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XHC restores cognition of Alzheimer's disease mice and reduces amyloid beta burden via repressing BACE1 promoter activity

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Alzheimer's disease (AD) is a chronic neurodegenerative disease. In developed countries, AD is one of the most financially costly diseases however, the cause of AD is still unclear. Among the many hypotheses, Amyloid hypothesis postulated that exceed extracellular amyloid beta (Aβ) deposits are the fundamental cause of the disease. Aβ is produced by sequential proteolysis to amyloid beta precursor protein (APP) by β-secretase (BACE1) and γ-secretase. Another important phenomenon in AD patient is increased BACE1 expression. Many big pharmaceutical companies have been focused on developing direct inhibitors for BACE1. However, direct and complete blocking of enzymatic activity of BACE1 can cause unpredictable side effects because of numerous physiological substrates of BACE1. Therefore, our strategy is to find specific drugs reducing BACE1 expression rather than direct inhibition of BACE1. Using USA FDA approved drug library (Prestwick Chemical Library), we could discover putative therapeutic chemicals by cell based assay. Among those candidates, XHC reduced the levels of BACE1 protein and mRNA in SH-SY5Y cells. A soluble APPβ and C99 which are the products of BACE1 protease, were also decreased by treatment of XHC. We also confirmed that XHC could improve cognitive functions of 3xTg-AD mice. Decreased level of Aβ deposition and BACE1 expression also observed in XHC-treated AD mice.

Biography

Gun Young has completed his B.S. from Sungkyunkwan University School of Pharmacy in 2015. He is doing his master's degree at Sungkyunkwan University School of Pharmacy. His major expertise is molecular cell biology.

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