tral portion of the growth cone. Phosphorylation of synapsin I by cAMP-dependent protein kinase (PKA) causes the dissociation of the protein from the SV membrane, allowing diffusion of the vesicles to the periphery of the growth cone and enhancing their rate of recycling. A similar mechanism also takes place at mature synapses, where cAMP-induced phosphorylation of synapsin I largely accounts for the modulatory effects of PKA in the potentiation of SV exocytosis.

These results indicate that molecular mechanisms similar to those operating at mature nerve terminals are active in developing neurons to regulate the SV life cycle prior to synaptogenesis. Moreover, the cAMP/synapsin pathway may underlie the synaptogenetic effect of synapsin by triggering the structural rearrangements which lead to the formation of a mature secretory compartment.

Keywords: Growth cone; Neurotransmitter release; Synapse formation; Lentiviruses

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[P61]

Presynaptic TrkB signaling mediates axon arbor growth and synapse maturation during the establishment of retinotectal synaptic connectivity

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Neurotrophins have been shown to exert multiple influences during the development of visual connectivity, from guiding the morphological differentiation of neurons to controlling the functional plasticity of visual circuits. In *Xenopus*, BDNF applications in the optic tectum influence the dynamic branching of retinal ganglion cell (RGC) axons and increase synapse density per axon terminal by rapidly promoting synapse formation and synapse stabilization. Thus, BDNF shapes retinotectal synaptic connectivity most likely by acting directly on presynaptic RGCs. Here, we combined *in vivo* imaging of fluorescently tagged synaptic specializations with expression of a dominant negative TrkB-EGFP fusion protein to differentiate between pre- and postsynaptic actions of BDNF. Disruption of TrkB signaling in individual RGCs influenced the branching and synaptic maturation of presynaptic axon arbors. Specifically, TrkB.T1-EGFP overexpression increased the proportion of axons with immature, growth cone-like morphology, decreased axon branch stability and increased axon arbor degeneration. In addition, TrkB.T1-EGFP overexpression reduced the number and stability of RFP-synaptobrevin labeled presynaptic specializations per axon terminal. In contrast, overexpression of TrkB.T1-EGFP in postsynaptic tectal neurons did not alter the morphology or dynamic behavior of their dendritic arbors, although manipulations in BDNF tectal levels elicited significant changes in postsynaptic specialization number in tectal neurons with intact TrkB signaling. Electron microscopy analysis of TrkB.T1-EGFP expressing RGC axons provided a direct correlate between changes in synaptic ultrastructure and the dynamic behavior of synaptic specializations in axon arbors observed *in vivo*. A decrease in the number of mature synaptic profiles and a significant decrease in docked synaptic vesicles at mature synapses were observed in RGC axons immunopositive for TrkB.T1-EGFP. Together, our results demonstrate that presynaptic TrkB signaling in RGCs is a key determinant in the establishment of visual connectivity and indicate that changes in tectal neuron synaptic connectivity are secondary to the BDNF-elicited enhanced stability and growth of presynaptic RGCs.

Keywords: In vivo imaging; Xenopus; Visual system; Retinal ganglion cells

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[P62]

Ca^{2+}-permeable AMPA/kainate channel expression in the somatosensory cortex of rat from peri-natal to adult stages

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There is presently limited understanding about the physiological roles of Ca^{2+}-permeable AMPA/kainite (Ca-A/K) channels in the brain. Studies have indicated that Ca-A/K channels likely play roles in brain development, synaptic plasticity, and neurotoxicity. In order to get more insights of the physiological roles of Ca-A/K channels, we want first to know the temporal change of the expression of the Ca-A/K channels during the course of development in the somatosensory cortex from perinatal to adult stages.

Acute cortical slices were dissected from rat fetus at embryonic (E) days 18 and 20 and rat pups at postnatal (P) days 1, 5, 13, 21 and adult. By using the kainite-stimulated Co^{2+} loading method, the neurons possessing Ca-A/K channels can be labeled as dark-brown color; referred to Co^{2+}-positive neurons here. In E18, only 25.9% of neurons are Co^{2+}-positive and enriched in the marginal zone (MZ) as well as in subplate (SP). They have small, smooth oval shaped soma and horizontal processes. From E18 to P5, the number of Co^{2+}-positive cell gradually increases in the cortical plate (CP) and decreases in the SP. Treating E18 slices with 250 μM glutamate for 30 min does not induce any detectable damage to the SP neurons. After P21, 73.1% of neurons are Co^{2+}-positive and distribute all over the 6 layers of cortex. Their soma size become bigger, and their morphology becomes complex. Most of their processes orient vertically. In adult somatosensory cortex, as identified by glutamate decarboxylase and NeuN immunoreactivity, 81.3% of inhibitory neurons are Co^{2+}-positive with an oval shape and enriched in layer II, III–IV border and V–VI, and 51.4% of excitatory neurons are Co^{2+}-positive with a pyramidal shape, enriching in layer II–III and V–VI.

In conclusion, the disappearance of SP neurons during development is unlikely due to the cell degeneration mediated by the