

Endocrinological regulation of food intake: a coping science

Philosophy

Nutrient partitioning is mediated by a variety of hormones. Hormones are involved in both short-term and long-term regulation of feed intake. The highest fluctuations in nutrient metabolism, substrate partitioning, and food intake usually occur around growth, parturition and diseases when levels of metabolic and reproductive hormones are highly variable. This article discusses how endocrinological regulation of food intake helps body cope with environmental variations.

Discussion

Estrogen as a reproductive hormone depresses food intake by acting primarily on the paraventricular nucleus of the hypothalamus. Insulin is another important hormone that possesses both long-term and short-term effects on nutrient partitioning and food intake. The long-term effects of insulin on food intake control relate mainly to pregnancy and lactation. These effects occur mainly during mid- and late-lactation when the mother tends to gain weight. Insulin, glucagon, gut peptides and adipokines of differential proteins are involved in up- and down-regulation of food intake in mammals.

When compared to prepartum levels, insulin secretion drops substantively shortly after parturition. Without the postpartum drop in insulin secretion rate, the mother would be unable to use body reserves and cope with insufficient food intake and healthy energy turnover. The low postpartum insulin will additionally enable the mother to gradually increase intake. The short-term insulin effects on nutrient metabolism and partitioning initiate upon or even shortly before eating or nutrient ingestion. Craving for food causes an insulin surge. The higher postprandial insulin surge leads to greater glucose uptake by peripheral tissues. By increasing the peripheral glucose uptake, the postprandial insulin secretion may contribute to satiety. A higher postprandial insulin secretion and, thus, the increased peripheral glucose use may induce satiety signals. In contrast, insulin may stimulate food intake in response to insufficient nutrient supply. Insulin is also linked to overconsumption of energy. The postprandial rise in insulin secretion in high-producing mammals including lactating mother (demanding much energy and nitrogen) may not necessarily depress food intake. Instead, due to the increased nutrient demand of the mammary gland, the postprandial insulin secretion might facilitate nutrient uptake by increasing food intake.¹⁻⁹

As far as glucagon is concerned, intravenous infusion induces satiety in humans. However, intraperitoneal glucagon stimulates glycogenolysis but does not affect food intake in rats. Glucagon stimulates hepatic glucose production via both glycogenolysis and gluconeogenesis. The hepatic glucose release does not appear to be the exclusive pathway whereby glucagon may affect satiety. It is by far possible that increased blood glucose not end the meal. The peritoneal use of rabbit glucagon antibodies in rats to reduce gluconeogenesis and blood glucose, increases food intake. These findings suggest that reduced blood glucose can induce hunger, but increased blood glucose may not induce satiety. Also, exogenous glucagon reduces feed intake

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in sheep models. Thus, the effect of glucagon on food intake control appears to be mediated by other agents than only glucose.¹⁰⁻¹⁴ Future research is required to elucidate independent and interactive impacts of insulin, glucagon, gut peptides and adipokines on food intake of normal, overweight, and obese young growing individuals as well as male and female adults.

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Conflict of interest

Author declares that there is no conflict of interest.

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