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Depression and Anxiety Mediate the Relationship Between Insomnia and Eating Disorders in College Women

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Abstract

Objective: This study examined the associations between insomnia, anxiety, and depression in college women with eating disorders (EDs).

Participants: Six hundred and ninety female students from 28 U.S. colleges who screened positive for an ED were assessed for psychiatric comorbidities. Women were, on average, 22.12 years old, mostly White (60.1%), and undergraduates (74.3%).

Methods: Two mediation models were tested to determine if depression and/or anxiety mediated the relationship between insomnia and ED symptomatology.

Results: One-fifth of the sample (21.7%) reported clinically moderate and severe levels of insomnia. Both depression (B=.13, p<.001) and anxiety (B=.13, p<.001) significantly mediated the relationship between insomnia and ED psychopathology.

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Declaration of Interest

The authors have no financial or non-financial interests to disclose at this time.

Keywords

anxiety; college students; depression; eating disorders; insomnia

Eating disorders (EDs) are serious mental illnesses associated with significant psychosocial impairment, clinical distress, and mortality.^{1,2} EDs are multifaceted and involve interactions among various genetic, behavioral, and cognitive risk and maintenance factors, including the internalization of a thin ideal of beauty,³ emotion dysregulation and distress intolerance,^{4,5} interpersonal dysfunction,⁶ and perfectionism.^{7,8} As such, ED presentations include various behaviors, such as dietary restraint, binge eating, purging (i.e., vomiting, laxative/diuretic abuse), and excessive exercise.⁹ Illness complications are further compounded by the fact that EDs are highly comorbid with a variety of psychiatric conditions, including mood, anxiety, and sleep disorders.^{1,2,10}

Insomnia is a unique manifestation of a disruption in a person's homoeostatic state, caused by circadian system irregularities,¹¹ prolonged hyperarousal,^{12,13} and behavioral factors (e.g., poor sleep hygiene, high consumption of caffeine and/or alcohol, excessive stress)¹¹ – all of which could exacerbate other altered homoeostatic processes, including eating. Clinical insomnia is both a risk and maintenance factor for ED psychopathology. Insomnia is associated with overall severity of ED symptoms as well as poorer quality of life and treatment outcomes.^{10,14,15} Past research has demonstrated that individuals with EDs endorse significantly higher levels of insomnia symptoms relative to controls.^{10,16,17} Although sleep disturbances are common across the ED diagnostic spectrum,^{10,15} insomnia is especially prevalent in individuals with binge/purge symptomatology.^{18–21} The high co-occurrence of insomnia and disordered eating behaviors (ranging from 25-30% in college-age women)¹⁴ is attributed, in part, to disrupted physiological mechanisms, including fluctuations in orexin, an important hormone involved in regulating wakefulness and appetite, in cortisol, a stress hormone, and in ghrelin and leptin, hormones responsible for signaling hunger.^{10,14,22,23}

College students are particularly vulnerable to developing both insomnia and EDs, with prevalence estimates hovering around 9.5% and 3.6-13.5%, respectively.^{24,25} Insomnia is highly prevalent in college women across the ED risk and diagnostic spectrum, with prevalence estimates increasing incrementally by ED risk status.¹⁴ Further, binge/purge symptomatology predicts the onset of later clinical insomnia in college samples.^{18,20} However, research to enhance the understanding of the associations between EDs and insomnia in college populations is needed, as it may inform screening and intervention initiatives, improve academic functioning, and reduce adverse long-term health outcomes for this vulnerable population.^{26–29}

To date, there has been little empirical investigation into the contributing role of additional mental comorbidities to the co-occurrence of eating and sleep disorders. Considering that clinical insomnia and EDs are highly comorbid with mood and anxiety disorders,^{2,30} it is possible that insomnia influences ED symptomology through changes in depression and anxiety.³¹ Indeed, depression has been found to mediate the association between insomnia and eating pathology in outpatient and adult community samples.^{16,32} Anxiety, on the other hand, is predictive of insomnia in the general population³⁰ and mediates the relationship between emotional eating and insomnia in adolescents.³³ Replicating and extending these mediation models to college samples is a key next step in learning how these disorders are associated with insomnia symptomatology in college students.

This study was designed to examine whether depression and anxiety mediated the relationship between insomnia and eating psychopathology in a large sample of college-age women with EDs. Based on prior research,^{16,32,33} we hypothesized that both depression and anxiety would mediate the relationship between insomnia and ED symptomology for the sample. Findings can help to identify new advancements in the clinical assessment, prevention, and treatment of both insomnia and EDs amongst this high-risk population.

Materials and Methods

Participants and Procedure

This study utilized baseline data from 690 participants of the Healthy Body Image (HBI) Program study, a randomized controlled trial testing the efficacy of an Internet-based, guided self-help program for college-age women with EDs at 28 U.S. universities (see earlier reports^{34,35} for more details on study design and data collection). Eligibility criteria for the parent trial included identifying as female, being 18 years, currently enrolled as a student at one of the participating universities, and screening positive for a *DSM-5* clinical or subclinical ED,⁹ with the exception of anorexia nervosa (AN). Given the medical complications associated with this illness, all individuals who screened positive for AN were given a referral to the healthcare center at their respective institution. The Institutional Review Boards at the principal investigators' universities approved all study procedures and materials prior to the commencement of the study. Informed consent was obtained prior to assessment completion.

Measures

All measures were self-report and completed online through Qualtrics.

Eating psychopathology.—The Eating Disorder Examination-Questionnaire (version 6.0; EDE-Q)³⁶ is a 28-item measure that was used to measure frequency and severity of ED symptoms (i.e., vomiting, laxative/diuretic abuse, compulsive exercise, binge eating) over the last 28 days. The EDE-Q consists of four subscales – Weight Concern, Shape Concern, Eating Concern, and Restraint – as well as a Global score that measures global severity of ED symptomatology. Subscale scores are averaged to derive the EDE-Global score. If a participant reports an EDE-Q Global score above 2.3, they are considered to have a clinical

ED.³⁷ The EDE-Q demonstrates good validity and reliability in undergraduate women.^{38,39} In this study, Cronbach's alpha for the EDE-Q Global score was .76.

Insomnia.—Participant's level of insomnia symptomatology was assessed using the sevenitem Insomnia Severity Index (ISI)⁴⁰ measure. Items are rated on a 5-point Likert scale ranging from 0 ("not at all") to 4 ("extremely"), with total scores ranging from 0–28. Higher scores indicate greater insomnia symptom severity, and participants that score 10 are considered to exhibit clinically significant levels of insomnia.^{40,41} Participants were categorized as either being non-clinical (scores of 0-7), or exhibiting subthreshold insomnia (8-14), moderately severe clinical insomnia (15-21), or severe clinical insomnia (22-28). The ISI demonstrates good face validity and internal consistency.⁴² Cronbach's alpha for the current study was .89.

Depression.—The Patient Health Questionnaire $(PHQ-9)^{43}$ is a nine-item survey that assessed participants' level of depressive symptoms over the past two weeks. Items are rated on a 4-point scale, ranging from 0 ("not at all") to 3 ("nearly every day"), with total scores ranging from 0–27. Higher scores correspond with greater depressive symptom severity. The PHQ-9 is considered to be a valid and reliable measure of depressive severity, with a clinical cutoff of 10 indicating moderate depression. ^{43,44} Since item three of the PHQ-9 assesses sleep disturbance (i.e., "trouble falling or staying asleep, or sleeping too much"), we removed this item and modified the PHQ-9 scores by totaling the remaining items to derive a total score for the resulting measure (Cronbach's α =.87). Similar methods have been used in past ED and sleep research.^{35,45} Hereafter, this measure is referred to as the "modified PHQ-9."

Anxiety.—Participants' anxiety symptoms were measured using the Patient-Reported Outcomes Measurement Information System (PROMIS) Short Form v1.0 – Anxiety4a questionnaire.⁴⁶ Items are rated on a 5-point Likert scale, ranging from 0 ("never") to 4 ("always"). Scores range from 4-20 and the clinical cutoff score is considered to be $8.^{47}$ The PROMIS demonstrates good precision, efficiency, and internal reliability.⁴⁸ In this study, Cronbach's alpha was .90.

Analytic Strategy

Preliminary tests of skewness and kurtosis and Mahalanobis distance indicated that the data did not violate assumptions of univariate or multivariate normality. None of the study variables violated the conditions of collinearity (i.e., tolerance > .10; VIF < 10). Secondary analyses using a cross-sectional dataset were conducted. Specifically, using the Hayes model,⁴⁹ two mediation analyses were performed, using bootstrapping, to determine whether the relationship between insomnia and global ED psychopathology was mediated by depression and anxiety symptoms, respectively. According to this method, in the final regression model, if the relationship between the independent and the dependent variables attenuates to non-significance after controlling for the mediating variable, then a significant mediation effect can be inferred. Additionally, in this model, an indirect effect is considered to be significant if the confidence interval does not include zero.⁵⁰ All data were analyzed using SPSS 25;⁵¹ mediation analyses were conducted using PROCESS version 3.⁴⁹

Results

Sample characteristics

Participants reported a mean age of 22.12 years (*SD*=4.84, range=18–58). They identified as White (60.1%), Black or African American (5.4%), Asian or South Asian (17.1%), American Indian or Alaska Native (0.4%), Native Hawaiian or Pacific Islander (0.1%), multi-racial (7.7%), and other (6.7%). Nearly one-fifth of the sample identified as Hispanic (17.4%). Most participants were either undergraduate (74.3%) or graduate students (24.8%). Mean body mass index was 25.70 kg/m² (*SD*=6.00). The distribution of insomnia symptomatology (*M*=9.47, *SD*=6.34) was as follows: non-clinical insomnia (42.3%), subthreshold insomnia (35.1%), clinically moderately severe insomnia (16.5%), and clinically severe insomnia (5.2%). The current sample reported relatively high scores on the EDE-Q Global (*M*=3.57; *SD*=1.10), and moderate scores on the modified PHQ-9 (*M*=9.74; *SD*=5.45) and the PROMIS (*M*=11.18; *SD*=4.22).

Mediation models

The overall mediation model – whereby insomnia symptom severity was modeled to contribute to ED psychopathology through depression – was significant, F(2, 680) = 190.31, p < .001, and accounted for 35.9% of the variance in ED psychopathology (See Figure 1). This model, conducted with 5,000 bootstraps, yielded a mean bootstrap estimate of the indirect effect of .05 (SE = .005). Because the 95% confidence interval did not include zero (0.05 - 0.06), we concluded that the indirect effect of depression on the association between insomnia and ED psychopathology was significant. In other words, insomnia was associated with greater depressive symptom severity, which in turn, was associated with global ED symptom severity for the sample.

A second mediation model was conducted whereby anxiety was modeled to mediate the association between global ED psychopathology and insomnia symptom severity (See Figure 1). The overall model was significant, F(2, 681) = 116.29, p < .001, and accounted for 25.5% of the variance in ED psychopathology. This model, conducted with 5,000 bootstraps, yielded a mean bootstrap estimate of the indirect effect of .03 (SE = .004). Because the 95% confidence interval did not include zero (0.03 - 0.04), we concluded that the indirect effect of anxiety on the association between insomnia and ED psychopathology was significant. Similarly, insomnia was associated with greater anxiety symptom severity, which in turn, was associated with greater global ED psychopathology for the sample.

Discussion

This study examined the prevalence of clinical insomnia in a large sample of college women with EDs, and whether depression and anxiety mediated the association between insomnia and EDs for this group. Findings revealed that one in five college women with EDs endorsed insomnia symptomatology that was clinically moderate or severe, similar to past ED research.¹⁴ As hypothesized, both depression and anxiety independently mediated the association between insomnia symptom severity and EDs, suggesting that insomnia symptoms per se may not directly exacerbate ED symptoms, but that they may

do so through indirect pathways. These findings both confirm and extend past research on community samples¹⁶ and adolescents,³³ suggesting that similar relationships apply for college women with EDs. Although many studies have demonstrated a robust link between depression, sleep disturbances, and EDs,^{10,15} the present study provides evidence that depressive symptoms, separate from the effects of sleep, significantly mediate the association between insomnia and ED symptoms. Furthermore, these findings illustrate how anxiety and insomnia symptoms can influence one another to increase ED symptom severity.

This is one of the first studies to assess for the associations between insomnia and comorbid mental illnesses among a large, geographically diverse sample of college women with EDs. However, there are a number of limitations. First, the data are based on self-report rather than objective, physiological measures. However, due to the logistic limitations imposed by the parent trial (e.g., study was deployed across 28 universities and designed to test Internet-based, technological advancements in ED treatment), self-report measures were the most accessible, efficient, and scalable means of obtaining data on college women with EDs in an anonymous, minimally invasive manner. Furthermore, these measures have strong psychometric support for use in clinical and research settings to assess for various mental health outcomes (i.e., sleep disturbance, eating psychopathology, depression, anxiety). Next, data were cross-sectional and thus causality cannot be inferred; longitudinal exploration into the nature of the relationship between EDs, insomnia, depression, and anxiety, as well as additional transdiagnostic constructs that affect both insomnia and EDs (e.g., emotion dysregulation, weight status)^{52,53} for college women is warranted. Last, future research should replicate these findings with other populations, including non-female students with EDs, and should examine whether these mediation models hold in a sample of college students without EDs.

Findings illustrate the complex interplay between sleep, eating, mood, and anxiety disorders and highlight these relationships within college women with EDs. Health professionals treating college students presenting with EDs should comprehensively assess for common comorbidities, such as depression, anxiety, and insomnia. For patients with both EDs and insomnia symptoms, targeting depression and anxiety early in treatment may have the added benefit of attenuating the impact of insomnia on eating psychopathology for this group.

Both sleep disorders and EDs place a significant economic burden upon college campuses.^{54,55} Although many universities offer mental health services through school-funded counseling centers, many college students are unable or unwilling to seek care due to a series of societal (e.g., stigma), institutional (e.g., understaffed and underfunded counseling centers), and personal barriers (e.g., denial of issue).⁵⁴ Considering this high-cost and the barriers associated with seeking and receiving care, early prevention and psychoeducation may be key to curbing the adverse consequences of sleep disorders and EDs.⁵⁶

In the current study, a sizeable group of college-age women with EDs reported clinically moderate and severe insomnia symptomatology, and this relationship was mediated by depressive and anxiety symptoms. These results underscore the importance of considering comorbidities when addressing the impact of insomnia and EDs in college women, and

offer additional insights into the complex relationships among these common psychological conditions.^{1,2} Health professionals should consider incorporating insomnia, depression, and anxiety screenings into ED assessment, prevention, and intervention efforts on college campuses.

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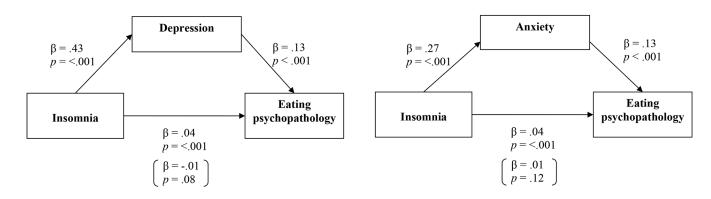


Figure 1.

Models testing mediation of the insomnia-ED psychopathology association through depression and anxiety (*N*=690). Values in parentheses represent the standardized association between insomnia and ED psychopathology after controlling for depression and/or anxiety.