gustav / June 15, 2010 10:44AM

[Bio-Medical] Mouse Study Sheds Light on Possible Mechanism Causing Hair Loss, Osteoporosis and Protein-folding Related Diseases

[Bio-Medical] Mouse Study Sheds Light on Possible Mechanism Causing Hair Loss, Osteoporosis and Protein-folding Related Diseases (Chinese Version)

Academia Sinica Newsletter (2010/06/11) An international team of researchers led by Academician Yuan-Tsong CHEN and Dr. Jeffery YEN, respectively Director and Research Fellow of the Institute of Biomedical Sciences at Academia Sinica have demonstrated that mutation of one gene can result in the manifestation of characteristics of a diverse array of serious diseases in mice. For the first time, the group has found a direct link between one particular type of protein modification and common problems, such as hair loss and osteoporosis, an exciting first step to understanding the molecular mechanisms underlying these diseases. The study was published in the international scientific journal PLoS Genetics on June 10, 2010 (US Eastern Standard Time).

The team, which included Dr. Monica JUSTICE, a Professor of the Baylor College of Medicine in Houston, found that defective “palmitoylation” resulted in the characteristics of several serious diseases being seen in the mice. Palmitoylation is a common protein modification that involves the addition of palmitate (a fatty acid) to proteins. Many soluble and integral membrane proteins have been shown to be palmitoylated, including signaling proteins, enzymes, scaffolding proteins, ion channels, cell adhesion molecules, and neuronal proteins. Despite the known functional importance of protein palmitoylation at the cellular and biochemical levels, its physiological role and its relevance to disease processes has not been clear.

In the study defective palmitoylation caused hair loss; severe osteoporosis; cachexia, a wasting syndrome characterized by loss of weight weakness and fatigue often seen in patients with chronic diseases such as cancer AIDS or tuberculosis; and systemic amyloidosis, a deadly protein misfolding disease in which misfolded proteins destroy vital organs such as the heart, kidneys and liver. It also caused early death.

The defective palmitoylation was caused by a mutation in the Zdhhc13 gene which codes for palmitoyl acyltransferase the enzyme that catalyzes the reaction of protein palmitoylation. The multi-organ/system failure of mice with mutated Zdhhc13 established a direct link between protein palmitoylation and the regulation of many important diverse physiological functions and indicates that the absence of Zdhhc13 can result in profound disease pathologies.

The mouse model developed by the team will be useful for further investigation of the mechanisms by which improper palmitoylation leads to disease including the identification of target proteins of ZDHHC13. The elucidation of these proteins would be an exciting first step to understanding the molecular mechanisms underlying human alopecia, osteoporosis and many neurodegenerative diseases caused by protein misfolding and amyloidosis.

The full article entitled “Mice with Alopecia, Osteoporosis and Systemic Amyloidosis due to Mutation in Zdhhc13, a Gene coding for Palmitoyl Acyltransferase” is available at the PLoS journal website at:
http://www.plosgenetics.org/home.action

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Further Information:

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weifeng / June 19, 2010 12:14PM

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