

عنوان مقاله:

Chitosan and its Anti-Inflammatory Role in CNS Damage

محل انتشار:

سومین همایش بین المللی التهاب سیستم عصبی و سومین فستیوال دانشجویی علوم اعصاب (سال: 1398)

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خلاصه مقاله:

Strategies and mechanisms used in order to protect apoptosis, against neuronal damage, degeneration and/ or dysfunction in the central nervous system (CNS) are called as neuroprotection. The target of neuroprotection is to bound neuronal death or dysfunction after CNS damage in a try to maintain the highest possible honesty of cellular interactions in the brain, thus minimizing disorder to neural role. According to its mechanism, neuroprotection can be classification into some mechanisms such as: anti-inflammatory, antioxidant (scavenger/free radical trapper), apoptosis inhibitor, antiexcitotoxic, etc. Chitosan is a linear polysaccharide that consists of β -(1 $\rightarrow$ 4)-2-amino-d-glucose and β -(1 $\rightarrow$ 4)- 2-acetamido-d-glucose units derived from partial deacetylation of chitin. Both Chito oligosaccharides (COS) and chitosan are known to possess a lot of biological activities such as anti-inflammatory activities, antioxidant, antibacterial, immunoenhancing, tc. The first response of a body's immune system to pathogens or irritation is inflammation. Some studies are discovering blinding communications between anumber of neurodegenerative disorders and chronic inflammation. The neuroinflammation process plays a substantial activity in the progression and initiation of different neurodegenerative diseases. Recently, several studies have found anti-neuroinflammatory role of chitosan and its derivatives. One study reported that high molecular weight water soluble chitosan (WSC) inhibits the production of pro-inflammatory cytokine in human astrocytoma cells activated by interleukin-1 β (IL-1 β) and A β peptide 25–35 (A β 25–35). Another study focused on the anti-neuroinflammatory effect of chitosan and its derivatives on NT2 neuronal cells. Chitosan employs anti-neuroinflammatory role by inhibits the activation of NF- κ B and upregulation of heat shock protein 70 (Hsp-70). The anti-inflammatory mechanism of Hsp-70 is mediated by the binding of Hsp-70 to NF- κ B and its following inhibition. A pre-treatment with 0.1 and 0.5%(w/v) chitosan prior to FeSO₄ and H₂O₂ .exposure has been proved to increase the level of heat shock protein (Hsp-70) to 1.4- and 1.6-times, respectively

کلمات کلیدی:

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