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OBESITY AND EXECUTIVE FUNCTIONS: THE CRITICAL ROLE OF THE PREFRONTAL CORTEX – A LITERATURE REVIEW

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ABSTRACT

Introduction: Obesity is a chronic metabolic disease characterized by excessive fat accumulation and a multifactorial etiology. One potential contributing factor is impaired executive functioning in individuals with obesity, including deficits in planning, decision-making, and inhibitory control. These impairments may lead to increased impulsivity and difficulties with self-regulation in the context of eating behavior, ultimately contributing to excessive body weight and challenges in achieving weight reduction. The primary brain region responsible for executive functions is the prefrontal cortex.

Aim of the study: This review aims to synthesize recent research on the relationship between obesity and executive functions, with particular focus on the prefrontal cortex as both a key modulator of these cognitive processes and a potential target for therapeutic interventions in the management of excessive body weight.

Methods: This review was compiled through an extensive analysis of the PubMed and Google Scholar databases. It includes a literature review from [insert year range], with particular emphasis on articles from the past 5 years focusing on the keywords: „obesity”, „prefrontal cortex”, „executive function”, „cognitive function”.

Conclusions: Obesity represents a growing health problem with significant clinical and social consequences. Alterations in the prefrontal cortex observed in individuals with obesity may lead to impairments in executive functions, such as reduced impulse control and decision-making difficulties. These dysfunctions can contribute to the development of obesity, while obesity itself may further exacerbate them, creating a vicious cycle. Therefore, interventions aimed at enhancing executive functions could serve as a valuable complement to obesity treatment. However, further consistent research is needed to determine their effectiveness and practical applicability.

KEYWORDS

Obesity, Prefrontal Cortex, Executive Function, Cognitive Function

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Introduction

Obesity constitutes one of the most pressing public health challenges of the contemporary era, with projections indicating a continued rise in its prevalence and associated comorbidities, thereby imposing a growing burden on healthcare systems, economies, and societies worldwide (1). Although body mass index (BMI) remains a commonly used tool for assessing obesity, it presents notable limitations, as it may both underestimate and overestimate actual adiposity and offer adequate information about an individual's metabolic or functional health. As a result, contemporary scientific and clinical societies increasingly define obesity as a chronic disease characterized by excess adipose tissue that impairs the normal function and structure of organs and systems. This pathological accumulation of adipose tissue can lead to organ dysfunction, resulting in serious and potentially life-threatening complications, such as arterial hypertension, myocardial infarction, stroke, certain types of cancer, as well as psychiatric and neurological disorders (2,3). Therefore, identifying and understanding the pathophysiological mechanisms underlying obesity is crucial for the development and implementation of effective treatment strategies(4).

One of the causes of the epidemic of excessive weight gain is considered to be dysregulation of executive functions, which are regulated by the central nervous system, primarily by the prefrontal cortex. Executive function refers to higher-order cognitive processes such as working memory, planning, decision-making, and inhibitory control, which are involved in top-down (i.e., non-stimulus-driven) regulation of human behavior (4).

The prefrontal cortex (PFC), located in the anterior region of the frontal lobes, is believed to play a role in a range of advanced cognitive abilities referred to as executive cognitive control, behavioral inhibition, or general intelligence. Executive functions refer to the integrated activity of multiple cognitive neural processes that enable flexible achievement of current goals. Examples of these functions include planning, judgment, decision-making, anticipation, and reasoning. Effective judgment, appropriate decision-making, and timely responses require a top-down regulatory mechanism that coordinates and modulates various neural processes

in a flexible and controlled way (5,6). The aim of this review is to provide an overview of the latest research on the relationship between obesity and executive functions, with particular emphasis on the key role of the cortex as a modulator of these functions and as a potential target for interventions in the treatment of excessive body weight.

Obesity and executive functions

Previous studies have indicated a potential link between overweight/obesity and impairments in executive functioning. In a comprehensive meta-analysis, Yang et al. examined 72 studies involving a total of 4,904 overweight or obese participants to identify which executive functions are particularly vulnerable to the effects of excess weight. The analysis revealed that individuals with obesity exhibited broad impairments across multiple domains of executive functioning. While some impairments are also observed in individuals who are overweight, they tend to be more limited in scope. Notably, these cognitive difficulties are not confined to a specific age group but are observed throughout the lifespan—from early childhood to late adulthood. Variability in findings across studies may be attributed to methodological differences, including the diversity and complexity of tasks used to assess executive functions. To clarify these relationships and identify potential mechanisms, further targeted research is warranted (7).

Studies indicate that individuals with morbid obesity exhibit globally reduced executive functioning compared to those with a normal body weight. In a study by La Marra et al. (2022), 208 outpatients were assessed: 52 with overweight, 76 with obesity, 80 with morbid obesity (BMI > 40), and 96 individuals with normal weight serving as the control group. The results demonstrated that individuals with morbid obesity showed markedly poorer performance across various cognitive domains, with the most pronounced impairments observed in psychomotor speed and cognitive flexibility. Such deficits may carry significant clinical consequences, potentially contributing to challenges in decision-making and behavioral regulation (8).

Deficits in executive functioning are also observed in eating disorders that may contribute to excessive weight gain, such as binge eating disorder—a condition in which an individual consumes large amounts of food within a short period of time while experiencing a loss of control over their eating behavior. The study by Brucar et al. identifies three domains that characterize the underlying mechanisms of binge eating, one of which involves cognitive dysfunction—specifically impulsivity as well as both insufficient and excessive control—contributing to a maladaptive cycle: undercontrol results in challenges in suppressing impulses to partake in gratifying yet detrimental behaviors, heightening the risk of losing mastery over eating and binge eating, in the same time overcontrol has been shown to elevate the probability of subsequent binge episodes, perpetuating a detrimental cycle of restriction and bingeing. Research on the impairment of executive functions as one of the causes of binge eating may help tailor behavioral and pharmacological treatment strategies for this dysfunction, and consequently improve obesity treatment (9).

Cognitive impairments in individuals with obesity can also be compared to those observed in individuals with anorexia, representing two extremes of eating disorders. The research by Foldi et al. demonstrate that individuals with obesity and those with anorexia display impairments in decision-making and cognitive flexibility. However, the nature of these impairments differs in opposite directions—people with obesity tend to struggle with impulsivity and poor control over their eating behaviors, whereas individuals with anorexia demonstrate excessive control (10).

Research by Syan et al. demonstrated that obese individuals may experience impairments in brain activity. Conducted on a group of young adults, the study compared obese individuals with a control group of lean participants during a set of neurocognitive tasks. Brain activity and structural changes in the cortex were examined using fMRI. Obese individuals scored significantly lower on cognitive performance tests. Moreover, those with excessive body weight showed abnormal patterns of brain activation — increased activity in regions associated with the default mode network during tasks requiring focus, indicating difficulties with its proper suppression. The authors emphasize that deficits in cognitive control and inefficient deactivation of the default mode network may contribute to disordered eating behaviors and the persistence of obesity, highlighting the need for further research and the inclusion of these aspects in treatment strategies (11).

Executive Dysfunction and the Prefrontal Cortex

The prefrontal cortex is a key brain region that affects dietary choices. Impairments in impulse control, inhibitory reactions, and self-regulation can result in excessive weight gain and, ultimately, obesity in today's world, where access to fatty and energy-dense meals is almost limitless (4).

Through neuroimaging techniques and an enhanced understanding of neurobiological alterations in individuals with obesity, it is possible to identify specific brain regions that exhibit changes in obese individuals compared to those with normal body weight. Research indicates that regions within the prefrontal cortex and the limbic system are particularly significant, especially with regard to impulse control, which constitutes a critical challenge in individuals suffering from excessive body weight. Studies indicate that obesity correlates with generalized hypoactivity in many parts of the prefrontal cortex, including the medial prefrontal cortex, orbitofrontal cortex, and supplementary motor area. This hypoactivity may lead to difficulties in controlling impulsive responses, disturbances in food perception, and impaired inhibition. Similar changes have been observed in individuals with substance use disorders. Additionally, data relating the lateral prefrontal cortex are inconsistent—some research reveal hypoactivity, while others indicate hyperactivity—potentially reflecting instability in attitudes linked to food control among patients with obesity (12).

The study by Takehana et al. indicates that healthy food choices are associated with self-regulatory and self-control mechanisms that favor long-term rewards over immediate gratification, with regulatory processes in the prefrontal cortex playing a key role in this preference. During functional MRI scanning, participants completed tasks assessing both food-related decision-making and working memory. Self-control was measured behaviorally through intertemporal choice, evaluating the degree to which individuals discounted delayed monetary rewards. Individuals who prioritized the health value of food exhibited increased activation in the superior, dorsolateral, and medial regions of the prefrontal cortex. The study, however, shows that working memory function is not impaired in individuals with obesity (13).

The study by Coquery et al., conducted on a sample of 50 healthy young women, revealed significant associations between eating behaviors and brain function, particularly within prefrontal cortex regions. Using functional magnetic resonance imaging (fMRI), the research identified differences in both activity and functional connectivity among brain areas involved in reward processing and motivation (striatum), as well as regions responsible for hedonic evaluation and cognitive control (orbitofrontal and prefrontal cortices). Participants with a preference for Western-style diets exhibited reduced functional connectivity between reward-related areas and the prefrontal cortex. These findings indicate that even among healthy individuals, distinct dietary patterns are linked to variations in neuropsychological mechanisms, which may serve as early indicators of vulnerability to eating disorders and other health complications (14).

Obesity as a Factor Affecting the Structure and Function of the Prefrontal Cortex

Studies have shown structural and functional changes, as well as disrupted neurobiological mechanisms in the brain—particularly in the prefrontal cortex—among individuals with obesity compared to those with normal body weight. Studies over the past decade have consistently demonstrated reductions in prefrontal cortex cortical thickness in individuals with obesity compared to those with normal body weight. Chen et al. conducted a meta-analysis of 25 studies, and in an analysis including over 7,600 participants, they found that a higher body mass index (BMI) was significantly associated with reduced gray matter volume in the right orbitofrontal cortex (Brodmann areas 10 and 11). Further analysis using the Neurosynth Network Coactivation method showed that this region is functionally co-activated with other areas involved in cognitive control, including the left medial prefrontal cortex and the right frontal pole. Although functional imaging studies have not always demonstrated consistent associations between orbitofrontal cortex activation and obesity, these findings underscore the importance of orbitofrontal cortex structural integrity in neurobiological models of obesity (15).

Changes in prefrontal cortical thickness have also been observed in children and adolescents. (16,17). A two-year follow-up study within the NIH-funded voxel-based morphometry (VBM) to evaluate gray matter volume in 258 children with obesity and 265 with normal body weight. Over time, children with obesity demonstrated more pronounced reductions in gray matter volume, especially in regions implicated in executive functioning and reward-related processing, compared to their normal-weight counterparts. Notably, BMI showed a consistent negative association with frontal lobe gray matter volume at both assessment points. These

results point to ongoing structural brain changes in childhood obesity, particularly affecting neural circuits responsible for self-regulation, decision-making, and motivation (16).

Functional neuroimaging (fMRI) has revealed a set of characteristic alterations in the brains of individuals with obesity, which may contribute to the persistence of maladaptive eating behaviors and difficulties in body weight regulation. These findings include increased activation of reward-related brain structures—such as the striatum, orbitofrontal cortex, insula, and amygdala—in response to food cues. This pattern suggests an enhanced motivational drive to eat ("wanting"), despite a potential reduction in the actual hedonic experience of food consumption ("liking"). In parallel, reduced activity in brain regions involved in cognitive control, particularly the dorsolateral prefrontal cortex (DLPFC), has been observed. This may impair inhibitory control and self-regulation related to eating behavior. Moreover, studies have identified both structural and functional brain alterations, including decreased gray matter volume in areas responsible for appetite regulation and decision-making, as well as disrupted functional connectivity between these regions (18).

Neuroimaging studies suggest that individuals with obesity process food-related stimuli differently than those with normal weight. The hypothalamus shows reduced and delayed activation in response to food intake, while reward-related regions — such as the striatum and orbitofrontal cortex — exhibit heightened responses to visual cues of high-calorie foods. During actual consumption, however, the reward response is blunted, likely due to decreased dopamine receptor availability. In parallel, reduced activation of the prefrontal cortex, responsible for impulse control, may impair self-regulation. This pattern of enhanced cue reactivity, diminished reward from consumption, and weakened cognitive control resembles neural mechanisms seen in addiction and may contribute to overeating and sustained weight gain (19).

Among individuals affected by obesity, distinct alterations in the activation of specific prefrontal cortex regions have been documented in response to hunger states and food-related stimuli. Increased activation of the left prefrontal cortex and parahippocampal gyrus was observed during hunger, along with enhanced activation of the anterior cingulate cortex (ACC), which integrates reward signals with cognitive control and may be involved in the conflict between the desire to eat and appetite regulation. Additionally, lateralization of function was demonstrated within the dorsolateral prefrontal cortex (DLPFC)—with decreased activity on the left side, associated with anticipation and regulation of appetite, and increased activity on the right side, responsible for impulse control and attention. These findings suggest a disrupted balance between anticipatory and immediate control, which may contribute to difficulties in inhibiting eating behaviors. Overall, the data confirm a complex interaction of deficits in somatosensory perception, memory, attention, and cognitive control systems in individuals with obesity, supporting the concept of obesity as a disorder with addiction-like features, albeit involving mechanisms distinct from classical addiction (20).

Obesity and neurobiological mechanisms

Research indicates that obesity may contribute to the impairment of executive functions through neurobiological pathways, including chronic inflammation and neurodegenerative processes. Both clinical studies in obese individuals and experimental mouse models have demonstrated that excessive adipose tissue leads to increased secretion of pro-inflammatory cytokines such as interleukin-1 β (IL-1 β), interleukin-2 (IL-2), interleukin-6 (IL-6), interferon-gamma (IFN- γ), and tumor necrosis factor-alpha (TNF- α), thereby promoting a persistent state of systemic inflammation. Chronic inflammation impairs the blood-brain barrier by causing endothelial cell dysfunction, which triggers neuroinflammation and increases oxidative stress, ultimately leading to cognitive decline (21). Research has demonstrated a negative correlation between obesity, serum levels of C-reactive protein (CRP)—a well-established biomarker of systemic inflammation—and cognitive impairment. The longitudinal study titled *Adiposity, Inflammation, and Working Memory: Evidence for a Vicious Cycle* assessed over 8,000 children and adolescents aged 9 to 15 years. The findings revealed that fat mass predicted subsequent decline in working memory, with this relationship being statistically mediated by CRP levels. Furthermore, poorer working memory predicted increases in fat mass and CRP over time, with the association between working memory and later CRP partially mediated by fat mass. These results suggest the presence of a vicious cycle wherein adiposity, inflammatory activity, and working memory impairment mutually reinforce each other longitudinally (22). The study by Binnuri et al., conducted on young and mature female mice, demonstrated that both age and how long obesity lasts play key roles in obesity-related brain damage, emphasizing the importance of early intervention to protect brain health. . Prolonged obesity led to widespread brain inflammation, activation of glial cells, and a reduction in protective brain

factors—effects linked to increased neurodegenerative risk. Notably, adult mice showed these negative brain changes even after a shorter period of obesity (23).

The brain of an individual with obesity may exhibit impaired responsiveness to metabolic signals, including leptin and insulin resistance. This dysregulation of appetite signaling contributes to reduced satiety perception and promotes overeating. Leptin is a hormone primarily secreted by adipose tissue that plays a key role in regulating energy balance by signaling satiety to the hypothalamus and suppressing appetite. In obesity, despite elevated leptin levels, a state of leptin resistance often develops, impairing the hormone's ability to regulate appetite and body weight effectively (24).

Associations between leptin levels and neuronal activity in specific brain regions are increasingly being investigated, as this relationship may suggest that the metabolic state of individuals with obesity influences the brain's reward system, thereby affecting eating behavior and body weight. In a study involving 44 women (14 with obesity and 30 with normal weight), we examined whether resting-state connectivity between the ventromedial prefrontal cortex (vmPFC) and other brain regions differs according to body weight and whether it can predict weight changes over 8 months. The participants with obesity underwent bariatric surgery. It was found that connectivity between the vmPFC and both the ventral striatum and dorsolateral prefrontal cortex was associated with obesity, but only the vmPFC–ventral striatum connection predicted weight changes. Changes in this connectivity were also linked to a decrease in leptin levels, suggesting that hormonal regulation may influence the brain's reward system, thereby affecting eating behavior and body weight (25).

Impaired metabolic function has been identified as a significant factor connecting elevated BMI levels, HOMA-IR (Homeostatic Model Assessment of Insulin Resistance - an index used to assess insulin resistance, which refers to the reduced sensitivity of cells to the action of insulin) and leptin to poorer neurocognitive outcomes and with an increased risk of Alzheimer's disease and related dementias (ADRD) (25, 26).

Obesity and overweight may play an instrumental part in the neuronal viability deficiencies observed in individuals with type 2 diabetes and may be among the main factors linking insulin resistance to the impaired cognitive abilities of the diabetic brain(27). The results of the study conducted by Kullmann et al. suggest that central insulin action in the brain may modulate appetite control and promote weight loss by modifying functional connectivity between brain regions responsible for cognitive and homeostatic regulation(28).

Metabolic dysfunction, including insulin and leptin resistance, is regarded as a potential mediating mechanism linking elevated BMI to neurocognitive impairment; however, empirical evidence remains inconsistent. The study by Gundogdu et al. evaluated executive function (EF), behavioral and emotional problems (BE), and quality of life (QoL) in adolescents with obesity, compared to a weight-matched control group, also investigating whether insulin resistance (IR) was associated with those issues. While IR was found to lower QoL, it was not directly linked to deficits in executive function or emotional–behavioral problems. (29).

The Prefrontal Cortex as a Moderator and Potential Target for Interventions

In the current model of obesity treatment, it is important to recognize that this is a multifactorial disease; therefore, a multidisciplinary approach is recommended, combining various strategies, including pharmacological, behavioral, and even surgical interventions. One of the goals of therapy may be targeting the prefrontal cortex and simultaneously improving executive functions through neuromodulation, cognitive training, mindfulness training, and cognitive-behavioral therapy.(30).

Obesity treatment also has neuroplastic potential – postoperative studies indicate that surgical interventions (e.g., bariatric surgery) can partially restore normal activity and connectivity in brain regions involved in the control of eating(18).

In the review by da Silva Lira -Batista et al., the impact of neuromodulation techniques such as tDCS and TMS on executive functions and food craving in individuals with overweight was assessed. In groups undergoing active neuromodulation, a reduction in food cravings and an improvement in executive functions were observed, suggesting that this is a promising technique for supporting obesity treatment by enhancing control over food intake. However, many of the included studies were short-term and had small sample sizes – larger, long-term randomized controlled trials (RCTs) are needed to determine its lasting effectiveness (31).

Physical activity has long been recognized as a way to improve brain blood flow and support neurogenesis, reducing inflammation, which consequently leads to improved executive functions. (32). A small-sample study comparing the effects of an intervention combining aerobic and resistance exercises on executive functions in obese adolescents showed that physical exercise significantly improved their executive functioning. Different components of executive function improved at different times during the intervention

— changes in inhibition and cognitive flexibility appeared after 12 weeks, while improvements in working memory were observed after 14 weeks(33). Research shows that in postmenopausal women with obesity (who are particularly at risk for cognitive impairment and health problems), a combined intervention of dietary changes and physical exercise provides greater health benefits, including improvements in cognitive function, compared to the control group and to groups using diet-only or exercise-only interventions(34).

The most commonly used intervention for obesity and the most well-established treatment for binge eating disorder is cognitive-behavioral therapy (CBT), which may be the first line of treatment among psychological methods. Research shows that prefrontal cortex recruitment during response inhibition may be enhanced by cognitive-behavioral therapy targeting impulsive eating behavior (35). CBT can also be a beneficial support for weight loss; however, it may not be sufficient for the long-term maintenance of the results. Consequently, it is essential to incorporate this therapy with other treatment strategies, such as holistic nutritional education (36).

Meditation, including one of its branches—mindfulness (practice of focused awareness)—can support cognitive functions, enhance stress resilience, and improve working memory, in part by strengthening the prefrontal cortex, increasing its functional connectivity, and enhancing gray matter volume(37). In the context of obesity, mindfulness may be a useful tool for modifying eating behaviors; however, there is a lack of convincing evidence for its effectiveness in reducing body weight (38). Another way to influence prefrontal cortex function in the context of obesity and excessive food intake may be through cognitive function training. Research shows that cognitive training yields beneficial effects on eating behaviors and weight reduction; however, the outcomes depend on the type of training, the nature of the stimulus, and the research approach. An additional limitation of these studies is their focus on short-term outcomes in individuals with normal body weight, making it difficult to generalize the results—particularly to people with obesity—and to assess the long-term effects of such training(39).

Conclusions

Obesity is one of the most significant and expensive health problems of our time, affecting people across all age groups. The number of individuals with obesity continues to rise, leading to an increasing prevalence of comorbidities and a decline in quality of life. Therefore, a thorough understanding of the pathophysiology of obesity, along with the development of appropriate treatment standards, is essential for both current and future populations. Numerous studies have shown alterations in the prefrontal cortex in individuals with obesity or those predisposed to it, including reduced cortical thickness, hypoactivation, and decreased connectivity with other brain regions, which may result in impaired executive functions. Executive functions in obesity have a dual nature – a tendency toward impulsivity and a lack of inhibitory control may contribute to excessive body weight, while obesity itself can further impair executive functions, making weight loss more difficult. As a result, a vicious cycle may develop. Interventions aimed at improving executive functions could therefore serve as a valuable complement to obesity treatment; however, there is currently a limited amount of data and inconsistent research findings on this topic.

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