OPEN ACCESS Freely available online

Medical Safety & Global Health

ISSN: 2574-0407

Commentry

Role of Hormones in Weight Regulation and Metabolic Pathways of Obesity

Samaher Hashim*

Depatrtment of Radiology Science, King Khalid University, Abha, Saudi Arabia

ABOUT THE STUDY

Obesity is a complex condition marked by excessive fat accumulation, leading to significant health challenges. A deep understanding of the biological mechanisms that contribute to obesity is essential for developing effective interventions. This exploration focuses on the role of hormones in weight regulation and the metabolic pathways involved in obesity. By examining these aspects, it can gain insight into the underlying causes of obesity and the ways to address it.

Role of hormones in weight regulation

Hormones are critical regulators of body weight, influencing appetite, metabolism, and fat storage. Several key hormones play pivotal roles in this process:

Leptin: It is produced by adipose (fat) tissue, is often referred to as the satiety hormone. It provides feedback to the brain about the body's fat stores. High levels of leptin signal the brain that the body has sufficient energy reserves, leading to reduced appetite and increased energy expenditure. Conversely, when fat stores are low, leptin levels decrease, signaling hunger and promoting food intake.

Ghrelin: The hunger hormone ghrelin, often termed the hunger hormone, is produced primarily in the stomach. It stimulates appetite and promotes fat storage. Ghrelin levels rise before meals and fall after eating, helping regulate meal timing and portion size. In obese individuals, the ghrelin system can be altered, leading to persistent hunger and overeating.

Insulin: The glucose regulator insulin, a hormone produced by the pancreas, is essential for regulating blood glucose levels. It facilitates glucose uptake into cells and promotes its storage as glycogen in the liver and muscles. Insulin also influences fat storage by promoting lipogenesis (the formation of fat) and inhibiting lipolysis (the breakdown of fat).

Cortisol: The stress hormone cortisol, produced by the adrenal glands, is known for its role in the body's response to stress. It helps regulate metabolism, including glucose metabolism, and influences appetite and fat distribution. Chronic stress and elevated cortisol levels are associated with increased appetite, particularly for high-calorie foods, and abdominal fat accumulation.

Thyroid hormones: Metabolic regulators thyroid hormones, including Thyroxine (T4) and Triiodothyronine (T3), are critical for regulating metabolism. They influence the rate at which the body burns calories and affects overall energy expenditure. Proper thyroid function is essential for maintaining a healthy body weight.

Metabolic pathways involved in obesity

Obesity results from an imbalance between energy intake and expenditure, with several metabolic pathways playing critical roles in this process:

Energy homeostasis and metabolic pathways: Energy homeostasis refers to the balance between energy intake (from food) and energy expenditure (through physical activity and metabolic processes). Disruptions in this balance can lead to weight gain and obesity.

Energy expenditure consists of Basal Metabolic Rate (BMR), physical activity, and thermogenesis. BMR, which is influenced by factors such as age, sex, and muscle mass, represents the energy required for basic bodily functions at rest. Thermogenesis, including non-shivering thermogenesis and exercise-induced thermogenesis, contributes to energy expenditure by generating heat and increasing calorie burn.

Insulin signaling pathway: The insulin signaling pathway is important for regulating glucose metabolism and fat storage. When insulin binds to its receptor on target cells, it activates a cascade of intracellular signaling events that promote glucose uptake and storage.

Insulin resistance, a key contributor of obesity, disrupts this pathway. In insulin resistance, the PI3K/Akt pathway, which promotes glucose uptake and glycogen synthesis, is impaired. Elevated blood glucose levels and increased fat storage result from this disruption.

Lipid metabolism: Lipid metabolism involves the synthesis, breakdown, and utilization of fats. Dysregulation of lipid metabolism contributes to obesity and related health issues.

Lipogenesis, the process of converting excess carbohydrates and fats into triglycerides for storage, involves enzymes such as

Correspondence to: Samaher Hashim, Depatrtment of Radiology Science, King Khalid University, Abha, Saudi Arabia, E-mail: hashimsam@30gmmail.com

Received: 26-Aug-2024, Manuscript No. MSGH-24-34114; **Editor assigned:** 28-Aug-2024, PreQC No. MSGH-24-34114 (PQ); **Reviewed**: 11-Sep-2024, QC No MSGH-24-34114; **Revised:**18-Sep-2024, Manuscript No. MSGH-24-34114 (R); **Published:** 25-Sep-2024, DOI: 10.35248/2574-0407.24.13.238

Citation: Hashim S (2024). Role of Hormones in Weight Regulation and Metabolic Pathways of Obesity. Med Saf Glob Health. 13:238.

Acetyl-CoA Carboxylase (ACC) and Fatty Acid Synthase (FAS). In obesity, increased lipogenesis can lead to excessive fat accumulation.

Inflammation and obesity

Chronic low-grade inflammation is a feature of obesity and plays a role in the development of insulin resistance and metabolic dysfunction. Adipose tissue, particularly visceral fat, releases pro-inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6), which impair insulin signaling and contribute to weight gain. Inflammatory pathways,including Nuclear Factor-kappa B (NF-κB) and c-Jun N-terminal Kinase (JNK), can lead to insulin resistance and metabolic disturbances.

Integrating hormonal and metabolic insights

The interplay between hormones and metabolic pathways is essential for understanding obesity. Hormones such as leptin, ghrelin, and insulin interact with metabolic pathways to regulate body weight. For example, leptin resistance can disrupt insulin signaling and lipid metabolism, exacerbating obesity. Similarly, insulin resistance can affect leptin signaling and appetite regulation.