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POMGnT1 Deficiency Promotes Tau Hyperphosphorylation and Cognitive Impairment in Alzheimer's Disease

Protein O-linked mannose β1,2-N-acetylglucosaminyltransferase 1 (POMGnT1) is a well-characterized mannosyltransferase widely distributed in the brain, and its dysregulation has been implicated in neurodegenerative diseases. Our research team found that POMGnT1 expression is downregulated while Tau protein phosphorylation levels are increased in the brain tissues of both Alzheimer's disease (AD) patients and AD mouse models. To elucidate whether POMGnT1 deficiency serves as a causative factor for Tau pathology in AD or merely represents an epiphenomenon during disease progression, we generated neuron-specific POMGnT1 conditional knockout (POMGnT1 cKO) mice and established POMGnT1-knockdown N2a cells. Furthermore, rescue experiments were conducted by overexpressing POMGnT1 in an N2a cell model stably transfected with the TauP301L plasmid (N2a-Tau). Cognitive function in mice was assessed using the Morris water maze test and open field test, while Tau phosphorylation levels in mouse brain tissues and cultured cells were examined via Western blotting (WB) and immunofluorescence (IF). Our results demonstrated that, compared to wild-type (WT) mice, 15-month-old POMGnT1 cKO mice exhibited cognitive decline and increased Tau hyperphosphorylation in the cerebral cortex and hippocampus. Similarly, POMGnT1 deficiency in cultured cells induced elevated Tau phosphorylation levels. Notably, overexpression of POMGnT1 in the rescue experiment reversed Tau hyperphosphorylation in the N2a-Tau cell model. Collectively, these findings demonstrate that POMGnT1 deficiency is a causative factor for Tau pathology in AD, suggesting that POMGnT1 may serve as a potential therapeutic target for this disease.

Keywords: POMGnT1, Tau phosphorylation, Alzheimer's disease, Conditional knockout mice, Neurodegeneration

Biography

Master's Supervisor, Deputy Director of the Department of Geriatrics at the Second Affiliated Hospital of Chongqing Medical University, and a "Kuanren Backbone Talent" at the Second Affiliated Hospital of Chongqing Medical University. She serves as Executive Committee Member of the Cognitive Impairment Branch of the Chinese Geriatrics Society. She has studied abroad in the United States and Canada. She has been engaged in neurology for 27 years. Her main research interests include the mechanisms, diagnosis, and treatment of memory decline, cognitive dysfunction, and dementia. She has published 70 academic papers.

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