D113 Distribution of Cholonergic Neurons in Newt Retina, and the Onset and Maturation of Cholonergic System during Retinal Development and Regeneration

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We have investigated the onset of appearance of cholinergic system including acetylcholinesterase(AChE), choline acetyltransferase(ChAT) and acetylcholine receptors(AChR) during retinal development and regeneration using immunocytochemical and histochemical methods. ChAT-ir and AChE activity were observed in the amacrine cells in the inner nuclear layer(INL) and displaced amacrine cells in the ganglion cell layer(GCL). Muscarinic-AChR immunoreactivity was observed in a large number of ganglion cells, and a few somata in the INL. mAChR-ir and AChE activity appeared first in the most proximal region of both developing and regenerating retinas before segregation of synaptic layer, while ChAT-ir first appeared at the time of the synapse formation. These results suggest that mAChRs appear earlier than ACh synthesis, and that the onset of appearance of cholinergic system in regenerating retina is identical to that in the developing retina.

D114 Cadherin-Catenin Mediation of TPA-Induced Neurite Outgrowth in GT1-1 Cells

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12-O-tetradecanoylphorbol-13-acetate (TPA), a protein kinase C (PKC) activator, stimulates morphological and functional differentiation in GnRH-producing GT1-1 neuronal cells. Differential display PCR identified β-catenin as a gene putatively involved in such differentiation process. Since β-catenin has recently been implied in the synaptogenic process, we probed into the role of β-catenin in GT1-1 neuritogenesis. While Northern blot analysis revealed a slight induction of β-catenin mRNA levels, Western blot analysis revealed that the protein levels of β-catenin dramatically increased within 1 hr of TPA treatment. Simultaneously, trans-activational properties of β-catenin were enhanced. Although β-catenin and N-cadherin complexes were specifically co-localized at cell-cell adhesion sites in GT1-1 cells, these complexes reallocated to growth cones after TPA treatment. In light of the PKC mediation of the Wnt signal during brain development, we propose an active role for β-catenin in TPA-induced neuritogenesis of GT1-1 cells.