Journal of Endocrinology, Diabetes and Metabolic Complications (JEDMC)

OLITES PUBLISHERS
A Global Approach For Research

Open Access

Review Article

Endocrinology of Obesity: Understanding the Hormonal Architecture of Weight Regulation

Zorov S, Kovatchev F, Sakane U, Wiseman G, Veremeyko D
Research Unit Molecular Endocrinology and Metabolism, South Korea
***Corresponding Author:** Sakane U**,** Research Unit Molecular Endocrinology and Metabolism, South Korea
Citation: Sakane U (2026). Research Unit Molecular Endocrinology and Metabolism, South Korea V2(2)
Copyright: © 2026 Sakane U, this is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited
Received date: June 11, 2026: Accepted date: June 22, 2026: Published date: June 30,2026

Keywords: obesity, endocrinology, leptin, insulin resistance, ghrelin, glp-1, adipokines, metabolism, hormonal regulation, metabolic disease

Abstract

Obesity is a complex endocrine and metabolic disorder in which excessive or dysfunctional adipose tissue interacts with the brain, gastrointestinal tract, pancreas, liver, skeletal muscle, and reproductive system. Although traditionally viewed primarily as an imbalance between calorie intake and energy expenditure, contemporary endocrinology demonstrates that body weight is strongly influenced by interconnected hormonal, neural, genetic, and metabolic mechanisms. Key regulators include leptin, insulin, ghrelin, glucagon-like peptide-1 (GLP-1), peptide YY, adiponectin, cortisol, thyroid hormones, and sex steroids. Alterations in these pathways can affect appetite, satiety, energy expenditure, glucose metabolism, fat distribution, and long-term weight maintenance. Obesity itself further disrupts endocrine function, creating a self-reinforcing cycle of insulin resistance, chronic low-grade inflammation, altered adipokine secretion, and metabolic dysfunction. This article explores the endocrine foundations of obesity, emphasizing the communication between adipose tissue and other organs, mechanisms of hormonal dysregulation, and the implications for modern obesity management. Understanding obesity as an endocrine-metabolic disease provides a more comprehensive framework for prevention, diagnosis, and individualized treatment.

Introduction

Obesity has emerged as one of the most important metabolic disorders worldwide. Its consequences extend beyond increased body weight and include type 2 diabetes mellitus, cardiovascular disease, fatty liver disease, reproductive dysfunction, sleep-related disorders, and several forms of cancer. The development and persistence of obesity cannot be explained solely by voluntary food intake or physical inactivity. Instead, body weight is regulated by a sophisticated endocrine-neural network that continuously integrates information about nutritional status, energy stores, gastrointestinal activity, stress, and reproductive function.

The endocrine system plays a central role in this network. Hormones act as biological signals that influence hunger, satiety, energy expenditure, glucose utilization, lipid storage, and tissue growth. When these signals become altered, the body's preferred weight range and metabolic responses may change. Importantly, many hormonal adaptations occurring during weight loss can promote weight regain, helping explain why long-term weight management can be difficult.

Adipose Tissue as an Endocrine Organ

Adipose tissue is no longer regarded simply as a passive reservoir for stored energy. It functions as an active endocrine organ that produces numerous signaling molecules known as adipokines. These substances influence appetite, insulin sensitivity, inflammation, vascular function, and energy metabolismLeptin is one of the most important adipose-derived hormones. It is produced predominantly by adipocytes and communicates information about energy stores to the hypothalamus. Increased fat mass generally results in increased circulating leptin concentrations. However, individuals with obesity commonly develop leptin resistance, meaning that elevated leptin levels do not produce an appropriately strong biological response. This impaired signaling can contribute to persistent appetite and difficulty maintaining weight loss.
Adiponectin represents another important adipokine. Unlike leptin, adiponectin concentrations generally

decrease with increasing adiposity. It has insulin-sensitizing, anti-inflammatory, and metabolic effects. Reduced adiponectin may therefore contribute to insulin resistance and cardiometabolic complications associated with obesity.

The Hypothalamic Control of Appetite

The hypothalamus serves as a major central regulator of energy balance. It receives hormonal and nutritional signals from peripheral tissues and coordinates behavioural and metabolic responses.

Within the hypothalamus, opposing neural pathways influence food intake. One pathway promotes appetite through molecules such as neuropeptide Y and agouti-related peptide, whereas another promotes satiety through pro-opiomelanocortin-derived signalling. Leptin and insulin generally favor the anorexigenic pathway, while fasting-related signals such as ghrelin stimulate hunger-promoting circuitsThis system allows the body to defend energy availability. During prolonged calorie restriction, several hormonal changes occur that increase hunger and reduce energy expenditure. These adaptations are biologically useful during periods of food scarcity but can become an obstacle during intentional weight loss.

Insulin andj Obesity

Insulin is essential for glucose regulation and also influences lipid metabolism and energy storage. After food consumption, insulin facilitates glucose uptake into tissues, suppresses hepatic glucose production, and promotes storage of excess nutrients.Chronic excess energy intake and increased visceral adiposity can contribute to insulin resistance. In this state, tissues respond inadequately to insulin, requiring greater insulin secretion to maintain normal glucose concentrations. Hyperinsulinemia may accompany insulin resistance and is closely associated with increased risk of type 2 diabetes

Implications for Modern Treatment

Understanding the hormonal basis of obesity has transformed treatment strategies. Lifestyle interventions remain fundamental, but modern obesity management increasingly recognizes the value of targeting biological pathways involved in appetite and metabolism

Therapeutic approaches may include nutritional modification, physical activity, behavioral interventions, anti-obesity medications, management of endocrine disorders, and metabolic or bariatric surgery when appropriate. Medications that target pathways involving GLP-1 and related incretin signalling have demonstrated the clinical importance of gut-brain endocrine communication

Conclusion

The endocrinology of obesity reveals a complex biological system in which adipose tissue, the gastrointestinal tract, pancreas, hypothalamus, thyroid, adrenal glands, and reproductive organs communicate continuously to regulate energy balance. Hormones such as leptin, insulin, ghrelin, GLP-1, peptide YY, adiponectin, cortisol, and thyroid hormones participate in this intricate network.

References

1. Kapoor, N.; Inian, S.; Thomas, N. Chapter 28. Obesity with Diabetes. In A Practical Guide to Diabetes Mellitus, 8th ed.; Jaypee Brothers Medical Publishers: New Delhi, India, 2018;
2. Lean, M.E.; Leslie, W.S.; Barnes, A.; Brosnahan, N.; Thom, G.; McCombie, L.; Peters, C.; Zhyzhneuskaya, S.; Al-Mrabeh, A.; Hollingsworth, K.G.; et al. Primary care-led weight management for remission of type 2 diabetes (DiRECT): An open-label, cluster-randomised trial. Lancet 2018, 391, 541–551.
3. Wing, R.R.; Bond, D.S.; Gendrano, I.N.; Wadden, T.; Bahnson, J.; Lewis, C.E.; Brancati, F.; Schneider, S.; Kitabchi, A.E.; Van Dorsten, B.; et al. Effect of Intensive Lifestyle Intervention on Sexual Dysfunction in Women With Type 2 Diabetes: Results from an ancillary Look AHEAD study. Diabetes Care 2013, 36, 2937–2944.
4. Wong, I.P.; Baldock, P.A.; Herzog, H. Gastrointestinal peptides and bone health. Curr. Opin. Endocrinol. Diabetes Obes. 2010, 17, 44–50.
5. Corbeels, K.; Verlinden, L.; Lannoo, M.; Simoens, C.; Matthys, C.; Verstuyf, A.; Meulemans, A.; Carmeliet, G.; Van Der Schueren, B. Thin bones: Vitamin D and calcium handling after bariatric surgery. Bone Rep. 2018, 8, 57–63.

Journal of Endocrinology, Diabetes and Metabolic Complications (JEDMC)

ThisworkislicensedunderCreativeCommonsAttribution 4.0 License
DOI:10/JEDMC/2026/017

Your next submission with

OlitesPublisherswillreachyouthebelowassets

- We follow principles of publicationledbythe CommitteeonPublicationEthics(COPE).
- Doubleblindedpeerreviewprocesswhichis justaswellasconstructive.
- Permanent archiving of yourarticle onourwebsite
- QualityEditorialservice
- Manuscriptaccessibilityindifferentformats(PDF,FullText)
- authorsretaincopyrights
- uniqueDOIforallarticles
- immediate,unrestrictedonlineaccess

Learn more: Journal of Endocrinology, Diabetes and Metabolic Complications – Olites Publishers (olitespublishing.org)

Journal of Endocrinology, Diabetes and Metabolic Complications (JEDMC)