1. Binge Eating Disorders as Substance Related and Addictive Disorders

Preclinical and clinical research suggests that a subset of individuals with binge eating disorder have a food or eating addiction phenotype(1-6) and display neurobiological, physiological, and genetic underpinnings that overlap with those of substance-related and addictive disorders(7-17).

* 1. Epidemiological and Diagnostic Support

Like substance-related and addictive disorders, binge eating disorder has been described as a chronic relapsing disorder characterized by preoccupation and craving, impaired control of consumption, social impairment, persistence despite risks, and aspects of impulsivity, sensitization, tolerance, withdrawal, and relapse, with environmental, genetic, and neurobiological factors (6-8, 10, 18). To better assess signs of addictive-like eating behavior, the Yale Food Addiction Scale Version 2.0 (YFAS 2.0) uses the DSM-V diagnostic criteria for a substance-related and addictive disorder but replaces the term “*drug”* with “*food or binge eating,”*(18-20). Using this criteria, between 42(18)–92%(6) of adults diagnosed with binge eating disorder meet DSM-V diagnostic criteria for a substance-related and addictive disorder (in which the substance/addiction is related to food/eating). Binge eating disorder also has a 27% incidence of comorbidity with true DSM-V substance-related and addictive disorders (21). Research is needed to disentangle the temporal sequence of this comorbidity.

* 1. Preclinical and Clinical Research Support

Feeding and eating are regulated by hunger/satiety mechanisms, ingestive (appetitive and consummatory) mechanisms, and hedonic/reward/reinforcement-based mechanisms (9, 22). The hedonic/reward/reinforcement-based mechanisms include the following reward- and cognition pathways: corticolimbic circuits involving the amygdala, hippocampus, insula, thalamus, hypothalamus, and brainstem/pons; mesostriatal dopamine circuits in the nucleus accumbens, ventral tegmental area, and ventral pallidum; and orbitofrontal circuits in the prefrontal cortex(7-9, 11-16). At the neurochemical level, opioid and dopamine systems within these circuits are primarily responsible for mediating three predominant aspects of reward and reinforcement-based eating: “liking,” associated with hedonic impact/reward value; “wanting,” associated with motivation/incentive salience; and “learning” implicit in cue association and reward predictions[[1]](#footnote-1)(9, 11, 14, 15, 17). These systems are thought to be inherent to all reinforcing processes, including those that drive (24)substance-related and addictive disorders and those that drive overconsumption, obesity, binge eating disorder, and “food and eating addictions,”(7-16, 25-28).

A variety of research demonstrates that processes mediating feeding behaviors can become linked to neurobiological processes that underlie reinforcement and addiction(9, 16, 28). Certain types of foods (e.g., high-sugar, high-fat, highly-palatable, and highly-processed foods)(9, 29, 30), food cue exposure(10), stressors and emotional reactivity(9, 10), and changes in weight or eating patterns(9, 31-33) can induce compulsive patterns of binge-like consumption, akin to those seen in substance-related and addictive disorders (in rodents and humans). Neuroimaging studies also show that food- and drug- related cues and the craving cascade are associated with increased activation in the same brain regions – the nucleus accumbens/striatum, orbitofrontal cortex, insula, caudate, hippocampus, and amygdala – in “food addicts” and drug-users respectively(10, 25, 34-36). Furthermore, the hedonic/reinforcement systems can become hypersensitized, particularly in response to cues (e.g., food cues) and in the context of stress, which can result in cue hyper-reactivity, excessive cravings, behavioral urges to seek/consume associated rewards, and appetitive, reward-seeking behaviors(15). The hedonic/reinforcement systems also have the capacity to delay and override systems related to hunger and satiety(12, 13), which can lead to overeating, obesity, eating disorders, and addictive processes, including binge eating disorders that manifest a phenotype of food and eating addiction(9).

Together, these and other findings suggest that in many cases, a subset of binge eating disorder may represent a substance-related and addictive disorder. Therefore, abstinence-based interventions effective for substance-related and addictive disorders may be effective in alleviating binge eating disorder symptoms(3, 4, 10, 37-39), although this has not yet been demonstrated.

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1. It should be acknowledged that the role of dopamine in addiction processes has been disputed, as it does not seem to be the driving mechanism in some substance-related addiction disorders (ex: cannabis and opiates)[33]. Furthermore, additional hormones/neurohormones, neuropeptides, and neurotransmitters such as orexins, endocannabinoids, glutamate, GABA, leptin, insulin, ghrelin, glucagon-like peptide 1, melanin-concentrating hormone, oxytocin, serotonin, and corticotrophin releasing factor/hormone (CRF/CRH) are also involved. [↑](#footnote-ref-1)