Commentary

Stem Cell Research and Regenerative Medicine

BDNF Promotes EGF-Induced Proliferation and Migration of Human Fetal Neural Stem/Progenitor Cells via the PI3K

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Abstract

Neurogenesis is a complex process, which contributes to the capability of the adult brain to serve typically and acclimatize to conditions. Epidermal growth factor (EGF) is known to play an important part in neurogenesis; still, the beginning medium is still unclear. Then, we hypothecated that brain- deduced neurotrophic factor (BDNF) can enhance the effect of EGF on neurogenesis. Using in vitro cell culture of aborted mortal fetal brain apkins, we delved proliferation and migration of neural stem/ ancestor cells (NSPCs) after treatment with EGF and different attention of BDNF. EGF stimulated proliferation and migration of NSPCs, and this effect was significantly enhanced byco-incubation with BDNF. In the NSPCs treated with 50 ng/ mL BDNF, BrdU objectification was significantly increased (from7.91 to17.07), as compared with that in the control. Also, the number of migrating cells was at least2-fold advanced than that in the control

Keywords: neural stem• ancestor cells• epidermal growth factor• brain- deduced neurotrophic factor• proliferation; migration preface

Introduction

Neurogenesis is a complex experimental process involving proliferation, isolation, migration, survival, development and functional integration of neural stem/ ancestor cells (NSPCs) into neuronal circuits. Adult neurogenesis in advanced organisms, including primates and humans, is largely confined to two regions the sub ventricular zone (SVZ) of the side ventricles and the sub granular zone (SGZ) of the hippocampus. The process of neurogenesis involves a dynamic nonsupervisory network of different natural motes, including hormones, recap factors, and neurotransmitters. Growing substantiation has indicated that growth factors, which are important for regulating a variety of cellular processes, may also play an important part in neurogenesis [1]. Epidermal growth factor (EGF) regulates cell growth by stimulating proliferation and migration of different types of cells. In the central nervous system (CNS), EGF mRNA has been detected in numerous regions, including the brainstem, cerebellum, cerebral cortex, hippocampus, olfactory bulb, and striatum. The loftiest situations, still, were set up in the olfactory bulb, rudimentary hypothalamus and cerebellum. Intra cerebral infusion of EGF redounded in a dramatic proliferation of endogenous SVZ precursor cells. EGF has also been demonstrated to increase the number of invigorated cells in the striatum either by stimulating migration of SVZ cells or by promoting proliferation of original ancestor cells [2]. Also, EGF has been shown to stimulate the migration and proliferation of murine ancestor cells in vivo after transplantation to the adult rat brain. Inclusively, substantiation has demonstrated that EGF provides important extracellular signals during development of CNS. Still , EGF and its associated signaling pathways during mortal neurogenesis remain unclear [3].

Results

Mortal NSPCs were insulated from the brain apkins of aborted mortal fetus. Following sowing into growth medium, we set up that summations of dividing cells formed into

neuro spheres. After seven days of incubation, individual neuro spheres were sectioned into 4 pieces using a fine glass needle and were independently transferred into fresh growth medium containing bFGF2 and EGF. The pieces formed new neurospheres within the coming 24 hours, ultimately reaching sizes near to that of the primary neuro spheres. When the Neuro spheres could be passaged stably over three passages, we began to passage the neuro spheres using Triple TM Express for digestion. Using these styles, we efficiently attained mortal NSPCs [4]. Results of immuno cytochemical staining showed these neurospheres were positive for nestin.

Discussion

In the present study, we've demonstrated the direct effect of BDNF on EGF- convinced proliferation and migration of NSPCs using an in vitro culture system. We insulated and dressed mortal fetal brain NSPCs and showed that 50 ng/ mL of BDNF produced the minimal effect on EGF- convinced NSPC proliferation and migration likewise, we've demonstrated that BDNF increased the phosphorylation of Akt- 1, a downstream target of PI3K still, this enhanced effect was abolished when NSPCs were pretreated with the PI3K asset LY294002. These results indicated that BDNF is involved in EGF- convinced proliferation and migration during neurogenesis via regulation of the PI3K/ Akt pathway. It has been shown that adult brain injuries similar as trauma and ischemia induce neurogenesis, furnishing stopgap for functional recovery after a CNS personality. Although postnatal neurogenesis can do, its capacity is vastly limited and rejuvenescence of new neurons typically occurs only in two regions of the adult brain, videlicet the hippocampus and olfactory bulbs. Therefore, transplantation of NSPCs at spots of brain injury may be a better way to promote functional recovery. On the other hand, growth factors needed for neuronal proliferation and isolation could be used in combination with NSPCs transplantation as a implicit treatment for neurodegenerative diseases or brain damage. In our study, we detected that BDNF enhanced EGF- convinced proliferation and migration rate of NSPCs. 50 ng/ mL of BDNF inspired the minimal effect, which wasn't affected by apoptosis rate. Still, compare to 50 ng/ mL of BDNF, a farther increase in the attention of BDNF(100 ng/ mL) convinced slightly lower proliferation and migration, which may be due to the effect of enhancing isolation of neuronal precursors. Farther studies should be done to confirm this enterprise. The result indicated that a certain range of attention BDNF enhances EGF-convinced cell proliferation and migration; beyond the range affect reduced or have contrary effect. Treated with applicable cure of BDNF may significantly ameliorate the effect of EGF-dependent NSPCs to control neurodegenerative conditions or injury [5].

Conclusions

Overall, we've handed substantiation that EGF can stimulate proliferation and migration of mortal NSPCs. Its regulation may be affected by BDNF via the PI3K/ Akt kinase pathway; therefore, both BDNF and EGF may functionally modulate the nonsupervisory network of neurogenesis. In addition, our results are applicable for defining the ideal culture medium for neural cells expansion in vitro. These results will contribute to our understanding of mechanisms underpinning growth factors in the development of NSPCs during neurogenesis, and will probably be helpful for development of remedial operations using NSPCs to ameliorate lifetime and integration ofpre-treated cells after transplantation [6].

Acknowledgement

None

Conflict of Interest

No conflict of interest

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