techman / October 11, 2012 07:02PM

[Medicine] Academia Sinica Research Team Has Found the Drugs for Frontotemporal Lobar Degeneration Treatment

[Medicine] Academia Sinica Research Team Has Found the Drugs for Frontotemporal Lobar Degeneration Treatment (Chinese Version)

Academia Sinica News (2012/10/04) Academician Che-Kun James SHEN at Institute of Molecular Biology, Academia Sinica recently has been cooperating with Professor Kuen-Jer TSAI at Institute of Clinical Medicine, National Cheng Kung University, leading the research team successfully to find potential drugs for alleviation and treatment of frontotemporal lobar degeneration (FTLD) in Taiwan. FTLD is one of the main causes of dementia, and so far no medication can be used. The research team used autophagy activator -rapamycin to treat the mouse model of FTLD pathology. The result of this research has been published in an important international journal, Proceedings of the National Academy of Sciences of the United States of America, PNAS.

The pathological and clinical syndrome of FTLD includs the brain atrophy of frontal and temporal lobe, memory loss, speechless, neuromotor disorders, and even would be complicating with motor neuron disease. In the elderly population over the age of 65, FTLD is the fourth most common causes of dementia, only after Alzheimer's disease, Lewy body dementia and vascular dementia. However, FTLD is the second common causes of dementia preceded by to Alzheimer's disease in the populations less than 65 years old. Recent studies have found the mis-metabolism of a protein, which can affect neuronal activity named TDP-43, is correlated to several neurodegenerative diseases, including FTLD and amyotrophic lateral sclerosis, ALS.

Previously the research team led by Professor TSAI had transgenically overexpressed TDP-43 in the forebrain of a mouse, successfully developing an animal 34 model existing phenotypic characteristics mimicking of FTLD. The research team applied autophagy activator to the model mouse in the early stage of pathology, discovering that it not only maintained the learning/memory ability of the animal model but also slowed down the loss the motor function, and reduced cytosolic overexpression TDP-43 and its abnormally aggregation, therefore ameliorating the proteinopathy-induced neuronal apoptosis. The research team also showed delivery of the autophagy activators at the late stage of disease progression can ameliorate the motor function. This finding is a major breakthrough for treatment of TDP-43 proteinopathy induced neurodegenerative diseases. Through this study, scientific and medical communities can investigate and improve a more effective application to treat FTLD.

Related Website:
http://www.pnas.org/content/109/37/15024.abstract

Further Information:
Academia Sinica News 2012/10/04

---
National Science Council International Cooperation Sci-Tech Newsbrief
---

Edited 1 time(s). Last edit at 10/11/2012 07:06PM by techman.