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Title: Effects of different types of spike frequency adaptation on spike timing and rate coding

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Spike frequency adaptation refers to the reduction of spiking during prolonged stimulation and is typically ascribed to one of two K^+ currents: The voltage-activated M-type K^+ current (I_M) or the calcium-activated K^+ current (I_{AHP}). Using a Morris-Lecar model and bifurcation analysis, we illustrate how the distinct activation properties of I_M and I_{AHP} impact modulation of spiking. Because activation of I_M is sustained at subthreshold voltages, I_M tends to stop the neuron from spiking after a delay proportional to stimulus intensity. In contrast, activation of I_{AHP} is pulsatile, rapidly increasing with each spike but waning between spikes so that spiking is slowed but not stopped. Consequently, effects of I_M and I_{AHP} on a neuron's steady-state frequency-current (*f-I*) curve are fundamentally different: I_M modulates offset whereas I_{AHP} modulates gain. Moreover, I_M can convert neuronal excitability from type 1 to type 2, whereas I_{AHP} does the opposite. Other effects of adaptation are also context-dependent, i.e. vary depending on the intrinsic excitability of the neuron. Because of these differences, the impact of each adaptation mechanism on neural coding is distinct: I_M improves time coding of stimulus fluctuations whereas I_{AHP} improves rate coding of time-averaged stimulus intensity - in that regard, I_M and I_{AHP} encourage the neuron to behave as either a coincidence detector or integrator, respectively. Thus, spiking is modulated in very different (potentially opposite) ways depending on the biophysical mechanism underlying adaptation and the context in which that adaptation occurs, which in turn has important consequences for neuronal coding properties. Spike frequency adaptation is therefore not a generic process; on the contrary, predicting the effects of adaptation requires identification of the underlying mechanism.

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