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Eating Disorders and Substance Use-Is There a Relationship?

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Editorial

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Abbreviations: ED: Eating Disorders; AN: Anorexia Nervosa; BN: Bulimia Nervosa; SUD: Substance Use Disorders; ASAM: American Society of Addiction Medicine; DBT: Dialectical Behavior Therapy.

Editorial

Eating disorders (ED) are relatively rare among mental disorders, with a lifetime prevalence of 0.6% for anorexia nervosa (AN), 1.0% for bulimia nervosa (BN), and 2.8% for binge eating disorder (BED) [1]. However, these disorders present with a significant mortality rate and a high prevalence of psychiatric comorbidities [2,3]. There is evidence that the presence of untreated comorbidities in patients with ED is associated with worse prognosis, due to greater symptom burden, lower likelihood of remission and a higher degree of functional impairment.

Substance use disorders (SUD) represent the third most prevalent psychiatric comorbidity among patients with ED. The pooled lifetime prevalence of SUD in people with ED is 21.9% (95% CI 16.7–28.0) [4]. Tobacco (36.1 ± 23.1%), caffeine (23.8 ±12.5%), and alcohol (20.6 ± 16.0%) are the most abused substances. In a study conducted by Fouladi F, et al. [5], substance use was more prevalent among patients with BN, to whom was attributed a prevalence of 80% for alcohol consumption and 22% for cannabis consumption.

According to the American Society of Addiction Medicine (ASAM), the definition of addiction includes [6]: compulsive use, impairment in behavioral control, craving (intense desire to consume), persistence of use despite negative consequences and a dysfunctional emotional response. Both BN and BED are characterized by: recurrent episodes in which larger amounts of food than initially intended are consumed in association with a feeling of loss of control; sometimes patients fantasize about and plan the binge episodes; compulsive ingestion persists despite the physical and emotional distress that it causes; patients report a higher frequency of binge episodes in relation to worse emotional states, using food for self-comfort. Thus, we could draw a parallel between ED presenting with binge behaviors and SUD for the so-called self-medication hypothesis [7], according to which drug use is conceptualized not only as a pursuit of pleasure, but also as a means to deal with uncomfortable affective states.

Therefore, a cycle of "food addition" is proposed [8]: when palatable foods are consumed, the brain releases dopamine (along with other neurotransmitters such as opioids), that will act on the mesolimbic reward pathway. With repeated intake, the increase in dopamine leads to downregulation of dopamine receptors, causing individuals to experience a reduction in pleasure during consumption of these foods (tolerance). The decrease in pleasure, combined with craving and social, emotional, and behavioral difficulties, results in a compensatory increase in food intake. Consequently, food consumption can become compulsive, perpetuating the cycle of food addiction. The more "addictive foods" according to the literature correspond to processed foods with high levels of sugars or sweeteners, refined carbs, fat, salt, and caffeine [9].

ED with binge/purging behaviors and SUD diagnoses share [4] higher prevalence of depressive and anxiety disorders and self-injurious behaviors; high risk of suicide; childhood adversity and trauma; family history of psychiatric illness; cluster B personality traits (particularly impulsivity); compulsive behavior; family and cultural influences

(particularly those that normalize disordered eating and substance use). Therefore, these issues should be addressed in consultation, and psychotherapy should be offered. Dialectical behavior therapy (DBT) [10] is based on Linehan's four primary units: mindfulness, interpersonal efficacy, emotion regulation, and distress tolerance. Modified DBT treatment for this dual diagnosis adds a psychoeducational and cognitive-behavioral focus on ED, SUD, and their interrelationships. It also incorporates training of strategies to build a more flexible, structured, and balanced life. Outcomes of this therapy include decreased severity and frequency of substance use, decreased emotional eating and raised levels of confidence in the ability to resist urges to consume.

Officially approved pharmacological treatments for ED [11] include: fluoxetine for bulimia nervosa, with positive effects in reducing the number of binge/purging episodes (up to 60mg/day); lisdexamfetamine (approved by FDA but not by EMA) for binge eating disorder (50-70mg/day). However, if we consider recent studies regarding ED with comorbid SUD, other pharmaceutical options emerge, such as topiramate [12] a glutamatergic agent that proved to be effective in either alcohol and cocaine use disorders, BED (reduction in frequency of binge episodes and obsessive thoughts related to binge) and BN (reduction in frequency of binge/purging episodes, reduction in craving for carbohydrates and overall improvement in quality of life). Another option that showed positive results is naltrexone [13], a weak opioid antagonist with action on the mesolimbic dopaminergic reward system that is proved to decrease craving and relapse in the treatment of alcohol use disorders. In BN and BED, naltrexone reduced the frequency of binge/purging episodes.

Concluding, patients with ED should be regularly screened for substance use and treatment should be offered simultaneously with treatment for ED. Assessment should include evaluation of personality traits (e.g., impulsivity), the occurrence of previous traumatic events, psychological/contextual factors related to consumption, and motivation for change. Psychiatric and organic comorbidities should also be sought exhaustively as they worsen the prognosis and increase mortality. We also propose psychoeducation for substance use to be routinely included in the follow-up of patients with ED.

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