Traitement d'un modèle rongeur de maladie de Huntington par un agoniste des 7nAChR
Alpha-7 nicotinic acetylcholine receptor agonist treatment in a rat model of Huntington's disease and involvement of heme oxygenase-1

Abstract

Neuroinflammation is a common element involved in the pathophysiology of neurodegenerative diseases. We recently reported that repeated alpha-7 nicotinic acetylcholine receptor (7nAChR) activations by a potent agonist such as PHA 543613 in quinolinic acid-injured rats exhibited protective effects on neurons. To further investigate the underlying mechanism, we established rat models of early-stage Huntington's disease by injection of quinolinic acid into the right striatum and then intraperitoneally injected 12 mg/kg PHA 543613 or sterile water, twice a day during 4 days. Western blot assay results showed that the expression of heme oxygenase-1 (HO-1), the key component of the cholinergic anti-inflammatory pathway, in the right striatum of rat models of Huntington's disease subjected to intraperitoneal injection of PHA 543613 for 4 days was significantly increased compared to the control rats receiving
intraperitoneal injection of sterile water, and that the increase in HO-1 expression was independent of change in 7nAChR expression. These findings suggest that HO-1 expression is unrelated to 7nAChR density and the increase in HO-1 expression likely contributes to 7nAChR activation-related neuroprotective effect in early-stage Huntington's disease.

Keywords
Alzheimer's disease; biological availability; chemical components; curcumin; early diagnosis; magnetic resonance imaging; nerve regeneration; neural regeneration; neurodegeneration; positron emission tomography; senile dementia

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