Psychiatric Comorbidities of Female Inpatients With Eating Disorders

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Objective: We analyze 27 point-prevalent *DSM-IV* Axis I comorbidities for eating disorder inpatients. **Methods:** The sample included 2436 female inpatients treated between January 1, 1995, and December 31, 2000, for primary *DSM-IV* diagnoses of anorexia, bulimia, and eating disorder not otherwise specified. Analyses were multivariate analysis of variance and multinomial logistic regression; sociodemographics and severity-of-illness measures were controlled. **Results:** Ninety-seven percent of patients evidenced ≥1 comorbid diagnoses; 94% evidenced comorbid mood disorders, largely unipolar depression, with no differences across eating disorders; 56% evidenced anxiety disorders, with no differences across eating disorders; and 22% evidenced substance use disorders, with significant differences across eating disorders ($p < .0001$). Five specific diagnoses differed across eating disorders. Alcohol abuse/dependence was twice as likely with bulimia ($p < .0001$); polysubstance abuse/dependence three times as likely with bulimia ($p < .0001$); obsessive-compulsive disorder twice as likely with restricting and binge/purge anorexia ($p < .01$); posttraumatic stress disorder twice as likely with binge-purge anorexia ($p < .05$); schizophrenia/other psychoses three times more likely with restricting anorexia ($p < .05$) and two times with binge-purge anorexia ($p < .05$). **Conclusions:** New findings emerged: extremely high comorbidity regardless of eating disorder, ubiquitous depression across all eating disorders, no difference in overall rate of anxiety disorders across eating disorders, greater posttraumatic stress disorder in binge-purge anorexia, more psychotic diagnoses in anorexia. Certain previous findings were confirmed: more obsessive-compulsive disorder in anorexia; more substance use in bulimia; and a replicated comorbidity rank-ordering for eating disorder patients: mood, anxiety, and substance use disorders, respectively. **Key words:** anorexia, bulimia, eating disorder, comorbid, co-occurring, Axis I.

ANB = anorexia nervosa, binge-eating/purging type; **ANOVA** = analysis of variance; **ANR** = anorexia nervosa, restricting type; **BMI** = body mass index; **BN** = bulimia nervosa; **ED** = eating disorder; **EDNOS** = eating disorder not otherwise specified; **LOS** = length of stay; **MANOVA** = multivariate analysis of variance; **OCD** = obsessive-compulsive disorder; **PTSD** = posttraumatic stress disorder; **SUD** = substance use disorder.

INTRODUCTION

Eating disorders (EDs) have substantial Axis I comorbidity (1,2). In one major study (1), 73% of patients with anorexia nervosa, restricting type (ANR), 82% with anorexia nervosa, binge-eating/purging type (ANB), and 60% with bulimia nervosa (BN) had one or more concurrent Axis I comorbidities (1). Higher lifetime Axis I comorbidities of 80% to 97% have been reported across EDs (3).

Psychiatric comorbidity may increase ED severity, chronicity, and treatment resistance (4,5). It suggests poorer ED recovery (4,5), and poorer comorbidity recovery due to effects of altered nutrition on illness course, cognition, and medication efficacy (6). ED patients with psychiatric comorbidity may require specialized treatment protocols (6–9), including exposure with response prevention for those with comorbid obsessive-compulsive disorder (OCD; 10) or attention to appetite-stimulating or suppressing properties of medications, and may have more ED medical complications (4). Because appetite regulation, food selection, and consummatory behavior are modulated and partially determined neurobiologically by the same neurotransmitter systems significantly involved in other psychiatric disorders, ED comorbidity may have genetic and psychopharmacologic implications (4). Because many psychiatric disorders besides EDs also involve appetite and eating disturbances (6), ED comorbidity patterns may have implications for differential diagnosis.

ED's common psychiatric comorbidities include mood, anxiety, and substance use disorders (SUD) (3,7,11–18). Across investigations, 20% to 98% of ED patients have mood disorders. Rigorous studies (5) suggest that mood disorders are ED's commonest psychiatric comorbidity. Seven percent to 65% of ED patients have anxiety disorders, with OCD and social phobia commonest (7,11). Six percent to 55% of ED patients have SUD (9). Comorbid ED and psychotic disorders have been reported sporadically (18–24), with recent studies emphasizing this association in AN (8,25–28). Comorbidities between EDs and other Axis I disorders are occasionally reported (1,7,14,18).

ED comorbidity requires a conceptual model (29) that: 1) specifies adequately large samples to detect multiple, and uncommon, comorbid relationships; 2) employs valid diagnoses; 3) distinguishes lifetime from point-prevalent comorbidities; 4) considers the time sequence of the illnesses; 5) utilizes control/comparison groups to establish risk-specificity for EDs and comorbidities; and 6) controls for subjects' sociodemographic, treatment-seeking, and severity-of-illness characteristics.

Existing studies miss some or all of these criteria. The current study addresses some of these limitations. Its large sample provides statistical power to detect multiple and uncommon comorbid relationships. *DSM-IV* ED diagnoses are carefully made. The present study assesses point-prevalent comorbidities for a specific population. Multiple sociodemographic and severity-of-illness measures are controlled statistically. Estimates from this sample should therefore improve understanding of ED comorbidity, helping to anticipate treatment needs for inpatient populations.

METHODS

Participants

Remuda Ranch Programs for Eating Disorders' institutional review board approved this retrospective chart review of all 2436 inpatients with primary ED diagnoses admitted between January 1, 1995, and December 31, 2000.

Results were presented at the Eating Disorder Research Society annual meeting, Albuquerque, New Mexico, November 2001.

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TABLE 1. Sociodemographic and Severity-of-Illness Measures by ED[a]

Measure, *N*	ANR, 520	ANB, 436	BN, 882	EDNOS, 598	All Patients, 2,436
Sociodemographics					
Admission age[b]	20.9[a] ± 9.1 Range: 11–68	23.8[b] ± 8.5 Range: 11–51	23.9[b] ± 7.6 Range: 12–58	24.4[b] ± 9.2 Range: 11–63	23.4 ± 8.6 Range: 11–68
Sex (% female)	100% a	100% b	100% b	100% b	100%
Education level[b]					
<High school	45%	25%	21%	33%	30%
High school degree	8%	15%	15%	15%	14%
Some college	31%	38%	42%	33%	36%
4-Year college degree	12%	15%	17%	14%	15%
Postgraduate	4%	7%	5%	5%	5%
Ethnicity					
Black	0.3%	0%	0.2%	0.3%	0.2%
Asian-American	1.7%	0.9%	1.6%	0.9%	1.3%
Hispanic	2.8%	3.7%	2.6%	2.9%	2.9%
Native-American	0%	0.5%	0.7%	0%	0.3%
White	95.1%	94.9%	94.9%	95.9%	95.2%
Marital status					
Never married	79%	72%	70%	68%	72%
Married	14%	18%	19%	22%	18%
Divorced/separated	7%	10%	11%	10%	10%
Severity-of-illness measures					
ED onset age	15.5 ± 5.3	15.8 ± 5.5	15.3 ± 4.1	15.7 ± 6.8	15.5 ± 5.4
Illness duration (yr)[b]	5.5[a] ± 7.7	8.3[b] ± 7.7	8.5[b] ± 7.1	8.9[b] ± 10.1	7.9 ± 8.2
Admission BMI[b]	15.0[a] ± 2.3	16.0[b] ± 2.1	21.6[c] ± 4.4	19.9[d] ± 4.5	18.8 ± 4.6
Length of stay[b]	58.6[a] ± 20.0	51.6[b] ± 18.5	44.7[c] ± 15.4	47.4[d] ± 16.4	49.6 ± 18.1
Prior inpatient ED Tx[b]	53%[a]	66%[b]	48%[a]	50%[a]	53%
Treated only at step-down center	2%	2%	5%	5%	4%

[a] Wilks' λ for MANOVA = 0.58, $F(27,4662) = 35.9$, $p < .001$. Using Scheffe's post hoc comparison test, $p < .05$, categories with same superscript are not significantly different and those with different superscripts are.
[b] Means are given with SD Bonferroni-corrected $p < .005$ from ANOVA.

T1,AQ: 2 Table 1 presents sample characteristics. Most patients with severe primary ED diagnoses meet Remuda's admission criteria, except those with uncontrolled dissociative identity disorder, who are excluded due management difficulties in the unlocked residential setting. Those with medical complications requiring hospitalization typically admit once medically stabilized.

Patients met *DSM-IV* ED criteria as follows: 520 ANR; 436 ANB; 870 BN, purging type; 12 BN, nonpurging type; 598 eating disorder not otherwise specified (EDNOS). Because few had BN, nonpurging type, all 882 BN patients were combined. Because Remuda did not treat binge-eating disorder during the study timeframe, EDNOS in this sample indicates partial syndrome anorexia or bulimia only.

Nine sociodemographic and severity-of-illness measures were available from records. See Table 1. All patients were female, reflecting Remuda's admission practices. Race and ethnicity, defined using US census categories, were included because of debate in the literature about relationships between race/ethnicity and EDs (30). ED onset age serves as a proxy for illness severity because, on average, ED patients with younger onset age have poorer outcomes (31).

Procedure

Measures

All patients were interviewed within 2 days of admission by a board-certified psychiatrist or licensed psychiatric nurse practitioner, primary care physician or nurse practitioner, master's-level therapist, registered dietitian, licensed practical or registered nurse, and licensed doctoral-level psychologist. Professionals gathered detailed information about patient background and symptoms using unvalidated proprietary structured formats.

Admission body mass index (BMI) was computed using admission height and first morning weight collected under supervision of registered dietitians. All patients received admission drug screens. Patients completed extensive psychological testing, including the Minnesota Multiphasic Personality Inventory-2 (32) or Minnesota Multiphasic Personality Inventory-A, depending on age (33); Eating Disorder Inventory-2 (34); Beck Depression Inventory-II (35); Beck Anxiety Inventory (36); Yale-Brown Obsessive Compulsive Scale, adapted (37); Trauma Symptom Inventory (38) or Trauma Symptom Checklist for Children (39), depending on age; Substance Abuse Subtle Screening Inventory (40); Wender Utah Rating Scale for Attention-Deficit/Hyperactivity Disorder (41); Dissociative Experiences Scale (42); Conners' Parent Rating Scale–Revised (Long Version); and Conners-Wells Adolescent Self-Report Scale (Long Version) for ADHD (43). Adults admitted after June 1, 1999, completed the Structured Clinical Interview for *DSM-IV* Screen Patient Questionnaire–Extended (44). Psychological testing provided objective measures of critical symptoms of each Axis I disorder considered in this study.

We stress the unusually strong assessments at this treatment center. Diagnoses occur with input from multiple licensed healthcare professionals working together in truly collaborative teams. Professionals observe patients for 40 to 60 days in a 24-hour milieu, communicating observations continually to refine assessment and diagnosis. Patients receive 40 hours minimum of therapeutic contact with licensed healthcare professionals during each treatment week, in a facility with a 3.5:1 staff-to-patient ratio. Professionals ascertain collateral history from family members, review previous treatment records, and speak with referring professionals. Diagnosis is therefore a thorough, ongoing process.

With input from these multiple data sources, the psychiatrist and psychologist reach consensus and assign Axis I diagnoses. To avoid overdiagnosis, attention is paid to symptom duration, developmental sequence, and overlap. Using ongoing assessments and input from the full treatment team, the psychiatrist and psychologist reach consensus and assign discharge Axis I diagnoses.

To corroborate ED diagnostic reliability, 50 patients were randomly chosen from the sample. One author reassigned admission ED diagnoses to these patients via chart review without knowledge of diagnoses already assigned. For all 50 patients, the same ED diagnosis was determined, suggesting diagnostic reliability (100%). Because of unusually thorough assessments relying on objective tools and diagnostic assignment by consensus between two licensed doctoral-level

professionals, comorbid diagnoses may also have acceptable reliability, but a precise reliability metric is unavailable.

For this study, patients received a comorbid diagnosis if this diagnosis appeared at either admission or discharge. Although this facility's admission diagnoses offer a comprehensive picture of patient functioning arising from extensive assessments, occasionally patients are defensive at admission to the point where denial obscures diagnosis. However, by conclusion of the minimum 40 treatment days, symptoms of defensive patients emerge. Thus, discharge diagnoses occasionally add detail. They were included to offer the most complete snapshot possible of patient comorbidity. When admission diagnoses are corrected or superseded by discharge diagnoses, changes are noted in patient records. Superseded admission diagnoses were removed from these analyses so that only correctly applicable diagnoses for each patient were used.

Comorbid diagnoses in this study represent a relatively short, discrete time period, called contemporary, concurrent, or point-prevalent diagnoses. However, because Remuda's Intensive Centers treat severe EDs, patients likely exhibited most lifetime diagnoses during this treatment episode.

For more powerful and meaningful statistical analyses, we reduced the sheer number of comorbidities. We combined abuse/dependence diagnoses for each substance. Several disorders (bipolar, major depressive, panic, and attention-deficit/hyperactivity), were examined without subtypes. Where possible, we combined diagnoses received by <10 patients into groups: dissociative, somatoform, and schizophrenia/other psychotic disorders. Six diagnostic categories were excluded because <10 patients received these diagnoses and no meaningful combination occurred: adjustment ($n = 6$); oppositional defiant and conduct ($n = 5$); impulse-control not elsewhere classified ($n = 7$); learning ($n = 9$); sleep ($n = 3$); and tic ($n = 3$) disorders.

Using these methods, 27 Axis I diagnoses or diagnostic groups occurred among ≥10 patients and were included in analyses. We represented each as a dichotomous dummy variable: 1 indicated presence and 0 indicated absence of the diagnosis (45). For comorbidity percentages by EDs, see Table 2. T2

Statistical Treatment

We analyzed sociodemographics and severity of illness (Table 1) in relation to ED using multivariate analysis of variance (MANOVA). Sex and ethnicity varied minimally and were omitted from the MANOVA. To include the categorical variable of marital status in the MANOVA, we followed standard procedures (46).

MANOVA was followed by univariate analyses of variance (ANOVA); univariate *F*s determined whether individual measures differed by ED. ANOVAs included 10 concurrent statistical tests, so we applied a Bonferroni correction,

TABLE 2. Percentages of Patients With Comorbid *DSM-IV* Axis I Diagnoses by ED

Axis I Diagnoses	ANR	ANB	BN	EDNOS	All	χ^{2a}
N	520	436	882	598	2,436	
Any Axis I disorder	96	98	97	97	97	2.05
Mood disorders						
Depressive disorder NOS	58	51	48	48	50	1.00
Major depressive disorder, all subtypes	40	50	46	48	46	0.90
Dysthymic disorder	4	5	8	8	7	6.26
Other mood disorders	0.6	0.7	1	0.5	0.8	2.37
Total: any unipolar depression	92	93	92	93	92	
Bipolar disorders	2	4	5	4	4	1.60
Total: any mood disorder	93	95	94	95	94	0.33
Anxiety disorders						
Obsessive-compulsive disorder	29	28	16	20	22	13.99**
Posttraumatic stress disorder	10	25	23	23	20	9.79*
Anxiety disorder NOS	20	15	20	21	19	7.25
Generalized anxiety disorder	7	9	7	10	8	2.87
Panic disorders	3	6	4	5	4	1.61
Social phobia	4	2	3	3	3	5.93
Other anxiety disorders	1	1	1	1	1	3.90
Total: any anxiety disorder	55	59	55	58	56	0.53
Substance-related disorders						
Alcohol abuse/dependence	3	14	26	14	16	25.11***
Cannabis abuse/dependence	0.6	4	5	4	4	5.13
Polysubstance dependence	2	4	10	5	6	18.14***
Other substance abuse/dependence	0	0.7	2	0.8	1	6.27
Amphetamine abuse/dependence	0.4	0.5	1.0	0.8	0.9	1.69
Sedative/hypnotic/anxiolytic abuse/dependence	0.2	1.4	0.3	1	0.7	2.85
Cocaine abuse/dependence	0.2	1.4	0.6	0.2	0.5	5.70
Hallucinogen abuse/dependence	0.2	0.2	0	0.3	0.2	2.58
Opioid abuse/dependence	0	0.2	0	0.7	0.2	6.61
Inhalant abuse/dependence	0	0	0.2	0	0.1	0.40
Total: any substance abuse/no EtOH	3	10	18	12	12	
Total: any substance disorder	5	20	34	20	22	45.94***
All other Axis I disorders						
Attention-deficit/hyperactivity disorder	3	3	9	6	6	1.39
Dissociative disorders	0.4	0.5	0.6	1.2	0.7	3.10
Somatoform disorders	0.2	0	0.9	1	0.6	4.12
Trichotillomania	0.4	0.7	0.3	0.5	0.5	1.47
Schizophrenia/other psychotic disorders	0.8	0.5	0.2	0.3	0.4	9.02*

* $p < .05$.**; $p < .01$.***; $p < .0001$.

[a] Likelihood ratio χ^2 from multinomial logistic regression analysis.

setting α at 0.005. For measures differing by ED using ANOVA, we employed Scheffe's multiple comparison tests, $p < .05$, to determine which EDs differed from one another.

For a comorbidity overview, we grouped the 27 comorbid diagnoses into meaningful categories: any Axis I comorbidity, mood disorders, anxiety disorders, SUD, unipolar depression, and SUD other than alcohol. See Table 2.

We analyzed EDs in relation to Axis I comorbidities using multinomial logistic regression. We included as covariates any sociodemographic or severity-of-illness variable significantly related to ED in the MANOVA. In these analyses, logistic regression is preferable to least squares methods (46). Due to validity constraints, we examined only main effects.

RESULTS

MANOVA indicates that sociodemographics and severity of illness differ significantly by ED; Wilks' $\lambda = 0.58$, $F(27,4662) = 35.9$, $p < .001$. ANOVAs reveal that age, education, illness duration, admission BMI, length of stay (LOS), and prior inpatient ED treatment differ significantly by ED. See Table 1. Specifically, using Scheffe tests, ANR patients are younger and have less education and illness duration than other ED patients. Patients in all ED groups differ significantly in admission BMI (ANR < ANB < EDNOS < BN) and LOS (BN < EDNOS < ANB < ANR). ANB patients more often had prior inpatient ED treatment.

The first regression model examining EDs in relation to any Axis I comorbidity plus six covariates was significantly better than an intercept-only model: $\chi^2 = 1176$, $df = 21$, $p < .0001$. Nagelkerke R^2 indicates that Axis I comorbidity and six covariates collectively explain 55% of variance in ED diagnosis. Four of six covariates significant in the MANOVA continued to be significantly related to ED when controlling for Axis I diagnosis: education, $\chi^2 = 12.33$, $df = 3$, $p = .006$; admission BMI, $\chi^2 = 887.60$, $df = 3$, $p < .0001$; LOS, $\chi^2 = 38.36$, $df = 3$, $p < .0001$; and prior inpatient ED treatment, $\chi^2 = 23.42$, $df = 3$, $p < .0001$. More importantly, logistic regression revealed that the likelihood of having any Axis I comorbidity does not differ significantly across EDs: $\chi^2 = 2.05$, $df = 3$, $p = .56$.

The second regression model examining EDs in relation to key comorbidity categories (mood disorders, anxiety disorders, and SUD) plus six covariates was significantly better than an intercept-only model: $\chi^2 = 1221$, $df = 27$, $p < .0001$. Nagelkerke R^2 indicates that Axis I comorbidity and six covariates collectively explain 57% of variance in ED diagnosis. Four of six covariates significant in the MANOVA continued to be significantly related to ED when controlling for Axis I diagnosis: education, $\chi^2 = 13.34$, $df = 3$, $p = .004$; admission BMI, $\chi^2 = 848.42$, $df = 3$, $p < .0001$; LOS, $\chi^2 = 33.39$, $df = 3$, $p < .0001$; and prior inpatient ED treatment, $\chi^2 = 23.55$, $df = 3$, $p < .0001$. More importantly, logistic regression revealed that the likelihood of having any mood disorder ($\chi^2 = 0.33$, $df = 3$, $p = .95$) or anxiety disorder ($\chi^2 = 0.53$, $df = 3$, $p = .91$) does not differ significantly across EDs, but likelihood of any SUD does ($\chi^2 = 45.94$, $df = 3$, $p < .0001$). Specifically, holding all other diagnostic categories and covariates constant, SUD are twice as likely to co-occur with BN than with other EDs and half as likely to co-occur with ANR.

The third regression model examining EDs in relation to 27 Axis I comorbidities and six covariates was significantly better than an intercept-only model; $\chi^2 = 1349$, $df = 99$, $p < .0001$. Nagelkerke R^2 indicates that 27 Axis I diagnoses and six covariates collectively explain 61% of variance in ED diagnosis. Four of six covariates significant in the MANOVA continued to be significantly related to ED when controlling for Axis I diagnoses: education, $\chi^2 = 12.19$, $df = 3$, $p = .007$; admission BMI, $\chi^2 = 814.82$, $df = 3$, $p < .0001$; LOS, $\chi^2 = 32.49$, $df = 3$, $p < .0001$; and prior inpatient ED treatment, $\chi^2 = 15.39$, $df = 3$, $p < .002$. More importantly, logistic regression revealed that five Axis I diagnoses differ significantly across EDs when the confounding effects of six covariates and all other Axis I disorders are statistically controlled: alcohol abuse/dependence, polysubstance abuse/dependence, OCD, posttraumatic stress disorder (PTSD), and schizophrenia and other psychotic disorders.

Logistic regression also revealed why these five Axis I diagnoses differ significantly across EDs. See Table 3. First, alcohol abuse/dependence is twice as likely to co-occur with BN than with other EDs. Second, polysubstance abuse/dependence is three times more likely to co-occur with BN than with other EDs. Third, OCD is twice as likely to co-occur with ANR and ANB than with other EDs. Fourth, PTSD is twice as likely to co-occur with ANB than with other EDs. Fifth, schizophrenia and other psychotic disorders are three times more likely to co-occur with ANR and twice as likely to co-occur with ANB than with other EDs. These several findings are not immediately forthcoming when examining raw comorbidity percentages in Table 2, but emerge when multivariate analyses tease out confounding effects of additional comorbid diagnoses, sociodemographics, and severity of illness.

TABLE 3. Odds of Axis I Diagnoses Occurring Together With ED[a]

	ANR	ANB	BN	EDNOS
Any substance abuse/dependence	Half as likely		Twice as likely	
Alcohol abuse/dependence			Twice as likely	
Polysubstance dependence			Thrice as likely	
Obsessive-compulsive disorder	Twice as likely	Twice as likely		
Post-traumatic stress disorder		Twice as likely		
Schizophrenia/other psychotic disorders	Thrice as likely	Twice as likely		

[a] Odds given only for diagnoses that differed significantly across EDs. Odds may differ from raw percentages in Table 2 because they are statistically adjusted using multivariate techniques to control for the effects of other Axis I diagnoses, sociodemographics, and severity-of-illness variables. Odds for schizophrenia/other psychotic disorders are based on raw percentages due to the small number of patients with these diagnoses.

DISCUSSION

Among female inpatients in the present sample, 97% evidenced one or more point-prevalent Axis I comorbid diagnoses, with virtually no difference in this high comorbidity rate across EDs. Regression analyses confirmed that these very similar raw comorbidity rates accurately express similarity across EDs in the likelihood of having any comorbidity. This high point-prevalent comorbidity rate exceeds findings from other studies, which have ranged from 60% to 82% (1). Higher comorbidity in the current sample potentially reflects hospitalization patterns since the mid-1990s that reserve inpatient mental health treatment for severe cases. The finding of the present study suggests that inpatient practitioners should anticipate and assess for comorbidity in virtually all patients whom they treat.

The most common point-prevalent comorbidities involved mood (94%), anxiety (56%), and substance use (22%) disorders. This ordering of comorbidities has emerged in analyses of many inpatient, outpatient, and community samples. The ratio of mood to anxiety to SUD in our sample was approximately 5:3:1, also roughly mirroring most prior findings (7,12,47). However, occasional studies in noninpatient samples have found a greater prevalence of anxiety over mood disorders (48). Thus, treatment-seeking ED samples appear to be consistent in the ranking and ratio of these three broad classes of comorbidity, but community samples may differ.

Existing comorbidity literature offers no truly parallel studies. The most similar investigation examined point-prevalent comorbidities for a range of Axis I disorders among ED inpatients, mostly males (18). This study examined 63 veterans. Although comorbidity rates differed somewhat from our sample, the rank ordering of comorbidities was the same: mood, anxiety, and SUD. In the veteran study, the ratio of mood to anxiety to substance use was 6:3:3. Substance use is generally more prevalent in male and veteran cohorts (49,50). The veteran study also found more comorbid schizophrenia/other psychotic disorders, 10%, again reflecting greater prevalence of these disorders among veterans (49).

Because the previously published inpatient point-prevalent comorbidity data for EDs consists of this single study of 63 veterans, the current study with 2436 patients fills a gap in our understanding of point-prevalent comorbidity in ED inpatients and may have implications for assessment and treatment of this population.

Mood Disorders

We found virtually no difference across EDs in likelihood of having one or more mood disorders. Rates were universally high, ranging from 93% to 95% across EDs. Multivariate regression analysis confirmed that these similar raw comorbidity rates accurately express similarity across EDs in the high likelihood of having one or more mood disorders. Even with multiple confounding variables controlled, the likelihood of having one or more unipolar depressive disorders is essentially identical: no difference across EDs.

Many other studies have similarly suggested high rates of depression across EDs (5,52,53), but in the present sample rates were extraordinarily high. Perhaps prolonged illness and chronicity in EDs initiate adverse relational experiences, nutritional casualty, and affective blunting that predispose longer-term ED patients to depressive symptoms. Inpatient practitioners may benefit from the current findings in expecting the vast majority of patients to have diagnosable depression.

Among mood disorders, most in our sample (92% out of 94%) were unipolar depressions, again mirroring most other findings (47,53,55). This consistent finding might be evaluated in future research with greater attention to diagnostic insertion sequence, particularly in light of a recent investigation suggesting that ED patients whose major depression begins before their ED more often engage in parasuicidal behaviors (56).

Anxiety Disorders

We found virtually no difference across EDs in the likelihood of having one or more point-prevalent anxiety disorders, with rates ranging from 55% to 59% across EDs. Multivariate regression analysis confirmed that these similar raw comorbidity rates accurately express similarity across EDs in the relatively high likelihood of having one or more anxiety disorders even with covariates controlled.

Among outpatient populations, lifetime comorbidity rates between EDs and anxiety disorders, 57% to 64%, are similar to the current inpatient point-prevalent rates (11,57,58). Since comorbid anxiety disorders arise first in 42% to 94% of ED patients (11,48,57,59), point-prevalent and lifetime comorbidity should be quite similar for anxiety disorders. It is important to recognize the clinical implications of this finding: to anticipate anxiety disorders in more than half the ED patients seen in treatment and to assess accordingly.

Although comorbidity for anxiety disorders as a group did not differ across EDs, there were statistically significant differences for OCD and PTSD. With comorbidity odds ratios adjusted for covariates using multivariate techniques, ANR and ANB patients were twice as likely to have OCD compared with BN and EDNOS patients. Most other studies have similarly found OCD higher in AN than BN (5,16,59), with one significant study finding no difference (11). These fairly consistent findings across studies might be evaluated in light of recent research suggesting distinct OCD symptom dimensions, wherein one particular dimension—contamination obsessions and cleaning compulsions—was related to ED prevalence (60). Future research might therefore consider OCD symptom clusters to better understand the apparent comorbidity of OCD and AN.

Unlike the raw percentages of comorbid PTSD presented in Table 2, when multivariate statistical techniques are employed and the effects of other comorbidities, sociodemographics, and severity of illness differences are controlled, the adjusted odds ratios reveal that ANB patients were twice as likely to have PTSD compared with ANR, BN, and EDNOS. A similar finding also emerged in one previous study (11), with no contradictory findings noted in the existing literature.

BN patients were no more likely to have any specific anxiety disorder or anxiety disorders in general than ANR, ANB, or EDNOS patients. This mirrors findings from a recent well-designed study (11), but diverges from other previous research, which found higher rates of social phobia (5,16,29), generalized anxiety disorder (5,29), and panic disorder (29) in BN. Since the divergent results derive primarily from community and outpatient samples, perhaps BN inpatients have lower rates of these specific anxiety disorders than outpatients. Since BN inpatients in the current sample have higher rates of PTSD than other samples, perhaps generalized anxiety disorder was diagnosed less often in the current sample because anxiety symptoms are captured by the PTSD diagnosis. Consistent with this interpretation, a high co-occurrence of BN and PTSD has been demonstrated elsewhere (61). The careful attention paid to diagnostic discrimination in the present study may also lower the likelihood of BN patients receiving a social phobia diagnosis, since symptoms of BN itself—secrecy and guilt about bingeing and purging, body image discomfort—mimic social phobia, potentially leading to this diagnosis in less carefully assessed samples.

Like the present study, a recent investigation (11) found no difference in the rate of panic disorder among patients with ANR. In contrast, other studies have found higher rates of panic disorder in ANR (58). However, these diverging studies examined different populations from the present sample and controls for sociodemographics and severity of illness were absent.

SUD

Similar to other studies (62), we found statistically significant differences between EDs in the likelihood of having one or more comorbid SUD. The ratio of all SUD for BN:ANB:ANR was 7:4:1. Similar ratios were found for alcohol use alone, 9:5:1, and for all SUD except alcohol, 6:3:1. Clearly, BN inpatients evidenced the greatest point-prevalent substance use; ANB, an intermediate amount; ANR, the least.

Multivariate regression analyses revealed that these differences in the likelihood of SUD between EDs are due primarily to BN patients. With the effects of multiple variables teased out through statistical control, using adjusted odds ratios BN patients were thrice as likely to have polysubstance abuse/dependence and twice as likely to have alcohol abuse/dependence. High comorbidity of alcohol and polysubstance use with BN echoes results of a large literature review (63) and large representative national sample (64) and evokes the common notion that BN patients evidence greater impulsivity, including substance use (65,66). These repeated findings must be harmonized with the conclusion (62,67) that substance abuse and BN may not share genetic mechanisms of transmission. The clear relationship between SUD and BN might also be evaluated more deeply in future research in light of recent investigations suggesting there may be distinct subgroups of BN patients both with and without SUD and pervasive dysregulation (68,69) and that relationship between SUD and BN is largely accounted for by the presence of PTSD and major depression (64).

High comorbidity of substance use with ANB accords with previous findings that patients who binge-eat and purge, whether diagnosed with AN or BN, use more substances than ED patients who primarily restrict (12,66,70). It is also supported by the finding (62) that substance use and ANB may share genetic transmission. The lower comorbidity of substance use with ANR echoes much previous research (7,14).

Schizophrenia and Other Psychotic Disorders

Until recently, comorbidity between eating and psychotic disorders has been reported only sporadically over the decades (18–21,71–76). Case studies have suggested no consistent diagnostic and clinical association between psychosis and ED, with affective, schizoaffective, and schizophrenic psychoses reported both to precede and follow, to evanesce and persist after AN (19,75). In the present sample, however, the likelihood of a point-prevalent comorbid diagnosis of schizophrenia/other psychotic disorders was three times greater for ANR and two times greater for ANB compared with BN and EDNOS. This is a new finding that could not have emerged without this sample's large *N*, which made it possible to detect this rare but potentially meaningful association. The association is suggestive but preliminary due to the relatively small number of patients with AN and psychotic disorders in the current sample ($n = 10$).

Research on AN and psychosis is sporadic. Suggestions exist that AN symptoms appear as a prodrome of schizophrenia or disguise an earlier onset psychosis (77). Others (78) noted that improved eating precipitated psychotic symptoms, suggesting that disordered eating may serve as a defense against psychosis. In contrast, one study (19) found that AN psychopathology persisted during psychosis. Recent investigations have emphasized distorted body perception's subdelusional to delusional qualities (9,25–28), noting AN′s positive response to atypical antipsychotics. Future investigations might analyze specific symptoms that distinguish full delusional psychoses from AN′s body image and food delusions (79). It would be optimal to examine these issues cross-culturally, since AN′s delusions may encompass culture-bound expressions of broader psychotic phenomena (80–82).

Studies of brain imaging utilizing PET and fMRI have identified cerebral cortical areas of increased activation (bilateral medial temporal, left medial orbital frontal, anterior cingulated) associated with ED symptoms, suggesting transdiagnostic significance, including affective and psychotic disorders, at the neural level (83,84). The relational withdrawal, affective blunting, ideational rigidity, obsessionalism, and self-perceptive dysfunction characteristic of AN (71,85–88), combined with the results of the present study, may suggest the need for continued research and clinical attention with AN patients to potential underlying neurobiologic/neuroendocrine variables, neurocognitive disturbances, and nutrition/body-image-related functional brain imaging challenge studies (25,89,90) that may lead to more

specific pharmacologic interventions (eg, atypical neuroleptics, selective serotonin uptake inhibitors, anxiety modulating and procognitive agents) and behavioral interventions for AN patients. The present findings also suggest the need in future research to differentiate the components of delusion and psychosis—such as extreme denial of illness, self-perceptive dysfunction, relationship to reality, reality testing, impaired discrimination and error correction—and examine these symptoms transdiagnostically in relation to AN.

Sociodemographics and Severity of Illness

MANOVA indicated that sociodemographic and severity-of-illness measures differed significantly by ED diagnosis. Patients with ANR were younger and had less education and fewer years of illness than patients with other EDs. Patients with all four EDs differed significantly in admission BMI and LOS. Patients with ANB had more prior ED inpatient treatment. Because these factors were controlled in the statistical analyses, it is important to note that these differences cannot account for the comorbidity differences found across EDs in the present sample.

Limitations

The present study has several limitations. The sample includes more whites (95%) than the US population (91). Even with recent research suggesting that 90% of women with EDs are white (92), the percentage of white patients in the current sample remains higher than expected for a US sample. The present patient sample may also have higher SES than the US population because as much as one third of patients paid for care entirely out of pocket. Ethnicity and SES may be related to comorbidity, thus limiting the generalizability of our results to ED patients at large, who may span ethnic and SES differences (93,94). Future studies might address these biases through more diverse subject recruitment and statistical weighting techniques.

Because Remuda offers faith-based treatment, it attracts more patients who express an active religious commitment than in the US population at large. A relationship may exist between religiosity and certain behavioral disorders (95), possibly altering the generalizability of our results.

This study examined a treatment-seeking sample. Psychiatric comorbidities may differ between such samples and community samples with the same illness (96). Treatment-seeking ED samples are more likely to have Axis II pathology (97,98), but no research has established differences in Axis I diagnoses. Because the current study examined Axis I, it is unclear how treatment-seeking status affected results. This study does not offer comorbidity data for ED patients in general but specifically for ED inpatients. As such, it should be useful primarily for clinicians who assess and treat ED inpatients in treatment-seeking settings and researchers interested in this specific population.

Finally, future point-prevalent ED comorbidity studies might not only control for sociodemographic and severity-of-illness variables, producing results for the "average" ED patient, but also by present comorbidity rates within sociodemographic and severity-of-illness breakdowns. Such breakdowns may be clinically meaningful, enabling practitioners more precisely to anticipate and assess for specific comorbid conditions inpatient subgroups.

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AUTHOR QUERIES

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AQ1— Please expand all abbreviations at first use in all major sections of the article (title, abstract, body, tables, and figure legends).

AQ2— Please use asterisks to indicate significance levels instead of superscript letters.

AQ3— Please indicate where references 51 and 54 should be cited in the text.