

Role of presenilin-1 mutations in Mitochondrial dynamics

Early stage of Alzheimer's disease reveals mitochondrial deficit and dysfunction. Mitochondrial dysfunction in Alzheimer's disease causes synaptic alteration, imbalance of lipid homeostasis, calcium homeostasis, and lack of ATP production. Familial Alzheimer's disease-linked Presenilin-1, catalytic subunit of γ -secretase, mutations cause early onset Alzheimer's disease. All mutation types have different pathological mechanism and ultimately break down cellular homeostasis. Our research shows more details about relationship between Presenilin-1 mutations (PS1A431E, PS1E280A, PS1H163R, PS1M146V, PS1 E9) and mitochondrial dysfunctions. All of PS1 mutants-expressing cells exhibited mitochondrial dysfunctions and reduced levels of proteins involved in mitochondrial dynamics without alteration of total mitochondrial biogenesis.

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 investigating for the relationship between mitochondrial dysfunction and Alzheimer's

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