Location and function of SK channels in pyramidal neurons in the amygdala

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SK channels are small conductance calcium activated potassium channels. In neurons these channels have traditionally been found to be activated by calcium influx during action potentials and their activation contributes to the control of neuronal firing frequency. Calcium influx during action potentials activated a number of calcium dependent potasisum currents that contribute to action potential repolarisation and the afterhyperpolarisation (AHP) that follows that action potential. SK channels contribute only to the AHP, however, bockade of these channels in amygdala neurons has little effect on neuronal firing properties. We have recently shown that these channels are also expressed at excitatory synapses in lateral amygdala pyramidal neurons where they are activated by calcium influx *via* NMDA receptors. Activation of SK channels at excitatory synapses shunts the synaptic potential and thus reduces synaptic transmission. Blockade of these SK channels with specific blockers reduces the shunt and enhances synaptic transmission and summation of excitatory synaptic potentials. This results in an enhanced ability to induce long term potentiation. Application of neurotransmitters that activate protein kinase A reduced the number of synaptic SK channels and enhanced synaptic transmission and plasticity. These actions of neurotransmitters are mediated by altering the endogenous trafficking of SK channels and their delivery to excitatory synapses. These results show that SK channels, rather than controlling action potential frequency, as in some other cell types, play a key role in controlling the strength of synaptic transmission and contribute to the control of synaptic plasticity.