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Poster Presentation

The Effects of Boswellia Serrate on Central Nervous System

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Abstract

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In the process of neuronal inflammation, an increased in inflammatory cytokines (IL-1 β , IL-6 and TNF- α) from immune cells (leukocytes and macrophages), brain cells (microglia, astrocytes and neurons) and in hippocampus, amygdala occurs. Raise the level of cytokines result in reduced in production of molecules that are related to plasticity, especially BDNF, IGF-1 and VEGF. Microglia activation lead to suppression of neurogenesis, differentiation of NPCs, decrease in long-term potentiation (LTP) and induction of learning and memory impairment. Also, the phenomenon of nerve inflammation with an increase in the level of TNF α cause inhibition of astrocytes in the removal of glutamate and led to neuronal death. Elevated in TNF α level result in increase dativity of the iNOS enzyme that is available in astrocytes in the CA1 hippocampus and it is responsible for increase in oxidant molecules and depression of LTP. Furthermore, the increase in inflammatory reaction mediators result in subsequent neurotoxic consequences. Indeed, inflammatory factors deliberate as a predisposing agent for neurodegenerative disease. Boswellia serrate from Burseracea family, it's resin (Frankincense or Ollibanum) and the main constituent of this resin (boswellic acid) play important role in suppression of neuronal inflammation with inhibition of 5-lipoxygenase, Prostaglandin E2 formation and expression of inflammatory cytokines and chemokines. Boswellia serrate and its derivatives with anti-inflammatory properties have therapeutic effects on memory retention, decrease in brain edema, facilitation in nerve impulse, improve the pathogenesis of neuroinflammatory disease like Alzheimer's disease.

Keywords: Neuroinflammation, Boswellia Serrate, Frankincense, Ollibanum, Boswellic Acid

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