Cotinine improves visual recognition memory and decreases cortical Tau phosphorylation in the Tg6799 mice

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Alzheimer's disease (AD) is associated with the progressive aggregation of hyperphosphorylated forms of the microtubule associated protein Tau in the central nervous system. Cotinine, the main metabolite of nicotine, reduced working memory deficits, synaptic loss, and amyloid ? peptide aggregation into oligomers and plaques as well as inhibited the cerebral Tau kinase, glycogen synthase 3? (GSK3?) in the transgenic (Tg)6799 (5XFAD) mice. In this study, the effect of cotinine on visual recognition memory and cortical Tau phosphorylation at the GSK3? sites Serine (Ser)-396/Ser-404 and phospho-CREB were investigated in the Tg6799 and non-transgenic (NT) littermate mice. Tg mice showed short-term visual recognition memory impairment in the novel object recognition test, and higher levels of Tau phosphorylation when compared to NT mice. Cotinine significantly improved visual recognition memory performance increased CREB phosphorylation and reduced cortical Tau phosphorylation. Potential mechanisms underlying theses beneficial effects are discussed. © 2017

Alzheimer's disease

Cotinine

CREB

Memory

Tau

cotinine

cyclic AMP responsive element binding protein

phosphoprotein

serine

tau protein

cotinine

Creb1 protein, mouse

cyclic AMP responsive element binding protein

tau protein

animal experiment

animal model

animal tissue

Article

binding site

brain cortex

controlled study

drug effect

drug mechanism

male

memory disorder

mouse

nonhuman

novel object recognition test

protein phosphorylation

visual memory

visual recognition memory

animal

drug effects

memory

metabolism

phosphorylation

recognition

transgenic mouse

vision

Animals

Cerebral Cortex

Cotinine

Cyclic AMP Response Element-Binding Protein

Male

Memory

Mice

Mice, Transgenic

Phosphorylation

Recognition (Psychology)

tau Proteins

Visual Perception