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Commentary

Regulation of Lipid Metabolism by Leptin and its Role

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DESCRIPTION

Lipid metabolism is the process by which the pancreas and small intestine secrete lipase, which hydrolyzes the fatty acids in fat into free fatty acids and monoglycerides, converts the majority of the fat the body consumes into small particles. The regulation of aging is heavily reliant on lipid metabolism. In a variety of model organisms, experimental observation indicates that lipid metabolism changes with age and those lipid-related interventions can alter age-related diseases and aging. Metabolism performs three primary functions: the process by which food's energy is converted into energy that can be used by cellular processes; the transformation of food into the components that go on to make proteins, lipids, nucleic acids, and some carbs; as well as the removal of metabolic wastes.

Adipocytes secrete a hormone called leptin that regulates food intake, energy expenditure, and neuroendocrine function. Leptin regulates glucose and lipid metabolism in rodent and human peripheral tissues independently of the anorexic effect. White adipose tissue is the primary site of leptin production, but the hormone is also expressed in the stomach, lungs, placenta, and possibly the brain. After preadipocytes differentiate into adipocytes and the early stages of cell lipid accumulation, leptin expression is seen in rodent adipose tissue. Expression is low when the infant is nursing, but it quickly rises when the infant is weaned. Although leptin expression varies between fat depots, there is a strong correlation between it and adipocyte size, and the rate of increase is proportional to white fat mass. Due to the presence of small vesicular stores of leptin in adipocytes, although leptin is continuously secreted, the rate of secretion may be regulated independently of leptin MicroRNA (mRNA) expression. To prevent the rapid exhaustion of these leptin stores, sustained stimulation of secretion requires a simultaneous increase in leptin mRNA expression.

Leptin is one of several hormones that play a role in regulating lipid metabolism. Fat mass is determined by a balance between lipid synthesis and degradation. The central nervous system adjusts food intake and energy expenditure based on the circulating leptin level, which is a gauge for energy reserves. By controlling appetite in the brain, leptin has immediate effects. Adipocytes, such as triglycerides, store over 90% of total energy and can be hydrolyzed (lipolyzed) to release fatty acids when hormone stimulation occurs. There are two outcomes for fatty acids, oxidation to make ATP or reesterification to make triglycerides again leptin to have a direct autocrine or paracrine effect on lipid metabolism. By altering the concentration and levels of enzyme mRNA, leptin appears to mediate fatty acid metabolism.

Leptin, for instance, prevents adipocytes from producing the rate-limiting enzyme Acetyl-CoA Carboxylase (ACC), which is necessary for the conversion of carbohydrates into fatty acids and the storage of calories as triglycerids. Leptin's effects on tissue lipid lowering and insulin sensitization likely stem from this stimulation of FA oxidation. It was demonstrated that either direct or indirect stimulation of AMP that inactivates ACC, decreases malonyl-CoA concentration, and thus stimulates CPT 1 mediated fatty acid oxidation in the mitochondria occurs, either through the central nervous system or a putative inhibition of stearoyl-CoA desaturase-1 activity. As a result, the well-documented lipolytic effect of leptin has been regarded as an antisteatotic hormone. Leptin-induced fatty acid oxidation reduces muscle lipid accumulation and suppresses "lipotoxicity," or functional impairments like insulin resistance. As a result, skeletal muscle glucose and fatty acid metabolism are tightly controlled by leptin.

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