

Insights Into Sleep Disturbances across Eating Disorders

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Description

Sleep plays a key role in overall well-being, impacting cognitive functions, emotional regulation, and immune system health. While sleep disturbances are well-documented in several psychiatric conditions, their relationship with Eating Disorders (EDs) remains complex and less understood. EDs, including Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Binge Eating Disorder (BED), are characterized by dysfunctional eating behaviors and significant concerns about body weight and shape.

Sleep disturbances in eating disorders

Individuals with EDs often experience disrupted sleep patterns. Factors such as food restriction, binge-eating episodes, and emotional distress associated with these disorders can contribute to poor sleep quality. For instance, food restriction in AN may lead to increased nocturnal awakenings and difficulty maintaining restful sleep similarly, individuals with BED, characterized by nocturnal binge-eating episodes, may experience disturbances in sleep onset and maintenance due to discomfort from late-night eating and subsequent digestion.

The neurobiological underpinnings linking sleep and EDs involve orexin, a neuropeptide involved in both wakefulness and appetite regulation. Orexin's activation during fasting periods may promote wakefulness and increase the drive for food-seeking behaviors, potentially disrupting sleep architecture. Moreover, alterations in orexin levels have been associated with changes in Rapid Eye Movement (REM) sleep, which plays a critical role in emotional processing and regulation factors that influence eating behaviors.

Sleep disturbances in individuals with EDs compared to healthy controls. The meta-analysis included studies assessing sleep parameters using both physiological and self-report measures. The main findings can be summarized as follows:

Sleep parameters and circadian preferences: Studies consistently reported poorer sleep quality among individuals

with EDs compared to controls. Reduced sleep efficiency, increased sleep latency, and fragmented sleep patterns were commonly observed across AN, BN, and BED.

Effects of treatment on sleep: Some evidence suggested improvements in sleep quality following treatment for EDs, particularly in AN where weight restoration and normalization of eating behaviors may positively impact sleep architecture. However, further longitudinal studies are needed to confirm these findings across different ED diagnoses.

Prevalence of sleep disorders: The meta-analysis highlighted a higher prevalence of clinically significant sleep disorders, such as insomnia and sleep apnea, among individuals with EDs compared to controls. These disorders contribute to the overall burden of illness in ED populations and warrant targeted interventions.

Understanding the bidirectional relationship between sleep and EDs is key for developing effective treatment strategies. Integrating sleep assessment and management into routine ED care may improve treatment outcomes and quality of life for patients. Clinicians should consider the impact of sleep disturbances on ED symptomatology and tailor interventions accordingly.

In conclusion, while the field has made strides in recognizing sleep disturbances in ED populations, more comprehensive studies are needed to elucidate the mechanisms underlying these disturbances and their implications for treatment outcomes. Addressing sleep as a core component of ED management holds promise for improving overall health outcomes and quality of life for affected individuals. Future research should focus on longitudinal studies to elucidate the temporal relationship between sleep disturbances and ED onset and progression. Additionally, investigating the role of orexin and other neurobiological factors in mediating sleep disturbances in EDs could provide insights into novel therapeutic targets.