



POSTER

Interaction of thymoquinone with alpha 7 nicotinic acetylcholine receptor in LPS-induced neuroinflammatory model

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Abstract

Alzheimer's disease (AD) is neurodegenerative disorder resulting from loss of cholinergic neurons in brain especially acetylcholine. It has been reported that $\alpha 7$ nicotinic acetylcholine receptors ($\alpha 7$ nAChRs) play an important role in cognitive function and can be target therapy for treating cognitive deficits. $\alpha 7$ nAChR agonists cause memory enhancement through phosphorylation of cAMP response element binding protein (CREB). Thymoquinone (TQ) was recently considered as acetylcholine esterase inhibitor and increased $\alpha 7$ nAChR expression in brain. However, the effect of TQ as $\alpha 7$ agonist has not been investigated. Our aim was to investigate the mechanism of action of TQ on $\alpha 7$ nAChR.

Neuroinflammatory AD rat model was developed by injecting LPS i.p (0.8 mg/kg) once. A specific $\alpha 7$ agonist and $\alpha 7$ positive allosteric modulator were used. Rats were injected

with TQ (10 mg/kg) i.p for 5 consecutive days with or without $\alpha 7$ positive allosteric and another group with $\alpha 7$ agonist + $\alpha 7$ positive allosteric modulator. After one week, rat brains were subjected to immunohistochemical studies. Molecular docking studies were done in which TQ was docked on chimeric acetylcholine binding protein.

Results indicate significant decrease in amyloid plaques with significant increase in p-CREB expression in TQ treated groups especially the group treated with TQ and allosteric modulator. Docking results show hydrophobic interactions of TQ similar to ligand interactions in complex with the receptors. This indicates the possible direct agonistic effect of TQ on $\alpha 7$ nAChR and its role in modulating cognitive defects. TQ can be promising therapeutic module for treatment of AD.

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