Opinion

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Neurobiology of Eating Disorders: Mechanisms of Dysregulated Appetite and Reward

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Introduction

Eating disorders such as anorexia nervosa, bulimia nervosa and binge eating disorder represent some of the most challenging psychiatric conditions, characterized by severe disturbances in eating behaviors, body image and emotional regulation. While social and psychological factors play pivotal roles in their onset and progression, increasing research highlights the neurobiological underpinnings that shape vulnerability to these disorders. The central nervous system is responsible for regulating appetite, food intake and energy balance and disruptions in these mechanisms can significantly alter eating patterns. Neurobiological studies reveal that eating disorders are not merely issues of willpower or self-control but are rooted in alterations within complex brain circuits involving reward, motivation and stress response. Neural pathways that integrate signals from hormones, neurotransmitters and sensory cues contribute to the perception of hunger and satiety. In eating disorders, these circuits become dysregulated, leading to either excessive restriction of food or uncontrollable episodes of overeating. These findings underscore the need to reframe eating disorders as brain-based conditions with biological as well as psychosocial dimensions [1].

Description

The regulation of appetite is orchestrated by a network of hypothalamic and cortical structures that respond to peripheral signals from hormones such as leptin, ghrelin and insulin. In healthy individuals, these signals maintain energy balance by stimulating or suppressing food intake. However, in individuals with eating disorders, these regulatory pathways are often disrupted. For example, patients with anorexia nervosa frequently exhibit altered ghrelin and leptin signaling, leading to impaired hunger perception and a paradoxical resistance to weight gain. Conversely, binge eating disorder is associated with heightened responsiveness to ghrelin and attenuated satiety signaling, promoting excessive food intake. Neuroimaging studies also indicate structural and functional changes in the hypothalamus of individuals with eating disorders, suggesting long-term alterations in how the brain processes hunger cues. This dysregulation of appetite regulation underscores the biological complexity of eating disorders and their persistence despite conscious attempts at behavioral change [2].

Beyond the hypothalamus, reward-related brain circuits play a central role in the development and maintenance of disordered eating. The mesolimbic dopamine system, particularly the nucleus accumbens and ventral tegmental area, is responsible for encoding the rewarding properties of food. In binge eating disorder and bulimia nervosa, hyperactivation of these pathways leads to exaggerated reward responses to palatable, high-calorie foods, reinforcing compulsive overeating behaviors. In contrast, individuals with anorexia nervosa often exhibit blunted dopamine responses, deriving less pleasure from food consumption, which may reinforce restrictive eating patterns. Functional MRI studies show that these individuals experience heightened reward responses to weight loss or food avoidance, further perpetuating pathological behaviors. This imbalance between food-related and non-food-related reward pathways highlights how altered neural reinforcement mechanisms contribute to the persistence of disordered eating behaviors [3].

The role of neurotransmitters, particularly dopamine and serotonin, further illuminates the neurobiology of eating disorders. Dopamine, as noted, regulates motivation and reward processing, while serotonin influences mood, satiety and impulse control. In anorexia nervosa, elevated serotonin activity

has been linked to heightened anxiety and obsessionality, contributing to rigid dietary control and fear of weight gain. In contrast, binge eating disorder is often associated with impaired serotonin signaling, leading to impulsive food consumption and poor satiety regulation. Altered glutamatergic and GABAergic transmission has also been implicated, suggesting that dysregulation extends beyond classical monoamine systems. These neurotransmitter imbalances may explain the high comorbidity between eating disorders and mood or anxiety disorders, as well as the limited effectiveness of traditional pharmacological treatments targeting serotonin alone. Understanding these neurochemical disruptions paves the way for novel therapeutic strategies that more effectively address the underlying neurobiology [4].

Stress and emotion-related brain circuits also contribute significantly to disordered eating. The amygdala, insula and prefrontal cortex are involved in processing emotions, interoceptive awareness and cognitive control over eating behaviors. Dysregulated activity in these regions is consistently observed in individuals with eating disorders, suggesting impaired integration of emotional and physiological cues. For instance, the insula, which mediates the perception of internal bodily states, often shows altered connectivity in anorexia nervosa, contributing to distorted body image and impaired hunger awareness. Stress-induced activation of the hypothalamic-pituitary-adrenal (HPA) axis can further exacerbate binge eating or restriction, as cortisol influences appetite and reward sensitivity. These findings demonstrate that eating disorders arise from a convergence of dysregulated appetite signals, maladaptive reward processing and impaired emotional regulation, making them highly resistant to simple behavioral modification [5].

Conclusion

The neurobiology of eating disorders reveals a complex interplay of disrupted appetite regulation, altered reward sensitivity, neurotransmitter imbalances and stress-related brain activity. Far from being purely psychological conditions, these disorders are deeply rooted in neurobiological mechanisms that distort how individuals perceive hunger, satiety and food-related reward. Anorexia nervosa, bulimia nervosa and binge eating disorder each reflect unique patterns of dysregulation across hypothalamic, limbic and cortical networks, explaining their persistence and resistance to conventional treatment. By deepening our understanding of these brain-based mechanisms, researchers and clinicians can move toward more targeted, effective therapies that integrate neurobiological insights with psychological and social interventions. Ultimately, this neurobiological perspective underscores the need to view eating disorders as complex, multifactorial illnesses requiring comprehensive and compassionate care.

Acknowledgment

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Conflict of interest

None.

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