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Neurotoxical Effects on Alpha Nicotinic Receptor

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The alpha-7 nicotinic receptor, also known as the α7 receptor, is a type of nicotinic acetylcholine receptor implicated in long-term memory, consisting entirely of α7 subunits. Further, recent work has implicated this receptor as being important for generation of adult mammal neurons in the retina.

Nicotinic acetylcholine receptors (nAChRs) are cholinergic receptors that form ligand-gated ion channels in the plasma membranes of certain neurons and on the postsynaptic side of the neuromuscular junction. As ionotropic receptors, nAChRs are directly linked to ion channels and does not use second messengers. Nicotinic acetylcholine receptors (nAChRs) are cholinergic receptors that form ligand-gated ion channels in the plasma membranes of certain neurons and on the postsynaptic side of the neuromuscular junction. Alpha7 nicotinic receptor biology and recent efforts to target the receptor in clinical trials, it is hoped that investigators will be motivated to explore novel, promising directions focusing on the receptor as a strategy to treat cognitive symptoms in schizophrenia.

The majority of the neurological affects of nicotine are meditated through its actions on the alpha nicotinic receptor1. It is highly susceptible to up regulation. Yet, neurotoxity produced by other drugs of abuse markedly decrease receptors populations. The majority of the neurotoxical affects of nicotine are meditated through its actions on the alpha nicotinic receptor Carlson et al. It is highly susceptible to up regulation. Yet, neurotoxity produced by other drugs of abuse markedly decrease receptors populations. Since receptor populations have decreased due to neurotoxicity produced by drugs; therefore, the objective is to investigate both the immediate and long term effects of this toxicity on this receptor population was investigated. Design method was implemented like Nicotine tartrate treatment in saline solution to rats also Immunohistochemistry of Hippocampus with Immunohistochemistry of Medial Habenula, Fascicuclus retroflexus, Thalamus For the experimental set-up, 32 female Sprague-Dawley rats and antibodies for the receptor subunits are required. Partcipants were Thirty two female Sprague-Dawley rats (n=17 per treatment group). Dosage of 7.13mg/kg of Nicotine tartrate in saline solution with standard control of saline for five days to 32 female Sprague-Dawley rats Immunohistochemistry analysis of Hippocampus, Medial habenula, Fascicuclus retroflexus, and Thalamus using antibodies for the alpha and alpha receptor subunits. 32 female Sprague-Dawley rats were given either 7.13 mg/Kg nicotine tartrate in a saline solution (n =17 per treatment group) or saline for 5 days. Mice was sacrificed either two or 14 days after treatment cessation. Immunohistochemistry was performed on the tissue from the hippocampis, medial habenula , fascicuclus retroflexus and thalamus using antibodies for the alpha and alpha receptor sub units. Treatment with nicotine Nicotine significantly reduced the number of both a4 and alpha 2 subunits, 14 days after the treatment compared with the saline treatment in all 3 three brain regions however the number of both receptor subunits was were significantly increased in all 4 four brain regions 2 two days after treatment compared with both the saline and long-term tobacco groups. Neurotoxic doses of nicotine cause an immediate down regulation of alpha nicotine receptor. However, the long-term effects of neurotoxic doses are deleterious in that they result in substantial decreases in this population. This research important accounted by the fact that the critical role of the alpha receptor in cognition. Various different future studies are needed to confirm these results.

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