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## Presentation Abstract

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Presentation Title: Membrane currents contributing to variability of neuronal firing

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**Abstract:** Many neurons are spontaneously active, and synaptic modulation of such activity is thought to be critical for information processing in neuronal networks. Recent work suggests that factors that increase variability of spike timing in spontaneously active neurons disrupt nervous system function. For example, loss of function Ca<sup>2+</sup> channel mutations that cause ataxia have been reported to increase variability of interspike intervals (ISI) in spontaneously firing cells that participate in cerebellar motor control. The mechanisms by which Ca<sup>2+</sup> channels influence firing regularity are unknown. We performed a computational study that includes stochastic channel gating in an effort to understand how channel activity affects the fidelity of spike frequency with an emphasis on Ca<sup>2+</sup> and Ca<sup>2+</sup>-dependent channels. For this purpose, we constructed a single-neuron model that includes fast sodium, delayed rectifier potassium, calcium, calcium-dependent potassium, and persistent sodium channels. Channel gating properties were based on literature values, and channel densities were adjusted to produce spontaneous activity. We found that introducing calcium channels influences firing rate variability, quantified as the coefficient of variation (CV) of the ISI. This effect depends on both Ca<sup>2+</sup> and Ca<sup>2+</sup>-dependent K<sup>+</sup> channels. Our results suggest that this is a possible mechanism by which Ca<sup>2+</sup> channel activity can influence spike rate variability in spontaneous firing neurons.

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CALCIUM CHANNEL

TIMING

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