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Neuro trophic factor reduces inflammation and improve brain neuronal regeneration in inflammatory brain injury

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The risk of suffering from serious permanent sequelae following a central nerve system (CNS) infection is high and varies in different studies from 30 to 60%, to some extent depending on the causative agent. Inflammation is emerging as a critical mechanism underlying neurological disorders of various etiologies, yet its role in altering brain function as a consequence of neuro infectious disease remains unclear. The main limitation to advance in prevention and treatment of the disease is incomplete knowledge of its pathogenesis and pathophysiology. There is now solid evidence that intense inflammatory host response causes important damage to the brain, thus inducing unfavorable outcomes of meningitis. Neuro trophic factor family(nerve growth factor, NGF, brain derived neuro trophic factor, BDNF, et al) plays an important role in the development, differentiation, and survival of neurons in the CNS. Dysfunction in the regulation of BDNF is associated with numerous disorders of CNS, including Alzheimer's disease (AD), multiple sclerosis (MS), depression, and unacceptable outcomes of bacterial meningitis. Our previous study showed that increased expression of BDNF in the acute S. pneumonia meningitis was abviously alleviated after antibiotic treatment. Similarly, increased BDNF levels were also observed in the serum and cerebrospinal fluid (CSF) of pediatric patients with CNS infections on the day of admission. Furthermore, Barichello et al. reported that decreases in BDNF levels during the long-term phase of meningitis were correlated with behavioral deficits in adult animals submitted to meningitis during the neonatal period. Interestingly, our previous study reported that administration of exogenous BDNF increased neuron survival in both the cortex and hippocampus, and reversed brain damage, and it was recently reported that exogenous BDNF increases neurogenesis of neuron stem cells in the hippocampus after S pneumonia meningitis. These findings indicate that regulatory expression of BDNF may be a part of the host inflammatory response in S. pneumonia meningitis, and the innate immune response could be a double-edged blade possessing both protective and damaging properties, and there could be interaction between them. However, the underlying regulatory mechanism is still not clear. We investigated the effects of BDNF-related signaling on the inflammatory response and hippocampal apoptosis in experimental models of pneumococcal meningitis. Pretreatment with exogenous BDNF or the tropo myosin-receptor kinase B (TrkB) inhibitor k252a was performed to assess the activation or inhibition of the BDNF/TrkB-signaling axis prior to intracisternal infection with live S. pneumonia. The results showed Rats administered BDNF exhibited reduced clinical impairment, pathological severity, and hippocampal apoptosis.

Biography

Ling Li is Presently working as a Doctor in Xinhua Hospital-Shanghai Jiao Tong University School of Medicine, China. His international experience includes various programs, contributions and participation in different countries for diverse fields of study. His research interests reflect in his wide range of publications in various national and international journals.

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