apophasis / August 27, 2010 02:19PM

[Molecular Biology] Researchers Develop Mouse Model for Study of Frontotemporal Lobar Degeneration, a Common Cause of Dementia

[Molecular Biology] Researchers Develop Mouse Model for Study of Frontotemporal Lobar Degeneration, a Common Cause of Dementia (Chinese Version)

Academia Sinica Newsletter (2010/08/27) A team of Taiwan researchers led by Academician Che-Kun James SHEN, a Distinguished Research Fellow of the Institute of Molecular Biology, Academia Sinica, has engineered the first mouse model of Frontotemporal lobar degeneration (FTLD). The advance is expected to speed up the development of new drugs and treatments for the disease. FTLD is a common cause of dementia for which there is currently no treatment. The study was published in the Journal of Experimental Medicine on July 27, 2010.

FTLD is associated with atrophy in the frontal and temporal lobes of the brain, memory loss, aphasia (impairment of language ability) and sometimes motor neuron diseases. In the under 65 age group, it is the second most common cause of dementia after Alzheimer's disease; and in the over 65s it is the fourth most common cause of dementia after Alzheimer's disease, dementia with Lewy bodies, and vascular dementia. Recent studies have shown that dysmetabolism of a type of cellular protein, known as TAR DNA binding protein 43 (TDP-43), is closely associated with a range of neurodegenerative diseases including FTLD-U, a subclass of FTLD, and amyotrophic lateral sclerosis (ALS, a fatal neurodegenerative disease paralyzing the victim). Different gene mutations including those in the TDP-43-encoding gene are believed to contribute to these neurodegenerative disorders. Also, the level of TDP-43 protein is often found to be elevated in FTLD-U and ALS.

In this study, the researchers genetically engineered mice with increased levels of TDP-43 in the forebrain, and discovered that these mice exhibited impaired cognition and motor performances, brain atrophy and a deficit in the long-term potentiation that mimic FTLD.

The finding that increased levels of TDP-43 protein in the forebrain are sufficient to cause FTLD and possibly other neurodegenerative diseases yields a new avenue for future studies of FTLD. Using the new model Academician SHEN's research team has already started to test a few potential drugs for treating FTLD. A collaboration with a laboratory based in Australia has also been established for drug screening.

Related Website: http://jem.rupress.org/content/207/8/1661.abstract

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Reference:
Academia Sinica Newsletter 2010/08/27

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National Science Council International Cooperation Sci-Tech Newsbrief

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Edited 2 time(s). Last edit at 08/27/2010 02:24PM by apophasis.