SK channels modulate NMDA receptor-mediated synaptic transmission in the lateral amygdala

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Small conductance calcium activated potassium (SK) channels have classically been thought to play a role in controlling the firing patterns in neurons. However in pyramidal neurons in the lateral amygdala (LA), a limbic region that attributes emotional value to sensory input, SK channels do not control firing patterns under physiological conditions. Thus we aimed to investigate the role of SK channels in the LA. Visualised whole cell recordings were made from 400 μm coronal brain slices obtained from 3-4 week old Wistar rats (anaesthetized with halothane). Recordings were made using a potassium based internal solution and the slices were perfused in oxygenated aCSF at 30°C. EPSPs were evoked by stimulating cortical afferents in the external capsule with a bipolar stimulating electrode in the presence of the GABAergic blockers picrotoxin and CGP55845A. Blockade of SK channels by apamin significantly enhanced the EPSP amplitude by between 20-50%. This effect was reversed by application of AP5, a selective blocker of NMDA receptors, or was prevented by pre-treatment with AP5. Perfusion of slices with low extracellular Mg enhanced the effect of apamin, with apamin potentiating the EPSP by 97 ± 24%. Loading cells with 10 mM BAPTA or a Cs based internal solution blocked the effect of apamin. Finally blockade of SK channels enhanced LTP at cortical inputs to pyramidal neurons, from 124 ± 10% to 202 ± 22%. These findings show that calcium influx through NMDA receptors activates SK channels, which shunt excitatory synaptic transmission, acting as a brake on both synaptic transmission and plasticity in pyramidal neurons in the LA.