Trigger mechanism for obesity elucidated:
The differences in predisposition to obesity clearly identified

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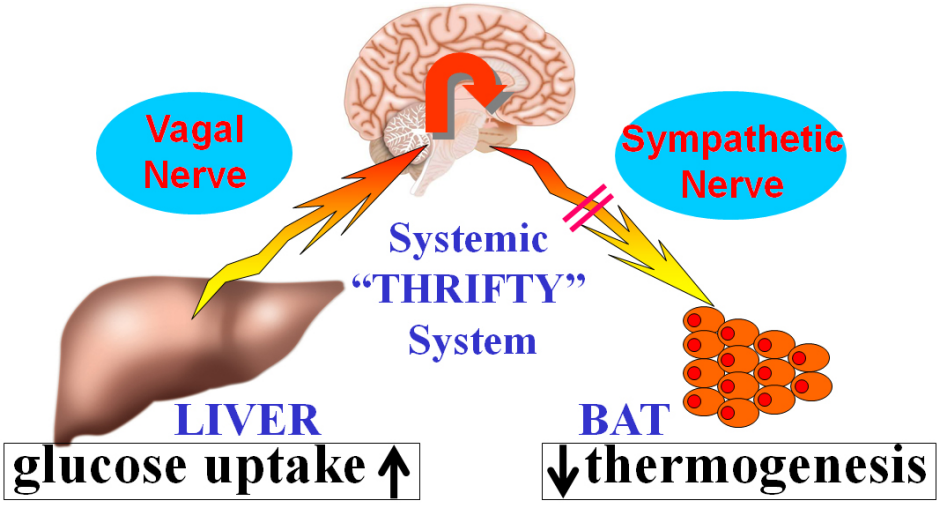
The research group led by Assistant Professor Tetsuya Yamada, Assistant Sohei Tsukita, and Professor Hideki Katagiri, Tohoku University Graduate School of Medicine, has found that nerve signals, emitted in response to the glucose metabolism in the liver due to overeating, reduced the caloric consumption of brown fat leading to obesity. This mechanism was also found to be involved in the differences in weight gain among different individuals.

The research results were published in the American scientific journal Cell Metabolism on December 4, 2012. The paper's title is "Hepatic glucokinase modulates obesity predisposition by regulating BAT thermogenesis via neural signals."



This inter-tissue (liver-to-BAT) energy-saving system contributes to determining obesity predisposition.

Hepatic overexpression of glucokinase, a rate-limiting enzyme of glycolysis, enhanced glucose metabolism in the liver but decreased adaptive thermogenesis in brown adipose tissue (BAT). This inter-tissue (liver-to-BAT) system consists of the afferent vagus from the liver and sympathetic efferents from the medulla. Furthermore, up-regulation of endogenous GK in the liver by high-fat feeding was more marked in obesity-prone than in obesity-resistant strains. Hepatic GK overexpression in obesity-resistant mice promoted weight gain, while hepatic GK knockdown in obesity-prone mice attenuated weight gain. Thus, this inter-tissue energy-saving system contributes to flipping a metabolic switch promoting obesity as well as determining obesity predisposition.

"Hepatic glucokinase modulates obesity predisposition by regulating BAT thermogenesis via neural signals"
Tsukita S, Yamada T, Uno K, Takahashi K, Kaneko K, Ishigaki Y, Imai J, Hasegawa Y, Sawada S, Ishihara H, Oka Y, Katagiri H.
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