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What makes DA neurons tick? Role of Ca2+ sources and inward currents in setting the regular pacemaking of dopaminergic neurons

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Dopamine (DA) neurons show regular, pacemaking firing in the range 1-8 Hz in *in vitro* preparations. The currents involved in this pacemaking behavior are not fully understood. The slow oscillatory potential (SOP), evident after the application of TTX, is known to depend on the mutual interaction between L-type Ca2+ and small conductance (SK) K+ currents. The SOP oscillates in a frequency range similar to the pacemaking firing rate and it is assumed to be responsible for determining this pacemaking frequency [1]. However, recent results indicate that the SOP's frequency is not correlated with spontaneous pacemaking frequency [2,3]. Furthermore there is experimental evidence indicating that pacemaking does not require the influx of Ca2+ through L-type Ca2+ channels [4,5]. Figure 1A illustrates this last point from a modeling approach. The black trace corresponds to the spontaneous firing of the model DA neuron and in blue the SOP. After simulated block of L-type current no activity is observed (green) but this is recovered (red) by injecting a virtual L-type current into the soma (cf. [5]). Figure 1B on the other hand illustrates the possible role of the fast Na+ current in reestablishing pacemaking. Black and blue traces are similar to those in panel A; however pacemaking is reestablished by shifting the Na+ activation curve in a hyperpolarization direction (green). We explore the role

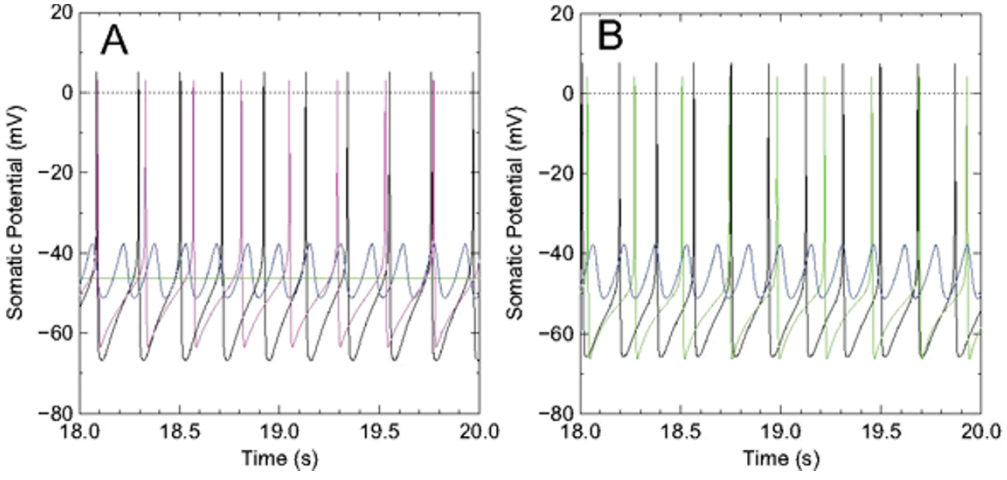

Figure 1 Pacemaking in model DA neuron in the absence of Ca2+ entry through L-type Ca2+ channels.

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of additional inward and outward currents, particularly focusing on other Ca2+ currents (e.g. T, N), also the release of Ca2+ from intracellular stores in establishing pacemaking.

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