Neurobiological Overlap Between Bipolar Disorder and Binge Eating Disorder: Implications for Screening and Management

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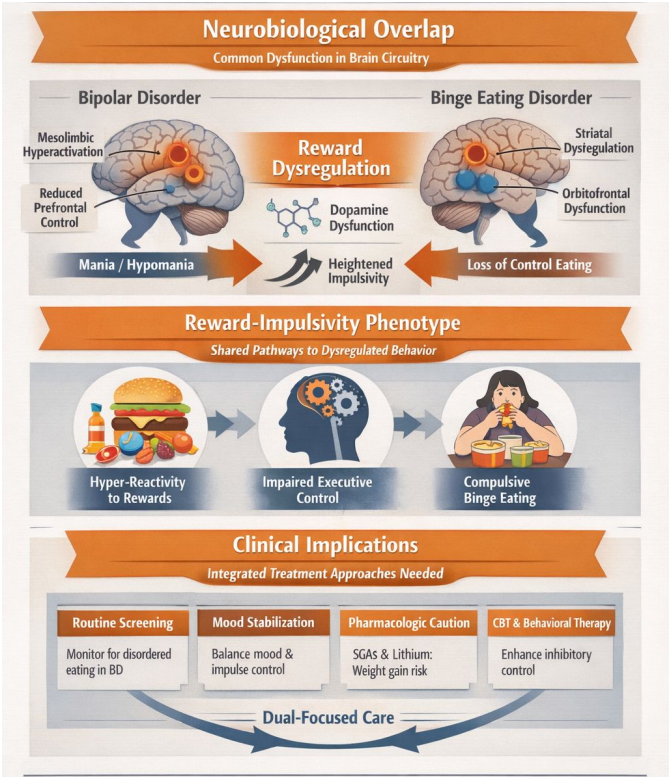
References

Background

Bipolar disorder (BD) and binge eating disorder (BED) demonstrate elevated rates of comorbidity, yet this overlap is often conceptualized as coincidental rather than mechanistically related. Emerging evidence suggests both disorders share core neurobiological features, including dopaminergic reward dysregulation, heightened reward sensitivity, and impaired executive inhibitory control. In BD, particularly during manic and hypomanic states, mesolimbic hyperactivation and reduced prefrontal regulation contribute to impulsivity and behavioral disinhibition. Similarly, BED is associated with altered ventral striatal reactivity, orbitofrontal cortex dysfunction, and deficits in cognitive control. We propose that binge eating behaviors in individuals with BD reflect a shared reward-impulsivity phenotype rather than an independent comorbidity.

Methods

A structured literature review was conducted using PubMed, focusing on epidemiologic, neurobiological, neuroimaging, and behavioral studies examining bipolar disorder, binge eating disorder, reward circuitry, dopamine signaling, impulsivity, executive function, and mood-state–dependent behavioral changes. Findings were synthesized to evaluate shared mechanisms and clinical implications.



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Results

Epidemiologic studies demonstrate increased rates of disordered eating and binge behaviors among individuals with BD, frequently associated with impulsivity and emotion dysregulation. Neuroimaging research in BD reveals aberrant frontostriatal circuitry, characterized by diminished prefrontal regulatory activation and heightened striatal reactivity during reward processing and impulsivity tasks. Parallel findings in BED include dysregulated dopaminergic signaling within the nucleus accumbens, orbitofrontal cortex abnormalities, and executive control deficits that impair decision-making and inhibitory capacity. Behavioral studies further demonstrate increased choice impulsivity and compulsivity across both conditions. Dopaminergic models of BED suggest dynamic alterations in reward processing, with early hyper-reward states potentially followed by compensatory downregulation. Collectively, these findings support shared dysfunction in reward valuation and impulse regulation networks.

Conclusions

The overlap between bipolar disorder (BD) and binge eating disorder (BED) may be best conceptualized as a shared reward-impulsivity phenotype mediated by dopaminergic and frontostriatal dysregulation. Bipolar disorder is characterized by reward hypersensitivity, ventral striatal hyperactivation, and diminished prefrontal inhibitory control, while BED demonstrates parallel abnormalities in dopaminergic signaling, orbitofrontal function, and executive regulation. Mood-state–dependent changes in BD [particularly during mania and hypomania] may further amplify impulsivity and appetitive dysregulation, increasing vulnerability to binge eating behaviors. Clinically, this framework supports routine screening for disordered eating in individuals with BD, especially during periods of mood elevation. Integrated treatment approaches that address mood stabilization alongside impulsivity and reward sensitivity may improve outcomes. Pharmacologic considerations are particularly relevant, as mood stabilizers and second-generation antipsychotics variably influence appetite, weight, and reward processing; proactive metabolic monitoring and thoughtful medication selection may reduce exacerbation of binge symptoms. Incorporating cognitive-behavioral strategies targeting inhibitory control and reward responsiveness may further support recovery. Prospective longitudinal and neuroimaging studies are needed to clarify causal pathways and inform targeted behavioral and pharmacologic interventions