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Postsynaptic Group II Metabotropic Glutamate Receptor activation in Dentate Gyrus Granule Cells

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Dentate gyrus granule cells (DGGCs) express group II metabotropic glutamate receptors (mGluR2/3) both on their axonal and somatodendritic membranes. Activation of axonal mGluR2/3s suppress the synaptic transmission from DGGCs due to down regulation of presynaptic Ca2+ channel activity. However, the function of dendritic mGluR2/3s in DGGCs is not known. First, we showed that bath application of mGluR2/3 selective agonists (DCG IV, APDC) resulted in significant, long-lasting hyperpolarization in DGGCs and this effect persisted in the presence of TTX but it was prevented by selective antagonists (APICA, LY 341495). DCG IV did not have any effect on the axonal membrane potential of DGGCs indicating distinct somatic and axonal mGluR2/3 mediated mechanisms. The hyperpolarizing DCG IV effect was absent in GABAergic and CA3 pyramidal cells. We also showed that the mGluR2/3 activation induced somatodendritic hyperpolarization was mediated by GIRK channels as intracellular GDPβS and bath application of inward-rectifier potassium channel blocker, tertiapin-Q occluded the DCG IV effect. Lastly, focal glutamate release by photolysis of MNI-glutamate in the perisomatic region of DGGCs resulted in mGluR2/3 mediated currents; however, similar current could be evoked in the dendritic region (>15 μm) suggesting that mGluR2/3 are activated by a specific glutamatergic source which innervates the perisomatic region of DGGCs and elicit mGluR2/3 mediated glutamatergic inhibition.