New genetic mechanism that controls body's fat-building process found

August 26 2009

At a time of alarming increases in obesity and associated diseases -- and fiery debates about the cost of health care -- a UCF research team has identified a new genetic mechanism that controls the body's fat-building process.

The discovery could open the door to new treatments for obesity and type 2 diabetes, and it has the potential to help hundreds of millions of people and dramatically cut health care costs.

A research team led by Pappachan Kolattukudy, director of UCF's Burnett School of Biomedical Sciences in the College of Medicine, found that a gene called MCPIP (Monocyte Chemotactic Protein-1 Induced Protein) controls the development of fat cells. Until now, a different protein, known as peroxisome proliferator-activated receptor gamma (PPAR gamma), has been universally accepted as the master controller of fat cell formation, known as adipogenesis.

The UCF findings give scientists a new direction for developing drugs that could benefit the more than 300 million people worldwide who are clinically obese -- and who have much higher risks of suffering from chronic disease and disability. In addition, it is projected that more than 300 million people will be diabetic by the year 2025.

Kolattukudy said MCPIP is potentially an ideal target for drugs that would prevent the body from becoming resistant to insulin and prone to type 2 diabetes.

"Our research has shown that MCPIP is a regulator of fat cell formation and blood vessel formation that feeds the growing fat tissue," he said. "Therefore, a drug that can shut down its function can prevent obesity and the major inflammatory diseases resulting from obesity, including diabetes and cardiovascular diseases."

The findings will be published in the October issue of the Journal of Biological Chemistry. An advance version is now available online on the journal's Web site.

Kolattukudy introduced MCPIP to living cells from mice that had been stripped of the PPAR gamma gene and found that the cells still completed the developmental process necessary to build fat.

His next step is to begin exploring chemical combinations to discover drugs that are effective at shutting down the novel gene. The development of new drugs that can block or slow down the formation of MCPIP likely would take several years. However, Kolattukudy is encouraged by the results of his research to date.

Kolattukudy, whose team in 2006 first identified the MCPIP gene as a contributor to heart disease, found its function as a fat inducer by focusing on its inflammatory influence.

Recent evidence has shown that the increased inflammation of fat cells causes them to become less sensitive to insulin, potentially triggering type 2 diabetes. A predominance of fatty tissue contributes to the inability to process insulin which, in turn, enables glucose or sugars to flow directly to the bloodstream instead of going into cells.

Source: University of Central Florida (news : web)