Trends in Diabetes, Obesity and Metabolism

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Case Report

Impact of Unhealthy Diet on Thalamic Function in Obesity

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Abstract

Diet-induced obesity is a complex condition associated not only with metabolic dysregulation but also with significant alterations in brain function. The thalamus, a central relay structure involved in integrating sensory, metabolic, and reward-related information, plays a crucial role in regulating feeding behavior and energy homeostasis. Chronic exposure to high-fat or high-calorie diets can induce changes in neuronal activity, synaptic plasticity, and functional connectivity within thalamic circuits. These alterations may disrupt communication between the thalamus and hypothalamus, leading to impaired appetite regulation and reduced satiety signaling.

Keyword: Stroke; Outpatients; Physical activity intensity levels; Public; Awareness; Revascularization

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Introduction

Additionally, diet-induced neuroinflammation and changes in neurotransmitter systems such as dopamine and leptin further affect reward processing and food-related motivation. Emerging evidence also suggests structural and functional modifications in the thalamus, which may contribute to altered decision-making and increased impulsivity toward food intake. Overall, these findings indicate that obesity involves both peripheral metabolic disturbances and central nervous system remodeling, with diet-induced changes in thalamic function playing a key role in the development and persistence of excessive eating behavior.

Diet-induced obesity is associated with functional and structural alterations in multiple brain regions involved in energy regulation, including the thalamus. The thalamus serves as a major relay center in the brain, transmitting sensory, visceral, and metabolic information to cortical and limbic structures. Through these connections, it plays an important role in regulating feeding behavior, motivation, and overall energy balance.

Research indicates that chronic exposure to high-fat or high-calorie diets can lead to significant changes in neuronal activity, synaptic plasticity, and functional connectivity within thalamic circuits. These dietary influences may disrupt the normal communication between the thalamus and hypothalamus, a key pathway responsible for appetite regulation, hormonal signaling, and metabolic homeostasis. Such disruptions can impair satiety signaling and enhance food-seeking behavior, increasing susceptibility to overeating and progressive weight gain.

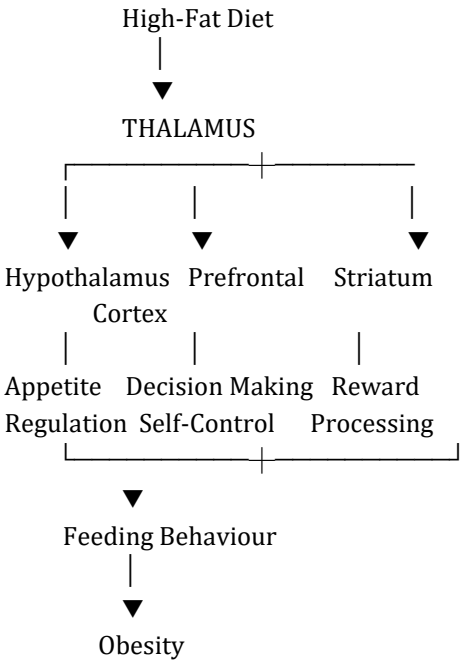

Figure 1: Neural network regulating feeding behavior.

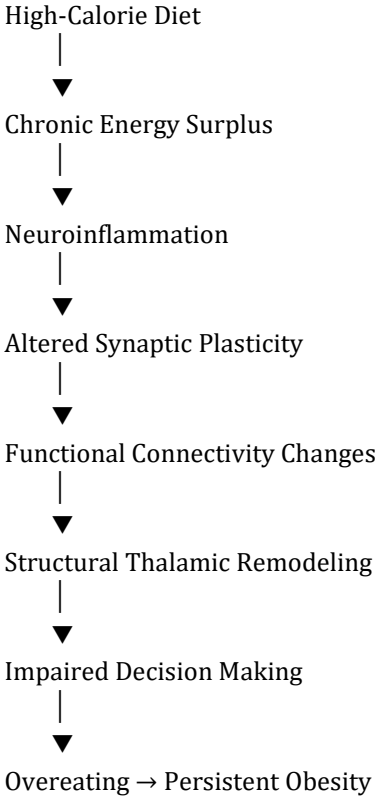

Figure 2: Neuroplastic changes during obesity.

In addition, diet-induced obesity has been linked to neuroinflammatory responses and altered neurotransmitter signaling within the central nervous system. Increased inflammatory markers and changes in dopamine and leptin signaling may further affect thalamic processing of reward and hunger-related cues. Over time, these neural adaptations can reinforce maladaptive eating behaviors and reduce sensitivity to natural satiety signals **[Figures 1, 2].**

Brain Region	Primary Function	Obesity-Related Changes	Clinical Consequence
Thalamus	Sensory and metabolic information relay	Altered activity and connectivity	Impaired appetite regulation
Hypothalamus	Energy homeostasis	Disrupted hormonal signaling	Reduced satiety
Prefrontal Cortex	Executive control	Reduced inhibitory control	Poor dietary decisions
Striatum	Reward processing	Increased food reward sensitivity	Food craving
Limbic System	Emotion and motivation	Enhanced emotional eating	Excess caloric intake

Table 1: Brain regions involved in diet-induced obesity.

In obesity, these changes are not limited to peripheral metabolic dysfunction but also involve widespread central nervous system remodeling. The thalamus, particularly through its connections with reward-related regions such as the striatum and prefrontal cortex, as well as homeostatic centers like the hypothalamus, acts as a critical hub integrating metabolic state and behavioral responses to food intake.

Furthermore, neuroimaging studies suggest that individuals with obesity may show altered thalamic volume and functional activity, indicating long-term neuroplastic changes associated with sustained dietary imbalance. These alterations may contribute to impaired decision-making, increased impulsivity toward food rewards, and difficulty in regulating eating behavior **[Table 1].**

Overall, the evidence highlights that obesity is not solely a metabolic disorder but also a complex neurobehavioral condition. Diet-induced changes in thalamic function and its broader neural networks play a significant role in sustaining excessive food intake and maintaining the obese state.

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