BDNF-mediated modulation of GABA and glycine release in dorsal horn lamina II from postnatal rats

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Recent studies show that excitatory glutamatergic transmission is potentiated by BDNF in superficial dorsal horn, both at the pre- and the postsynaptic site. The role of BDNF in modulating GABA and glycine-mediated inhibitory transmission has not been fully investigated. To determine whether the neurotrophin is effective in regulating the spontaneous release of the two neurotransmitters, we have recorded miniature inhibitory postsynaptic currents (mIPSCs) in lamina II of post-natal rats. We show that application of BDNF enhanced the spontaneous release of GABA and glycine, in presence of tetrodotoxin. The effect was blocked by the trk-receptor inhibitor k-252a. Amplitude and kinetics of mIPSCs were not altered. Evoked GABA and glycine IPSCs (eIPSCs) were depressed by BDNF and the coefficient of variation of eIPSC amplitude was significantly increased. By recording glycine eIPSCs with the paired-pulse protocol, an increase of paired-pulse ratio during BDNF application was observed. We performed parallel ultrastructural studies to unveil the circuitry involved in the effects of BDNF. These studies show that synaptic interactions between full length functional trkB receptors and GABA-containing profiles only occur at not-peptidergic synaptic glomeruli of types I and II. Expression of trkB in presynaptic vesiclecontaining dendrites originating from GABAergic islet cells, indicate these profiles as key structures in the modulation of inhibitory neurotransmission by the neurotrophin. Our results thus describe a yet uncharacterized effect of BDNF in lamina II, giving further strength to the notion that the neurotrophin plays an important role in pain neurotransmission.