

NEUROPROTECTIVE POTENTIAL OF ALLICIN AGAINST 3-NP INDUCED HUNTINGTON DISEASE IN RATS

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Acetylcholine (ACh) has been the first molecule to be identified as neurotransmitter. The cholinergic and cholinceptive areas, both in central and peripheral nervous system, have been well documented. Acetylcholine has been described to control, during embryogenesis, cell proliferation as well as neuron and glial cell survival and differentiation. In the adult, acetylcholine and its receptors are distributed in many tissues other than in the nervous system. More recently, new physiological roles in neuronal and non-neuronal tissues have been proposed for ACh as well as its possible involvement in different pathologies. Altered levels of ACh or modified receptors expression and function, in selected areas of the nervous system, have been described in several neurodegenerative diseases such as Alzheimer's, Parkinson's and Huntington as well as in psychiatric disorders such as schizophrenia. Frequently own cognitive, behavioral and motor disabilities that characterize these pathologies are correlated to cholinergic circuit dysfunction. Moreover the involvement of ACh as modulator of the inflammation, in and out of the nervous system, has suggested that its altered functions might represent an additional pathogenetic mechanism negatively influencing the disease outcome as recently suggested in multiple sclerosis. The present review will focus on identifying the cause/effect relationship that may explain the cholinergic dysfunction in several nervous system disorders. Moreover the possible therapeutic novelties including cholinesterase inhibitors, muscarinic agonists and antagonists, and genetic therapy will be discussed.

