

Insulin Sensitizer Also Serves As Energy-conserving Signal To The

Insulin Sensitizer Also Serves As Energy-conserving Signal To The Brain

Science Daily A fat-derived protein known for its effects on the liver and skeletal muscle might also serve as an energy-conserving signal to the brain during periods of starvation, suggests a new study in the July issue of *Cell Metabolism*, a publication of Cell Press. The substance, known as adiponectin, acts on the brain to boost appetite and slow energy expenditure in an effort to maintain adequate fat stores during lean times, the researchers report.

Energy homeostasis may be mediated by both short-term regulators, such as gut hormones, and long-term regulators, said Takashi Kadowaki of the University of Tokyo. In this study, we identified, for the first time, a potential long-term regulator that allows energy to be stored efficiently, namely, adiponectin. The findings offer critical insight into adiponectin's influence over the central nervous system and suggest that selective inhibition of the chemical in the brain may represent a novel therapeutic strategy for obesity and obesity-linked diseases, he added.

White adipose tissue (WAT) is a major site of energy storage and plays an important role in energy balance, the researchers said. It is also recognized as an important endocrine organ that secretes a number of biologically active signaling proteins, called adipokines. Adiponectin, an adipokine secreted exclusively by WAT, is present at relatively high concentrations in the circulation and has been shown to increase the body's response to insulin. Studies have also suggested that decreased circulating levels of adiponectin in obesity and type 2 diabetes may contribute to the insulin resistance that characterizes both conditions.

In addition to its peripheral actions on the liver and skeletal muscle, adiponectin has also been reported to have central actions, Kadowaki said. Recently, however, it was reported that adiponectin is undetectable in human cerebrospinal fluid and does not cross the blood-brain barrier, leaving some doubt about its physiological role in the central nervous system, he added.

The researchers now report evidence in mice that adiponectin receptors are present in the hypothalamic region of the brain and that some forms of the chemical enter the cerebrospinal fluid from the blood. Once in the brain, adiponectin enhances the activity of a metabolic enzyme called AMP-activated protein kinase (AMPK) to stimulate greater food consumption.

Moreover, the researchers found that adiponectin decreased energy expenditure. They also showed that blood and spinal fluid adiponectin levels in the brain normally increase during fasting and decrease after refeeding, suggesting that adiponectin acts mainly during food shortages.

In adiponectin-deficient mice, AMPK activity in the brain slowed, causing the animals to eat less and expend more energy. That action, in turn, made the animals resistant to becoming obese even on a high-fat diet. Moreover, animals lacking adiponectin lost more fat after 12 hours of fasting than normal mice did.

Blood levels of another fat hormone, leptin, are regulated inversely in relation to serum adiponectin levels, the researchers noted.

Thus, central adiponectin/leptin signals may represent the physiological pathway by which hypothalamic AMPK activity and food intake are stimulated during fasting and suppressed after refeeding, they said. In addition to this short-term regulation of food intake and energy expenditure by adiponectin and leptin, these two adipokines may also participate in the long-term regulation of energy homeostasis. The fundamental roles of leptin and adiponectin seem to be to preserve an adequate fat reserve: leptin acts as a satiety signal, and adiponectin acts as a starvation signal.

The researchers include Naoto Kubota, Iseki Takamoto, and Takashi Kadowaki of the University of Tokyo, CREST, Japan Science and Technology Corporation, and the National Institute of Health and Nutrition in Tokyo, Japan; Wataru Yano, Shinsuke Itoh, Hiroki Kumagai, Hideki Kozono, Hidemi Satoh, Atsushi Tsuchida, Ryozi Nagai, and Kohjiro Ueki of the University of Tokyo in Tokyo, Japan; Tetsuya Kubota of the University of Tokyo, National Institute of Health and Nutrition, and Toho University, Ohashi Hospital in Tokyo, Japan; Toshimasa Yamauchi, Ryo Suzuki, Kazuyuki Tobe of the University of Tokyo, CREST, and Japan Science and Technology Corporation in Tokyo, Japan; Shiki Okamoto, Tetsuya Shiuchi, and Yasuhiko Minokoshi of National Institute for Physiological Sciences in Okazaki, Japan; Masao Moroi and Kaoru Sugi of Toho University, Ohashi Hospital in Tokyo, Japan; Tetsuo Noda of the Japanese Foundation for Cancer Research and Tohoku University School of Medicine in Miyagi, Japan; Hiroyuki Ebinuma of Diagnostics Research Laboratories, Daiichi Pure Chemicals in Ibaraki, Japan; Yoichi Ueta of University of Occupational and Environmental Health in Fukuoka, Japan; Tatsuya Kondo and Eiichi Araki of Kumamoto University in Kumamoto, Japan; Osamu Ezaki of National Institute of Health and Nutrition in Tokyo, Japan; Yasuo Terauchi of Yokohama City University School of Medicine in Kanagawa, Japan.

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