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Inhibition of mitochondrial fission ameliorates the pathogenesis of Alzheimer's disease

Heejin Park
Sungkyunkwan University, Republic of Korea

Excessive mitochondrial fission is a prominent early event, and contributes to mitochondrial dysfunction, synaptic failure and neuronal cell death in the progression of Alzheimer's disease (AD). In the present study, we examine the role of Drp1, a key regulator of mitochondrial fragmentation, in mitochondrial and synaptic dysfunction-induced by Aβ, and AD-like neuropathology and cognitive functions in AD mice. Our results demonstrate that the inhibition of Drp1 alleviates mitochondrial fragmentation, loss of mitochondrial membrane potential, ROS production, and ATP reduction in neurons treated with Aβ oligomers. An inhibitor of Drp1 also significantly restores Aβ-mediated depression of synaptic vesicle exocytosis. Furthermore, Drp1 inhibition significantly improves learning and memory, synaptic density, and prevents mitochondrial fission, lipid peroxidation, BACE1 expression and Aβ deposition in an AD mouse model. These results provide evidence that Drp1 plays an important role in Aβ-mediated and AD-related neuropathology, and in cognitive function in an AD animal model. Thus, inhibiting excessive Drp1-mediated mitochondrial fission may be an efficient therapeutic avenue for AD.

Biography

Heejin Park is studying at Molecular Cell Biology laboratory, from Sungkyunkwan University School of Pharmacy, South Korea, for her M.S. Course. She completed her bachelor degree from the Department of Genetic engineering of Sungkyunkwan University and decided to transfer to School of Pharmacy because she was interested in neurodegenerative diseases and Alzheimer's Disease. Now she is investigaing for the relationship between mitochondrial dysfunction and Alzheimer's Disease.

pania994@naver.com

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