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ProBDNF inhibits proliferation, migration and differentiation of mouse neural stem cells

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ProBDNF, a precursor of Brain-Derived Neuro-trophic Factor (BDNF), is an important regulator of neuro-degeneration, hippocampal long-term depression and synaptic plasticity. ProBDNF and its receptors Pan-Neurotrophin Receptor p75 (p75NTR), vps10p domain-containing receptor Sortilin and Tropomyosin receptor kinase B (TrkB) are expressed in neuronal and glial cells. The role of proBDNF in regulation of neurogenesis is not fully defined. This study aims to uncover the function of proBDNF in regulating the differentiation, migration and proliferation of mouse Neural Stem Cells (NSCs) *in vitro*. We have found that proBDNF and its receptors are constitutively expressed in NSCs when assessed by immunocytochemistry and western blotting. MTT (3-[4,5-dimethylthiazol-2-yl]-2,5 diphenyl tetrazolium bromide) assay showed that exogenous proBDNF treatment reduced mouse NSCs viability by 38% at 10 ng/mL. The migration of NSCs was also reduced by exogenous proBDNF treatment in a concentration-dependent manner (by 90% at 10 ng/mL) but increased by anti-proBDNF antibody treatment (by 50%). BrdU (5-Bromo-2′-Deoxyuridine) incorporation was performed for detection of newborn cells. We have found that proBDNF significantly inhibited proliferation of NSCs and reduced the number of differentiated neurons, oligodendrocytes and astrocytes, while anti-proBDNF antibody treatment promoted proliferation and differentiation of NSCs. In conclusion, proBDNF may oppose the functions of mature BDNF by inhibiting the proliferation, differentiation and migration of NSCs during development. Conversely, anti-proBDNF antibody treatment promoted proliferation and differentiation of NSC.

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