ER immediately beneath somatic, but not axonal or dendritic, plasma membrane. Moreover, immunolabeling demonstrated close apposition of ryanodine receptors and BK channels. Double-nanodomain coupling between somatic plasma membrane and hypolemmal ER cisterns provides a unique mechanism for rapid control of action potentials on the millisecond timescale.

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