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Psychiatric Co-morbidity in Women Presenting across the Continuum of Disordered Eating

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Abstract

Objective—To compare the prevalence and correlates of psychiatric co-morbidity across a large sample of college women without an eating disorder, those at high risk for an eating disorder and women diagnosed using DSM-5 criteria for an eating disorder.

Participants—549 college age women aged 18–25.

Methods—Data from the Eating Disorder Examination, the Structured Clinical Interview for DSM-IV Axis I disorders and self-report questionnaires were analyzed using logistic regression for categorical data and ANCOVA for continuous measures.

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Contributors

Vandana Aspen wrote the paper in full
Hannah Weisman managed the data set and completed the tables for the manuscript
Anna Vannucci conducted the statistical analysis
Najia Nafiz performed the literature review and wrote up a summary of review
Dana Gredysa performed the literature review and wrote up a summary of review
Mickey Trockel helped with the data analysis, managing the data set and design of the study
Denise Wilfley designed the study, wrote the protocol and proofed the manuscript
C. Barr Taylor designed the study, wrote the protocol and proofed the manuscript
All authors contributed to and have approved the final manuscript.

Conflict of Interest

All authors declare that they have no conflicts of interest.

Results—Eating disordered symptomatology was strongly associated with anxiety disorders, mood disorders and insomnia. These co-morbidities (type and severity) tend to increase with eating disorder symptom severity.

Conclusions—Prevention and treatment programs for eating disorders need to address the high levels of mood, anxiety and sleep problems in this population. The findings on insomnia are novel and suggest that sleep disturbance may play an integral role in eating-related difficulties.

Keywords

High risk; Co-morbidity; Eating Disorders

1.1 Introduction

Eating disorders (EDs) are common, with 2–4% of the population meeting DSM-IV criteria for a full syndrome ED (Hudson, Hiripi, Pope, & Kessler, 2007) and many more suffering from partial syndromes (Stice, Marti, Shaw, & Jaconis, 2009). EDs are associated with significant functional impairment and numerous serious psychological problems, including elevated rates of mood, anxiety, substance use, and impulse control disorders (Baker, Mitchell, Neale, & Kendler, 2010; Godart et al., 2007; Herzog et al., 2006; Hudson et al., 2007; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). These associated psychiatric co-morbidities increase the complexity of the EDs and contribute to overall impairment and decreased quality of life.

While it is not fully understood what causes this high degree of co-morbidity, there is evidence that both genetic and environmental factors are likely at play. For instance, from an environmental perspective, childhood adverse events (i.e. abuse) may act as a common “diathesis” as these events have been shown to significantly increase the likelihood of developing both depression (Chapman et al., 2004) and EDs (Akkermann et al., 2012). In terms of genetics, Steiger et al. (Steiger et al., 2005) postulated that the high rates co-morbidity could be explained by a short allele(s) in the promoter region of the 5-hydroxytryptamine (5-HT) transporter gene (5HTTLPR). Others have argued that a common "diathesis" for EDs and affective disorders is poor affect regulation/negative affectivity (Gilboa-Schechtman, Avnon, Zubery, & Jeczmien, 2006). For instance, a subset of women with EDs may use substances and binge eating to cope with distress.

While efficacious treatments (e.g., cognitive behavioral therapy; CBT) for EDs are available, they are not a panacea. The best results have been in bulimia nervosa (BN) (Wilson, Grilo, & Vitousek, 2007) and binge eating disorder (BED) (Wilson, Wilfley, Agras, & Bryson, 2010). Results from the extensive literature on BN suggest that after a full course of CBT, approximately 30–50% remit completely at post treatment (Wilson, 2005) leaving a large portion of patients symptomatic. Recent evidence suggests that the presence of co-morbidity predicts worse treatment outcome (Keel, Brown, Holm-Denoma, & Bodell, 2011; Schork, Eckert, & Halmi, 1994; Wilfley et al., 2000) and that for many individuals co-morbidity persists after the completion of treatment (Berkman et al., 2006). Additionally, the negative impact of insomnia (both as a risk factor and a maintaining factor) on general psychopathology in college students has been given more attention in recent years (Taylor et

al., 2013; Taylor, Bramoweth, Grieser, Tatum, & Roane, 2011). While little research has been conducted on sleep difficulties amongst those with EDs, it seems likely that sleep difficulties could also contribute to impairment and/or poor treatment outcome.

Given the recalcitrant nature of EDs, early intervention is the most reasonable and cost effective option (Ozler and Henry, 2011). Presumably, intervention would occur at the first sign of serious symptoms that indicate a subclinical ED or when other factors (e.g., elevated weight and shape concerns) indicate that a person is at high risk (HR) of developing a full syndrome ED (Taylor et al., 2006). While the few studies on subclinical EDs confirm the existence of a range of co-morbidities (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002; Touchette et al., 2011), the extent and severity of the co-morbidities in comparison with other disordered eating groups is unclear. Even less is known about individuals at HR of developing an ED, with some studies reporting high rates of substance use (Field et al., 2002; Khaylis, Trockel, & Taylor, 2009; Krahn, Kurth, Gomberg, & Drewnowski, 2005), and depressive symptomatology (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004).

Furthermore, while co-morbidity amongst individuals with DSM-IV EDs is well established, it is unclear how the changes made in DSM-5 (American Psychiatric Press, 1994) will influence the profile of co-morbidity across subclinical and clinical EDs. One recent study (Keel et al., 2011) found that based on the DSM-5 criteria, the AN, BN, BED, and Feeding and Eating Conditions Not Elsewhere Classified (FECNEC) groups had greater lifetime Axis I co-morbidity than matched controls. However, conclusions based on this study are limited as they examined only broad categories of co-morbid pathology as opposed to specific psychiatric diagnoses, considered few dimensional variables of psychological symptoms, and did not investigate co-morbidity among the specific FECNEC variants of EDs.

The primary objective of this study is to compare women without an ED, those at HR for an ED, and women diagnosed using DSM-5 criteria with a FECNEC or clinical ED with regard to measures of psychiatric and family history, eating pathology and psychiatric co-morbidity.

1.2 Materials and Methods

1.2.1 Sample

The current study utilizes baseline data from a community sample recruited to participate in an on-line treatment program to prevent eating disorders. Participants were 549 women ages 18–25 years with a body mass index (BMI) between 18 and 32 kg/m^2, the majority of whom were enrolled in universities in the St. Louis, Sacramento, or San Francisco Bay areas. Exclusionary criteria included no regular internet access (for the randomized trials), starting a new medication or changing dosage within the past 3 weeks (for the randomized trials), suicidality or psychosis, and residency outside the metropolitan regions of the university sites.

1.2.2 Procedures

Recruitment—Participants were recruited via study flyers, email advertisements from university student groups, referrals from campus health centers and Volunteers for Health (a Washington University-based organization), Craigslist, Facebook advertisements, and word of mouth. Participation was voluntary and interested individuals completed a brief initial screening questionnaire online or over the phone, and women identified as at HR for developing an ED were invited for an in-person assessment to confirm study eligibility. A subset of no ED/low risk (i.e., "control") participants were recruited and assessed in-person using the same procedures, with the exception that they were not identified as HR during the screening questionnaire.

Determination of ED Category—Diagnosis of EDs (AN, BN, BED) and not elsewhere specified EDs (FECNEC[1]: subthreshold BN, subthreshold BED, purging disorder) was made based on DSM–5 criteria assessed during administration of the Eating Disorder Examination [EDE; (Cooper & Fairburn, 1987)]. Women were considered HR if they scored 47 or above on the Weight Concerns Scale (WCS; defined below) (Killen et al., 1994). Women were identified as controls if they did not meet DSM-5 criteria for an ED and were not considered at HR for an ED.

1.2.3 Assessments

Participants completed a 2–hour in-person interview with a trained assessor, including two semi-structured diagnostic interviews: the Eating Disorder Examination (Cooper & Fairburn, 1987) previously adapted to include the diagnostic criteria for binge eating disorder (Wilson et al., 2010) and the Structured Clinical Interview for DSM-IV Axis I Disorders (Spitzer, 1987).

Questionnaires included the WCS (Killen et al., 1994), a 5-item self-report questionnaire that measures weight and shape concerns, fear of weight gain, dieting frequency, importance of weight, and feelings of fatness. The WCS has demonstrated good predictive validity and test-retest reliability (Killen et al., 1996; Killen et al., 1994). The Eating Disorder Examination – Questionnaire (EDE-Q) is a 39-item, self-report version of the EDE used to assess ED psychopathology in the last 28 days, yielding a global score and four subscale scores (restraint, eating concerns, weight concerns, and shape concerns; Fairburn & Beglin, 1994). The EDE-Q has demonstrated good internal consistency, temporal stability, and reliability (Luce & Crowther, 1999; Mond, Hay, Rodgers, & Owen, 2006; Mond, Hay, Rodgers, Owen, & Beumont, 2004; Peterson et al., 2007; Reas, Grilo, & Masheb, 2006). The Eating Disorder Inventory (EDI-II) is a self-report measure of disordered eating behaviors comprised of eight subscales (Garner, 1991). For the current study, two of the subscales were utilized: drive for thinness, and perfectionism. The EDI-II and its subscales have demonstrated high internal consistency, reliability, and validity (Bardone-Cone & Boyd, 2007; Peterson et al., 2007). The Clinical Impairment Assessment 3.0 (CIA) is a 16-item, self-report questionnaire that measures psychosocial impairment in the past 28 days across multiple domains (mood and self-perception; cognitive functioning; interpersonal

[1]We did not assess for feeding disorders as they primarily occur in children

functioning and work performance) due to ED features (Bohn et al., 2008). The CIA has demonstrated high levels of internal consistency, test-retest reliability, sensitivity to change, construct validity, and discriminant validity (Becker et al., 2010; Bohn et al., 2008; Reas, Rø, Kapstad, & Lask, 2010). The Diet Aids Checklist (DACL) is a comprehensive list of 53 diet aids currently available to the public, which assesses lifetime endorsement of each diet aid and frequency of use over the past six months. The Difficulties in Emotion Regulation Scale (DERS) is a 36-item self-report questionnaire measuring degree of emotional self-regulation, and has demonstrated high internal consistency, good test-retest reliability, construct validity, and predictive validity (Gratz & Roemer, 2004). The Center for Epidemiological Studies Depression Scale (CES-D) is a 20-item self-report questionnaire that measures depressed mood and negative affect, (Radloff, 1977) and has demonstrated good internal reliability and consistency (Plutchik & van Praag, 1987). An abbreviated version of the Adverse Childhood Events Scale (ACE) was used (Felitti et al., 1998). The ACE is a 68-item self-report questionnaire that measures the type, severity, and frequency of adverse events experienced in the first 18 years of life. For the current study, 10 items related to abuse were selected. The Life Events Checklist is a self-report questionnaire that measures the frequency of recent stressful events that may affect eating patterns or daily living habits (Johnson, 1980). The State-Trait Anxiety Inventory (STAI) consists of two, 20-item scales that measure anxiety as an emotional state (state anxiety) and anxiety proneness as a personality trait (trait anxiety) (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), and has been shown to have good construct validity, test-retest reliability, and sensitivity to change (Novy, Nelson, Goodwin, & Rowzee, 1993; Spielberger, 1989). The Insomnia Severity Index (ISI) is a 5-item self-report questionnaire which measures severity of insomnia (scores range from 0–28; scores above 14 are indicative of clinical insomnia) as experienced over the past two weeks and has been shown to be reliable and valid (Bastien, Vallières, & Morin, 2001). The Drug Checklist assesses frequency of use of 58 current drugs and illegal substances over the past 12 months, and was modified for the present study based on the version used by Taylor et al. (2006).

Anthropometrics were measured before the interview and self-reported socio-demographic data were collected. All measures were completed between September 2009 and April 2010. Informed consent was obtained for all interested and eligible participants prior to completing the first assessment. The study protocol was approved by the institutional review board at each participating site.

1.2.4 Statistical Analyses

All analyses were conducted with SPSS v. 19.0 (SPSS Inc., Chicago, Illinois). Outliers were examined on continuous variables to see if they skewed reported group means. Overall variable group means were compared with five percent trimmed means; excluding the outer five percent of data points did not significantly alter the mean or the pattern of results between groups. Therefore, all outliers were included in subsequent analyses.

1.2.5 Analytic Plan

Logistic regression models were used to obtain odds ratios, which indicated differences between the ED categories (control, HR, FECNEC, and clinical ED) on psychiatric co-

morbidity, medication and treatment history, and family history of psychiatric co-morbidity. The control category served as the reference group for all logistic regression models; hence statistically significant differences indicated differences between ED groups and the control group. The magnitude of the odds ratios provided an indicator of the relative differences in co-morbidity between ED groups. Analyses of covariance (ANCOVA) were used to examine differences between ED categories with regard to body composition, eating pathology, and psychological symptoms. All statistical models controlled for age, race/ethnicity, and parental education status. Simple planned contrasts were conducted where ANCOVA omnibus tests were significant to examine group differences. No statistical tests were performed to assess differences between specific ED diagnosis groups due to insufficient group sizes.

1.3 Results

1.3.1 Overview of Findings

The prevalence and corresponding odds ratios for psychological (ED and co-morbid) disorders, in most cases, increased incrementally by symptom level, with the control group endorsing the lowest odds ratio and the clinical ED group endorsing the highest and minimal differences between the FECNEC and clinical ED groups. Women at HR were most similar to controls in terms of co-morbid pathology but were distinct from all groups in terms of eating behaviors and attitudes.

1.3.2 Socio-demographic Characteristics and Clinical Service Use

Socio-demographic characteristics and clinical service use are presented in Table 1. Women at HR and with a clinical ED had a significantly higher body mass index (BMI) than control women. Age, race/ethnicity and parental education did not differ significantly across groups. Significant differences emerged regarding professional treatment history, with a higher prevalence of women with FECNEC having received general treatment and a higher prevalence of women with a clinical ED having received treatment specific to an ED as compared to the control group. In terms of family history, women with FECNEC or a clinical ED reported a significantly higher prevalence of depression in their family as compared to the control group. The HR group did not significantly differ from the control group with regard to any measures of psychiatric and medical histories.

1.3.3 Eating Pathology

Across the ED-related self-report (CIA, WCS, EDE-Q subscales; except EDI-II perfectionism) and interviewer-rated (objective binge episodes, compensatory behaviors) measures, there was a consistent trend such that the FECNEC and clinical ED groups had the highest scores followed by the HR group and then the lowest scores in the control group, respectively (See Table 2). Women in the clinical and FECNEC groups diverged on eating related pathology; the clinical ED group had a significantly higher number of women engaging in objective binge episodes, and the FECNEC group reported significantly higher scores on perfectionism (EDI-II) and reported significantly higher use of weight control behaviors (appetite suppressant and diet pill use and other category) as compared to the control group.

1.3.4 Co-morbidity with Other DSM-IV Mental Disorders

The prevalence of co-morbidities increased incrementally by risk category (See Table 3). The odds of having co-morbidities were significantly higher for the FECNEC and clinical groups, and women in the HR group were more likely to be diagnosed with *any* overall co-morbidities as compared to the control group. Across co-morbidities, lifetime mood disorders were the most common. Within the mood category, past depression was most prevalent in each of the groups and current depression was significantly more prevalent amongst all disordered eating groups as compared to controls. Rates of suicidal ideation appeared high among women in the clinical group; however significance was not tested because of the lack of cases in the control group. Post hoc analyses revealed that the women in the FECNEC group (3.0%; AOR = 0.14, CIs = 0.05–0.49, $p < 0.001$) and HR group (2.6%; AOR = 0.16, CIs = 0.03–0.77, $p = 0.02$) were significantly less likely to endorse suicidal ideation as compared to the clinical group (17.5%), which served as the reference group. The prevalence of specific anxiety disorders varied considerably, with GAD as the most commonly diagnosed anxiety disorder across groups, and panic disorder as the second most common. Rates of alcohol abuse and dependence were non-significant and low to nonexistent across groups.

Self-reported depression (CES-D), trait anxiety (STAI), and insomnia (ISI) ratings were incremental and significantly different across groups: scores for the HR women were greater than control women and the scores for the FECNEC and clinical women were greater than both the HR and control groups, which did not differ from each other (see Table 2). In addition, rates of insomnia in the clinical range (as measured by the ISI cutoff) were significantly greater in all ED groups as compared to the control group, with prevalence rates increasing significantly by category. Post hoc analyses indicated that women with clinical insomnia had significantly higher nocturnal eating frequency ratings as compared to those without clinical insomnia ($t(543) = 3.21$, $p = .001$). Finally, no group differences in self-reported binge drinking in the previous month, illicit drug use, or tobacco use were found.

1.3.5 Association with Stress, Trauma and Affect Regulation

In terms of emotion regulation (DERS), results suggest an incremental and significant relationship: women in the HR group demonstrated significantly poorer regulation skills compared to women in the control group, and women in the FECNEC and clinical ED groups demonstrated significantly poorer regulation skills than women in both the HR and control groups (see Table 2). No significant findings emerged in relation to current perceived stressful life events. Women in the clinical ED group were significantly more likely to have a history of an adverse childhood event as compared with women in all other groups.

1.4 Discussion

This is the first study to provide comprehensive data on co-morbidity across a large sample of women at high risk (HR) for an eating disorder (ED), as compared to controls and individuals with not elsewhere specified (FECNEC) and clinical EDs, diagnosed using the

DSM-5 diagnostic ED criteria. Overall, the results suggest an incremental increase in co-morbidity and ED symptomatology between those at HR and those with a DSM-5 diagnosis, with minimal distinction between FECNEC and clinical EDs. These findings support the need to address co-morbidities as part of early intervention amongst women presenting at HR for an ED.

Across ED pathology and associated mood, anxiety, and substance related disorders, we found few differences between the FECNEC and clinical ED groups. The similarities between these two diagnostic groups is intriguing, particularly given that two of the three diagnoses assessed in the FECNEC category are sub-threshold. These preliminary data suggest that those with FECNEC disorders may be just as impaired as those with clinical EDs.

A unique part of our dataset was the inclusion of a large sample of HR women. Little research has been done with this population in terms of associated co-morbidities. Our data revealed that HR women were distinguishable from the DSM-5 diagnostic groups (FECNEC and clinical EDs) in that they displayed less eating- and general-related pathology. Additionally, HR women demonstrated few differences from the controls with the exception of ED attitudes and behaviors, which were higher in HR women compared to controls, but lower compared to either women with FECNEC or women with clinical EDs. HR women also differed on some non ED-related diagnoses, including significantly higher odds of any non-ED psychiatric disorder, current depression, any anxiety disorder, and clinical insomnia as compared to the control women. Thus as a whole, HR women appear to be impaired, but to a lesser extent than those with DSM-5 diagnoses. Given that many of these HR women will likely develop full syndrome EDs (Taylor et al., 2006), selective intervention efforts for this risk group offer an opportunity to attenuate ED attitudes and behaviors before they escalate. Furthermore, given that HR women experience more general pathology than healthy controls, intervention programs designed for this risk group need to comprehensively address mood, anxiety and sleep related co-morbidities, in addition to ED attitudes and behaviors.

Results within the FECNEC group revealed interesting findings relating to appetite suppressant use and the prevalence of GAD. First, we found a significantly higher prevalence of self-reported diet pill and appetite suppressant use in the FECNEC group. The use of these products was almost nonexistent in the clinical sample, whereas it was quite prevalent amongst the FECNEC group. This finding may relate to the severity of illness. In other words, those with subclinical EDs might be more likely to experiment with ‘less harmful’ means of controlling their weight. Second, the odds of a co-morbid GAD diagnosis were 9 times higher in the FECNEC group as compared to controls, and approximately 3 times higher than women with clinical EDs. These findings provide preliminary evidence that weight control via pills and GAD may be associated with the FECNEC diagnosis.

High prevalence of insomnia among HR women and even higher prevalence among those with DSM-5 ED diagnoses were significant and notable. Specifically, we found that approximately 5% of controls, 14% of the HR women and 25–30% of the women with DSM-5 diagnoses experienced clinically significant insomnia symptoms. To our knowledge,

no other studies have looked at insomnia in HR or ED samples except in relation to night eating syndrome (NES), where insomnia is common (Townsend, 2007). Although we cannot rule out NES in this population because it was not assessed, it is unlikely to account for our findings, given that only 24% (3.6% of those at a threshold level of twice per week) of women with clinical insomnia engaged in nocturnal eating—a core component of the syndrome. Furthermore, although insomnia is a hallmark sign of depression, it seems unlikely to account for our findings since only 25% of those with clinical insomnia were diagnosed with concurrent depression. Though more information is needed to elucidate the relation between eating and sleep disturbance, our findings suggest that clinicians should assess and address sleep patterns in ED-based prevention and treatment approaches.

Our findings on insomnia are particularly noteworthy since previous research has shown that impaired sleep is associated with a range of negative psychiatric outcomes common to ED populations, including increased anxiety and depression (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). One theory that may help to explain the relation between sleep and EDs focuses on the appetite-regulating hormones leptin and ghrelin and their role in energy consumption. In general, the hormones are key to signaling 1) when the body needs fuel (ghrelin is released and stimulates a hunger response 2) when the body is satiated (adequate concentration of leptin circulates through the body and signals satiety). Interestingly, many studies have shown that when an individual is sleep deprived the concentration of leptin decreases and ghrelin increases leading to increased appetite (Calvin et al., 2013; Copinschi, Leproult, & Spiegel, 2014; Shlisky et al., 2012). Indeed, studies have found that individuals who are sleep deprived show an increase in appetite and desire for calorically dense foods (Spiegel, Tasali, Penev, & Van Cauter, 2004) which could potentially increase the vulnerability to binge eat. Additionally, sleep duration can affect both energy intake and energy expenditure. It also results in sleepiness that may hamper physical activity and extra time awake provides increased opportunity for food intake. Another plausible hypothesis is that there is some common diathesis for both sleep impairment and EDs (e.g., depression, stressful life event), These theories suggest mechanisms by which insomnia may play a role in the development and/or maintenance of an ED and suggest that treating the sleep impairment could reduce some ED symptoms and co-morbid pathology.

This study has some notable strengths. Our findings were based on a large sample of ethnically-diverse women across the spectrum of disordered eating. We used structured clinical interview assessment methods to measure eating pathology and co-morbidity. We examined a wide variety of socio-demographic and psychological problems to provide a comprehensive examination of pathology. Finally, we included women with FECNEC, a new diagnostic category and a large sample of HR women, a population often neglected in the literature.

1.4.1 Limitations

In terms of limitations, our confidence intervals tended to be wide (likely attributable to the small sample size in co-morbid conditions with low base rates). Additionally, our data were cross-sectional, limiting our ability to determine the temporal relation between onset of ED pathology and co-morbid disorders. We were also unable to examine co-morbidity among

specific DSM-5 disorders due to small sample sizes, though our descriptive data point to important future directions. Our sample volunteered to participate in the study and, like any self-selected sample, may have differences from the general public in their levels of psychopathology.

1.4.2 Conclusions

Our findings suggest that ED attitudes and behaviors among college women are associated with numerous co-morbidities, including anxiety disorders, mood disorders, and clinical insomnia. In general, these co-morbidities (type and severity) increase as ED severity increases. Our data further demonstrate that there are few differences in ED- and general-related pathology between the clinical and FECNEC groups. Given the level of co-morbidity at all levels of disordered eating, treatment programs for ED patients regardless of severity need to comprehensively address psychological co-morbidities as these disorders can exacerbate ED symptoms and hinder treatment progress as a result (Keel et al., 2011; Schork et al., 1994; Wilfley et al., 2000).

In terms of future directions, replication of our findings using a larger clinical sample is needed; our novel findings on insomnia, in particular, need to be replicated and its potential role as a causal/maintaining factor evaluated. In addition, prospective data following these women is needed to determine the order in which symptoms emerge. If, for instance, one or more co-morbidities predate the ED symptoms, it may be that treating the co-morbidity might prevent the ED from exacerbating and/or developing. Future work should also consider examining endophenotypes, such as emotion dysregulation or impulsivity, which may account for the extensive co-morbidity among individuals with disordered eating behaviors and the recalcitrant nature of the disease.

In sum, this study adds to the literature on co-morbidity by including both women at high risk of developing an ED and those with FECNEC. This study also provides important data on co-morbidity in college women with a range of disordered eating attitudes and behaviors, and points to the need for early intervention and more comprehensive treatment programs which address mood, anxiety and insomnia in addition to ED symptomatology.

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Highlights

- We examined the prevalence and correlates of psychiatric co-morbidity in college women with varying levels of eating disorder symptoms using DSM-5 criteria
- Eating disorder symptomatology was strongly associated with insomnia, mood and anxiety disorders
- Type and severity of co-morbidity tended to increase with eating disorder symptom severity
- The findings on insomnia are novel and suggest that sleep difficulties may play an important role in eating related difficulties

Table 1

Sociodemographic Characteristics by Eating Disorder (ED) Category.

	Control	High Risk for an ED		FECNEC		Clinical ED	
Number of Cases	96	346		67		40	
	% (SE)						
Race/ethnicity							
White/Caucasian	56.3 (.05)	53.8 (.03)		64.2 (.06)		55.0 (.08)	
African American	7.3 (.03)	9.8 (.02)		7.5 (.03)		5.0 (.04)	
Hispanic/Latino	3.1 (.02)	10.4 (.02)		3.0 (.02)		12.5 (.05)	
Asian American	29.2 (.05)	20.2 (.02)		17.9 (.05)		20.0 (.06)	
Other	4.2 (.02)	5.8 (.01)		7.5 (.03)		7.5 (.04)	
Parental Education[b]							
Less than High School	0.0 (.00)	2.3 (.01)		3.0 (.02)		2.5 (.03)	
High School	26.0 (.05)	28.3 (.02)		26.9 (.06)		17.5 (.06)	
College Degree	24.0 (.04)	25.4 (.02)		17.9 (.05)		32.5 (.08)	
Graduate Degree	50.0 (.05)	43.9 (.03)		50.7 (.06)		47.5 (.08)	
	M (SE)[a]						
Age (years)	20.3 (2.0)	20.6 (2.0)		20.8 (2.0)		20.7 (2.0)	
Body Composition							
BMI (kg/m^2)	22.4 (0.5)[a]	24.8 (0.3)[b]		25.5 (0.6)[b]		24.5 (0.8)[b]	
Psychiatric History[c]	*% (SE)*	*% (SE)*	*AOR (95% CI)*[a]	*% (SE)*	*AOR (95% CI)*[a]	*% (SE)*	*AOR (95% CI)*[a]
Medication for Psych. Reasons[d]	14.6 (.04)	13.3 (.02)	0.9 (0.4–1.7)	22.4 (.05)	1.5 (0.7–3.4)	20.0 (.06)	1.4 (0.5–3.7)
Professional Tx. for Psych. Disorder	12.5 (.04)	12.7 (.02)	1.0 (0.5–2.0)	28.4 (.06)	2.7 (1.2–6.1)*	25.0 (.07)	2.3 (0.9–6.0)
Professional Tx. for Eating Disorder	2.1 (.02)	2.0 (.01)	0.9 (0.2–4.6)	9.0 (.04)	4.6 (0.9–24.1)	12.5 (.05)	6.0 (1.1–33.0)*
Family History							
Eating Disorders	11.5 (.03)	14.2 (.02)	1.3 (0.6–2.6)	22.4 (.05)	2.0 (0.9–4.8)	22.5 (.07)	2.4 (0.9–6.6)
Depression	35.4 (.05)	43.4 (.03)	1.3 (0.8–2.1)	55.2 (.06)	2.1 (1.1–4.1)*	57.5 (.08)	2.4 (1.1–5.2)*
Anxiety	27.1 (.05)	24.6 (.02)	1.2 (0.6–2.5)	34.3 (.06)	2.1 (1.1–4.1)	42.5 (.08)	1.8 (0.8–4.2)

	Control	High Risk for an ED		FECNEC		Clinical ED	
Schizophrenia	3.1 (.02)	2.6 (.01)	0.7 (0.2–2.5)	1.5 (.02)	0.3 (0.1–3.4)	7.5 (.04)	2.3 (0.4–12.6)
Alcohol and/or Substance Abuse	12.5 (.03)	18.2 (.02)	1.2 (0.6–2.6)	19.4 (.05)	1.6 (0.6–2.5)	20.0 (.06)	1.5 (0.5–4.3)

[a] Adjusted for age, race/ethnicity, and parental education status.

[b] Did not know parental education (n=1).

[c] Treatment within the past year.

[d] Any prescription medication for mood, anxiety, or other psychological reasons in the last year

[*] Denotes $p<0.05$ for adjusted odds ratios of ED Categories in comparison to the Control Group

Note: Superscripted letters for means indicate statistically significant post-hoc differences at the $p<0.05$ level; superscripted letters for means that are the same indicate that the ED groups do not differ from each other.

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Table 2

Association between Eating Disorder (ED) Categories and Self-Reported Eating Pathology and Psychological Symptoms.

	Control	High-Risk for an ED		FECNEC		Clinical ED	
Number of Cases	96	346		67		40	
	M (SE)[a]						
Weight Concerns Scale	28.5 (1.6)[a]	56.6 (0.9)[b]		69.0 (2.0)[c]		68.0 (2.5)[c]	
EDE-Q							
Restraint	0.9 (0.1)[a]	2.1 (0.1)[b]		2.6 (0.1)[c]		2.7 (0.2)[c]	
Eating Concern	0.4 (0.1)[a]	1.1 (0.1)[b]		2.0 (0.1)[c]		2.3 (0.1)[c]	
Shape Concern	1.6 (0.1)[a]	3.0 (0.1)[b]		3.9 (0.1)[c]		4.0 (0.2)[c]	
Weight Concern	1.2 (0.1)[a]	2.6 (0.1)[b]		3.6 (0.1)[c]		3.5 (0.2)[c]	
Global Score	1.0 (0.1)[a]	2.1 (0.1)[b]		3.0 (0.1)[c]		3.1 (0.1)[c]	
Eating Disorder Inventory							
Drive for Thinness	2.4 (0.9)[a]	3.5 (0.1)[b]		4.2 (0.1)[c]		4.2 (0.1)[c]	
Perfectionism	3.9 (0.1)[a]	4.1 (0.1)[a]		4.5 (0.1)[b]		4.1 (0.2)[a]	
Clinical Impairment	3.6 (0.8)[a]	10.0 (0.4)[b]		20.1 (1.0)[c]		21.8 (1.3)[c]	
	% (SE)	*% (SE)*	*AOR (95% CI)*[1]	*% (SE)*	*AOR (95% CI)*[1]	*% (SE)*	*AOR (95% CI)*[1]
EDE[b]							
OBEs	1.0 (.01)	8.7 (.02)	8.8 (1.2–65.7)*	61.2 (.06)	175.4 (22.6–1362.9)*	85.0 (.06)	678.5 (76.4–6027.1)*
Self-induced vomiting	2.1 (.01)	2.3 (.01)	1.1 (0.2–5.4)	16.4 (.05)	9.1 (1.0–43.1)*	17.5 (.06)	10.1 (2.0–54.1)*
Diuretic or laxative misuse	0.0 (.00)	0.9 (.00)	N/A	19.4 (.05)	N/A	10.0 (.05)	N/A
Driven exercise	2.1 (.01)	11.0 (.02)	5.9 (1.4–25.3)*	32.8 (.06)	25.4 (5.7–114.0)*	45.0 (.08)	40.9 (8.7–191.6)*
Any compensatory behavior	4.2 (.02)	13.9 (.02)	3.7 (1.3–10.7)*	53.7 (.06)	28.5 (9.3–87.2)*	57.5 (.08)	31.3 (9.5–102.6)*
Weight Control Products[c]							
Diet Pills	2.1 (.02)	4.6 (.01)	2.4 (0.5–10.6)	19.4 (.05)	11.3 (2.4–53.1)*	2.5 (.03)	1.3 (0.1–14.5)
Fat Blockers	0.0 (.00)	0.9 (.01)	N/A	3.0 (.02)	N/A	2.5 (.03)	N/A

	Control	High-Risk for an ED		FECNEC		Clinical ED	
Appetite Suppressant	1.0 (.01)	1.7 (.01)	1.8 (0.2–15.0)	10.4 (.04)	10.9 (1.3–91.8)*	0.0 (.00)	N/A
Other	2.1 (.02)	1.7 (.01)	0.8 (0.2–4.1)	7.5 (.03)	3.7 (0.7–20.0)	2.5 (.03)	1.1 (0.1–12.8)
Drugs of Abuse[d]							
Illicit Drug Use[e]	5.2 (.02)	3.5 (.01)	0.7 (0.2–2.0)	7.5 (.03)	1.3 (0.3–4.8)	2.5 (.03)	0.5 (0.1–4.9)
Tobacco Use	2.1 (.02)	2.3 (.01)	1.1 (0.2–5.6)	3.0 (.02)	1.3 (0.2–10.1)	0.0 (.00)	N/A
	***M (SE)*[1]**						
Binge Drinking Episodes in Past Month	1.1 (0.2)	1.8 (0.1)		2.2 (0.3)		1.7 (0.3)	
DERS	70.7 (2.1)[a]	78.2 (1.1)[b]		89.8 (2.5)[c]		88.3 (3.3)[c]	
CESD	13.7 (0.9)[a]	16.5 (0.5)[b]		20.9 (1.0)[c]		21.1 (1.3)[c]	
ACE	1.1 (0.2)[a]	1.3 (0.1)[a]		1.6 (0.2)[a]		2.1 (0.3)[b]	
Stressful Life Events	1.1 (0.1)	1.3 (0.1)		1.5 (0.1)		1.1 (0.2)	
STAI							
State Anxiety	36.8 (1.2)[a]	41.2 (0.5)[b]		45.4 (1.1)[b]		43.6 (1.4)[b]	
Trait Anxiety	35.0 (1.5)[a]	41.8 (0.6)[b]		47.1 (1.4)[c]		45.8 (1.8)[c]	
Insomnia Severity Index	6.8 (0.6)[a]	8.1 (0.3)[b]		10.7 (0.7)[c]		10.8 (0.9)[c]	
	% (SE)	***% (SE)***	***AOR (95% CI)*[1]**	***% (SE)***	***AOR (95% CI)*[1]**	***% (SE)***	***AOR (95% CI)*[1]**
Insomnia Severity Index Clinical Cutoff > 14	5.2 (.02)	13.9 (.02)	2.7 (1.0–7.1)*	25.4 (.05)	5.6 (1.9–16.3)*	30.0 (.07)	7.7 (2.4–24.1)*

Abbreviations ACE, Adverse Childhood Events Scale; CESD, Center for Epidemiologic Studies Depression Scale; DERS, Difficulties in Emotion Regulation; ED, Eating Disorder; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination Questionnaire; FECNEC, Feeding and Eating Conditions Not Elsewhere Classified (DSM-5 terminology); OBE, Objective Bulimic Episode; STAI, State and Trait Anxiety Inventory.

[a] Adjusted for age, race/ethnicity, and parental education.

[b] Presence in the past 28 days.

[c] Any self-reported use in the past 6 months.

[d] Self-reported current, regular use.

[e] Sedatives, Cannabis, Stimulants, Opioids, Hallucinogens. No one reported use of Cocaine, PCP or other drugs (steroids, glue, ethyl chloride, nitrous oxide, other inhalants, amyl or butyl nitrate, or non prescription sleeping pills).

* Denotes $p<0.05$ for adjusted odds ratios of ED Categories in comparison to the Control Group

Note Superscripted letters for means indicate statistically significant post-hoc differences at the $p<0.05$ level; superscripted letters for means that are the same indicate that the ED groups do not differ from each other.

Table 3

Association Between Eating Disorder (ED) Categories and Psychiatric Co-morbidity, Measured by the Structured Clinical Interview for DSM-IV Disorders.

	Control	High-Risk for an ED		FECNEC		Clinical ED	
Number of Cases	96	346		67		40	
	% (SE)	*% (SE)*	*AOR (95% CI)[a]*	*% (SE)*	*AOR (95% CI)[a]*	*% (SE)*	*AOR (95% CI)[a]*
Overall Prevalence							
Any co-morbidity	41.7 (.05)	52.0 (.03)	1.6 (1.0–2.6)*	79.1 (.05)	5.2 (2.5–10.7)*	75.0 (.07)	4.0 (1.8–9.2)*
Exactly 1 co-morbidity	27.1 (.05)	23.4 (.02)	0.9 (0.6–1.6)	29.9 (.06)	1.1 (0.5–2.2)	20.0 (.06)	0.6 (0.2–1.5)
Exactly 2 comorbidities	7.3 (.03)	15.0 (.02)	2.3 (1.0–5.6)	17.9 (.05)	3.5 (1.2–9.8)*	12.5 (.05)	2.0 (0.6–6.9)
3 or more comorbidities	7.3 (.03)	13.6 (.02)	2.0 (0.8–4.8)	31.3 (.06)	6.1 (2.3–16.4)*	42.5 (.08)	11.3 (3.9–32.2)*
Lifetime Prevalence							
Alcohol Abuse	4.2 (.02)	7.5 (.01)	1.9 (0.6–5.6)	10.4 (.04)	2.4 (0.7–8.9)	10.0 (.05)	2.5 (0.6–11.0)
Alcohol Abuse and Dependence	0.0 (.00)	2.9 (.01)	N/A	9.0 (.04)	N/A	5.0 (.04)	N/A
Mood Disorders							
Any Mood Disorder	35.4 (.05)	40.5 (.03)	1.2 (0.7–1.9)	68.7 (.06)	4.0 (2.0–7.8)*	70.0 (.07)	4.2 (1.9–9.4)*
MDE (current)	1.0 (.01)	8.1 (.02)	7.9 (1.1–59.4)*	11.9 (.04)	12.8 (1.6–105.8)*	22.5 (.07)	28.8 (3.5–239.0)*
MDE (past)	31.3 (.05)	34.1 (.03)	1.1 (0.7–1.8)	61.2 (.06)	3.4 (1.7–6.6)*	62.5 (.08)	3.5 (1.6–7.8)*
Minor Depression	4.2 (.02)	4.0 (.01)	1.1 (0.3–3.4)	3.0 (.02)	0.8 (0.2–4.6)	10.0 (.05)	2.8 (0.6–12.3)
Dysthymia (current)	0.0 (.00)	1.4 (.01)	N/A	1.5 (.02)	N/A	7.5 (.04)	N/A
Dysthymia (past)	3.1 (.02)	6.1 (.01)	1.7 (0.5–5.8)	6.0 (.03)	1.6 (0.3–7.5)	12.5 (.05)	3.8 (0.8–17.1)
Suicidal Ideation	0.0 (.00)	2.6 (.01)	N/A	3.0 (.02)	N/A	17.5 (.06)	N/A
Mania	1.0 (.01)	0.0 (.00)	N/A	3.0 (.02)	3.2 (0.3–37.8)	2.5 (.03)	3.1 (0.2–57.0)
Anxiety Disorders							
Any Anxiety Disorder	11.5 (.03)	23.4 (.02)	2.2 (1.1–4.3)*	38.8 (.06)	4.9 (2.2–10.9)*	42.5 (.08)	5.5 (2.2–13.5)*
Panic Disorder	4.2 (.02)	9.2 (.02)	2.3 (0.8–6.6)	13.4 (.04)	3.3 (1.0–11.4)	15.0 (.06)	4.0 (1.0–15.1)*
Agoraphobia	1.0 (.01)	1.7 (.01)	1.5 (0.2–12.5)	6.0 (.03)	5.7 (0.6–53.2)	2.5 (.03)	2.0 (0.1–33.6)
Social Phobia	5.2 (.02)	7.2 (.01)	1.4 (0.5–3.9)	11.9 (.04)	2.8 (0.9–9.1)	10.0 (.05)	2.3 (0.6–9.2)

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	Control	High-Risk for an ED		FECNEC		Clinical ED	
Obsessions	0.0 (.00)	1.7 (.01)	N/A	4.5 (.03)	N/A	2.5 (.03)	N/A
Compulsions	0.0 (.00)	4.6 (.01)	N/A	3.0 (.02)	N/A	5.0 (.04)	N/A
GAD	3.1 (.02)	7.5 (.01)	2.3 (0.7–7.9)	23.9 (.05)	9.2 (2.5–33.2)*	10.0 (.05)	3.3 (0.7–15.8)
PTSD	0.0 (.00)	1.4 (.01)	N/A	3.0 (.02)	N/A	12.5 (.05)	N/A

[a] Adjusted for age, race/ethnicity, and parental education status.

* Denotes p<0.05 for adjusted odds ratios of ED Categories in comparison to the Control Group