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PDE2, a component of the NO/cGMP signalling in the hippocampus

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Background

NO/cGMP-mediated signal transduction is involved in synaptic plasticity in various brain regions. NO effects are transduced by the NO receptor guanylyl cyclase (NO-GC) that exists in two isoforms, NO-GC1 and NO-GC2, with indistinguishable regulatory properties. Mice deficient in either NO-GC1 or NO-GC2 revealed that both NO-GC isoforms are required for LTP indicating the existence of two separated NO/cGMP pathways. Recently, we demonstrated a presynaptic role of NO/cGMP in facilitation of glutamate release and indentified eNOS and NO-GC1 as the participating enzymes. Yet, the involved cGMP-hydrolysing phosphodiesterases (PDE) remained unknown.

Results

Here we demonstrate that PDE2 accounts for 50% of cGMP-hydrolysing activity in hippocampal homogenates. In hippocampal slices of WT, NO-GC1 and NO-GC2 KO mice, PDE2 inhibition increased NMDA-induced cGMP levels.

Conclusion

This suggests PDE2 as a component of both NO-GC1- and NO-GC2-mediated signalling pathways. Moreover we analyzed the physiological role of the PDE2 on glutamatergic transmission in the hippocampal CA1 region by single-cell recordings in acute slices.

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