Exploring the overlap between eating disorders and addiction disorders: implications for treatment

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Abstract

Eating disorders (EDs) are complex psychiatric conditions characterised by disturbances in eating behaviours and associated psychological impairments. Despite being traditionally distinct from addiction disorders, increasing evidence highlights significant overlaps in their neurobiological and behavioural underpinnings. This article explores the potential for EDs to be conceptualised, in part, as addiction disorders. It examines mechanisms such as the activation of dopamine reward pathways, endorphin release and the anxiolytic effects of starvation-induced ketosis, which may create addictive processes within EDs. Behavioural parallels, including impulsivity, compulsivity and neurochemical alterations in bulimia nervosa (BN) and binge eating disorder (BED), further align these disorders with substance use disorders. Additionally, food and exercise addiction, often comorbid with EDs, are evaluated in the context of their shared neural circuits and psychosocial drivers. Distinctions between EDs and addictions are also discussed, highlighting differences in aetiology, reward mechanisms, social acceptability and recovery dynamics. Understanding EDs through the lens of addiction has profound implications for treatment, including the integration of addiction-focused therapeutic strategies and pharmacological interventions targeting dopaminergic and opioid pathways. This framework offers new insights into the aetiology, psychopathology and treatment of EDs, with the potential to improve outcomes for individuals suffering from these life-threatening disorders.

Introduction

Key points

1. EDs share significant neurobiological overlap with addiction disorders
2. Starvation triggers homeostatic adaptations that may produce anxiolytic and euphoric effects
3. Impulsivity in BN and BED mirror behaviours observed in addiction disorders
4. Distinctions between EDs and addiction are discussed
5. Viewing EDs as addiction disorders informs developing therapies and treatment approaches

Eating disorders (EDs) are a group of conditions characterised by persistent disturbances in eating behaviours and associated psychological impairment. EDs have high mortality rates and are frequently comorbid with other psychiatric disorders, including addiction (1).

Anorexia nervosa (AN) is defined by the restriction of energy intake, leading to significantly low body weight, accompanied by an intense fear of weight gain and disturbances in the perception of one's body shape or weight. Bulimia nervosa (BN) is characterised by recurrent episodes of binge eating followed by inappropriate compensatory behaviours, such as self-induced vomiting, excessive exercise or the misuse of diuretics and laxatives. Binge eating disorder (BED) shares the binge eating component of BN but lacks compensatory behaviours (2).

The cognitive-behavioural theory of EDs suggests that overvaluation of shape and weight is central to ED psychopathology. Consequently, behaviours such as food restriction, exercise, purging and bingeing are seen as manifestations of these disordered cognitions.

Goodman defined addiction as a process in which a behaviour provides both pleasure and relief from negative affect. This behaviour is characterised by a recurrent inability to control or stop it, despite negative consequences (3).

The DSM-5 introduced significant changes to the categorisation of addictions, renaming the Substance-Related Abuse and Dependency category as Substance-Related and Addictive Disorders with two subdivisions: substance-related disorders and non-substance-related disorders. The latter, currently exemplified only by gambling disorder, encompasses addictive behaviours that do not involve the ingestion of psychoactive substances (2).

The co-occurrence of EDs and addiction has been a topic of interest in psychiatry. Compared to the general population prevalence of addiction (approximately 9%), studies suggest that up to 50% of individuals with an ED also abuse or depend on substances such as alcohol or psychoactive drugs (4). Furthermore, 35% of individuals diagnosed with a substance use disorder (SUD) report ED behaviours, compared to 5% of women in the general population (5).

This article explores whether certain ED behaviours may stem from behavioural addiction or induce physiological changes that mimic substance dependence, producing withdrawal symptoms and physical dependence. Mechanisms such as dopamine (DA)-mediated reward pathways, endogenous opioid activation (e.g., through purging or

exercise) and the GABAergic effects of starvation ketosis (mediated by beta-hydroxybutyrate (BHB)) are discussed. These insights could enhance our understanding of ED aetiology, psychopathology and treatment.

Is starvation addictive?

Dysfunctional reward processes have been proposed as a basis for the compulsive behaviours observed in AN. Imaging studies and animal models provide evidence for this hypothesis (6). Bergh et al. suggested that starvation and overactivity elevate glucocorticoid hormones released from the adrenal grand in response to stress which may induce euphoria through DA pathway activation (7).

Starvation-induced ketosis produces BHB, a ketone body produced from the breakdown of fats in the liver in response to glucose scarcity, to serve as an alternative fuel source for the brain. BHB has anxiolytic and euphoric effects mediated through modulation of the inhibitory neurotransmitter GABA (8). These effects may explain why individuals with AN report a sense of calm or euphoria during prolonged fasting, reinforcing starvation behaviours.

Alterations in the brain's reward systems also contribute. Wagner et al. and Park et al. demonstrated structural and functional abnormalities in reward-related brain regions in individuals with AN. These alterations shift initial food restriction from being positively reinforcing to becoming habitual and negatively reinforced through maladaptive cognitive control and excessive habit formation (6, 9).

Chronic stress and the hypothalamic-pituitary-adrenal (HPA) axis also play roles. Corticotropin-releasing hormone, the primary regulator of the stress response, is released excessively following dysregulation of the HPA axis. This reduces appetite and enhances the drive for food avoidance. Starvation-induced hormonal shifts, including increased glucocorticoids and reduced insulin sensitivity, further reinforce altered reward pathways (10, 11).

Impulsivity and dopaminergic neural circuits

Impulsivity, a multidimensional construct, has been implicated in BN and associated with psychiatric complications such as SUDs and personality disorders (12). Impulsivity correlates with DA imbalances in the frontal cortex and striatum, impairing cognition, self-regulation and planning (13).

In BN, disruptions in striatal DA and reduced dopamine D2 receptor availability have been observed. Broft et al. reported significant differences in DA response between individuals with BN and healthy controls, with greater binge-purge frequency correlating with reduced DA response (14).

Food addiction

Food addiction, characterised by compulsive overconsumption of highly palatable foods (rich in sugar, fat and salt), parallels substance addiction in its neurobiological underpinnings (15). Neuroimaging studies show that such foods activate DA reward pathways (16). The Yale Food Addiction Scale (YFAS) assesses food addiction based on DSM-5 addiction criteria (15).

Meule et al. found that all BN participants in their study met YFAS food addiction criteria, while remission of BN corresponded to a remission of addictive behaviours. Furthermore, a study conducted by Cassin et al. found that 92% of participants with BED also met the DSM-5 criteria for substance addiction (17). Recent reviews highlight marked parallels between overconsumption of highly palatable foods and SUDs in terms of DA, opioid, acetylcholine and serotonin pathways (18). While phenotypic overlap exists, this does not necessarily indicate that both disorders are identical. Evidence is lacking to support the translation of all addiction criteria to food addiction and further research is needed.

Exercise addiction

Exercise addiction, with a comorbidity rate of up to 48% in EDs, is characterised by compulsive, excessive exercise often driven by weight-loss motivations or avoidance of negative affect (19). High-intensity exercise releases endogenous opioids, creating a euphoria that can lead to "auto-addiction" (20). Rodent studies suggest that exercise may also impair reward circuitry, reducing the hedonic effects of other rewarding stimuli (21, 22).

Distinctions between eating disorders and addictions

1. **Aetiology**

   EDs, particularly AN and BN, are underpinned by disturbances in body image and weight-related beliefs, absent in traditional addiction models.

2. **Immediate versus sustained reward**

   Addictions involve acute, immediate reward, while restrictive EDs rely on prolonged reinforcement. BN and BED share impulsive traits resembling addiction (23).

3. **Social acceptability**

   Pursuit of thinness in EDs often garners social approval, whereas substance addiction frequently leads to social rejection.

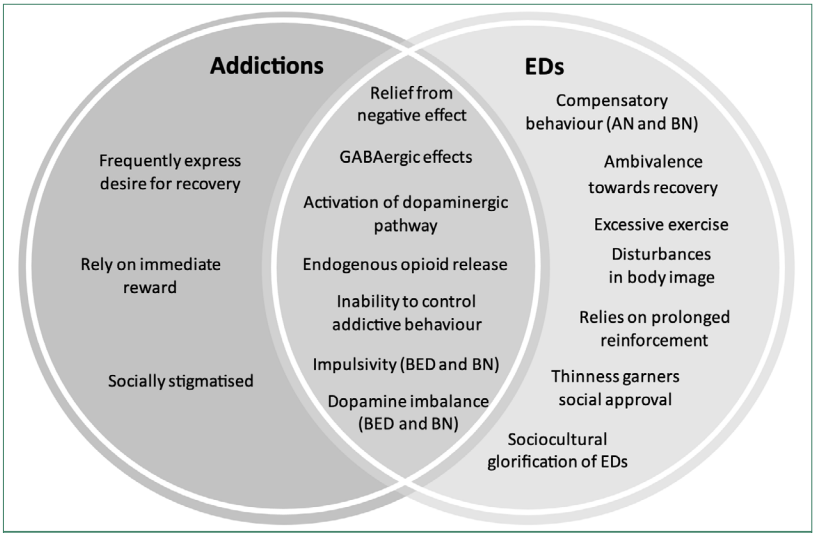

Figure 1. Conceptual model of overlap between addiction disorders and eating disorders

4. Recovery

Individuals with EDs often exhibit ambivalence toward recovery, contrasting with the abstinence-focused goals in addiction recovery (24).

5. Sociocultural factors

Diet culture and the glamourisation of thinness within Western society often aid in the development of ED ideals from a young age, while addictions are often cautioned against (25).

Treatment implications

Conceptualising EDs as addictions could inform novel treatment approaches. Addiction-focused interventions such as DA-targeted pharmacotherapy or opioid antagonists like naltrexone may hold promise for ED-related behaviours (26).

Enhanced cognitive behavioural therapy remains the first-line treatment for EDs, but integrating SUD-focused modules (e.g., reward sensitivity and impulsivity management) could improve outcomes in dual-diagnosis cases (27).

Self-help programs, such as Overeaters Anonymous, provide structured recovery frameworks, though risks of pathological competition among ED patients in group settings must be carefully managed (24).

Conclusion

While EDs and addictions are distinct entities, significant overlaps in neurobiology and behaviour suggest a continuum. Conceptualising EDs within an addiction framework could deepen our understanding of their aetiology and psychopathology, offering new directions for treatment and management of these complex disorders.

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