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Effects of Tachykinin Receptor Activation on Prefrontal Cortex Neuronal Activity

Tachykinins are neuromodulatory peptides found in the mammalian brain and high levels of tachykinin receptors are expressed on pyramidal neurons of the prefrontal cortex (PFC). The present study used the whole cell patch clamp technique and a mathematical model to investigate the ionic mechanisms underlying the responses of guinea pig PFC neurons to senktide, a tachykinin receptor agonist. Senktide (500nM) depolarized neurons, and at -70 mV in voltage-clamp, induced an inward shift of the holding current, a change that was accompanied by a decrease in membrane conductance. Current-voltage (IV) relationships showed that the senktide-sensitive current reversed at -91.6 mV, very near the calculated potassium equilibrium potential (E_K) of -93 mV. Elevations of extracellular potassium shifted this reversal potential in accordance with the Nernst equation. The response to senktide was similar to the response seen with extracellular cesium (5mM) or barium (200µM), known inhibitors of the inward rectifier potassium current (I_{Kir}). Simulations using a biophysically-based neuronal model consisting of twelve differential equations showed that decreased I_{Kir} conductance yielded responses consistent with our biological data. We thus conclude that tachykinin receptor activation on PFC neurons may result in inhibition of the potassium current, I_{Kir}.