Alcohol Use Disorder Comorbidity in Eating Disorders: A Multicenter Study

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Background: Eating disorders and alcohol use disorders (AUDs) commonly co-occur, although the patterns of comorbidity differ by eating disorder subtype. Our aim was to explore the nature of the comorbid relation between AUDs and eating disorders in a large and phenotypically well-characterized group of individuals.

Method: We compared diagnostic and personality profiles of 97 women with lifetime anorexia nervosa only, 282 women with lifetime bulimia nervosa only, and 293 women with a lifetime history of both anorexia nervosa and bulimia nervosa or anorexia nervosa with binge eating (ANBN) (DSM-IV criteria). All individuals were participants in a multicenter study of the genetics of anorexia nervosa and bulimia nervosa. We explored pattern of onset, Axis I and II comorbidity, and personality characteristics of individuals with and without AUDs by eating disorder subtype. Personality characteristics were assessed with the Multidimensional Perfectionism Scale, the Temperament and Character Inventory, and the Barratt Impulsivity Scale.

Results: Alcohol use disorders were significantly more prevalent in women with ANBN and bulimia nervosa than in women with anorexia nervosa ($p = .0001$). The majority of individuals reported primary onset of the eating disorder, with only one third reporting the onset of the AUD first. After eating disorder subtype was controlled for, AUDs were associated with the presence of major depressive disorder, a range of anxiety disorders, and cluster B personality disorder symptoms. In addition, individuals with AUDs presented with personality profiles marked by impulsivity and perfectionism.

Conclusions: Individuals with eating disorders and AUDs exhibit phenotypic profiles characterized by both anxious, perfectionistic traits and impulsive, dramatic dispositions. These traits mirror the pattern of control and dyscontrol seen in individuals with this comorbid profile and suggest that anxiety modulation may be related to alcohol use in this group.

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Estimates of the prevalence of comorbid substance abuse and/or dependence in clinical samples of women with bulimia nervosa range between 3% and 50%.[1] Parameters that have varied across studies and that influence comorbidity estimates include the nature of the clinical service (inpatient vs. outpatient), the definition of and assessment procedures for both eating disorders and substance use disorders, whether current or lifetime diagnoses were assessed, the distorting effects of any exclusion criteria for clinical trials, and the age of patients studied.[1] Overall, the majority of studies have observed an elevated prevalence of alcohol use disorders (AUDs) in clinical samples of women with bulimia nervosa. In a review of 25 studies of the prevalence of alcohol abuse or dependence in women with bulimia nervosa in clinical samples, Holderness et al.[2] calculated a median prevalence of 22.9%.

Comorbidity of AUDs appears to differ across subtypes of eating disorders and may reflect differences in patterns of familial liability to psychiatric illness or differences in temperament and personality between individuals with anorexia nervosa and bulimia nervosa.[1] In sum, the majority of studies suggest that although AUDs are relatively uncommon in the restricting subtype of anorexia nervosa, the prevalence of AUDs in women with anorexia nervosa with bulimic features appears to be comparable to or exceed that in women with normal-weight bulimia nervosa.[3–9]

Careful illumination of onset patterns may shed light on differential mechanisms of comorbid association or unique mechanisms of symptom causation across eating disorder subtypes. For example, if AUD preceding bulimia nervosa is associated with statistically different patterns of Axis I or II comorbidity compared with bulimia nervosa preceding AUD, then nested within the larger category of bulimia nervosa may be heterogeneity of etiologic factors. In some cases, for example, AUD may function to moderate binge eating, whereas other onset patterns may imply a common underlying liability. Therefore, variations in risk or liability factors may be implied or imputed through the study of comorbidity, as well as the chronology of onset, of these disorders. In essence, this approach is central to the development of paradigms for refining the study of causative mechanisms underpinning coaggregated disorders.

Several pertinent issues central to this approach have been only partially addressed, due either to the size and nature of the samples investigated or to limitations in the range of variables that have been explored. For example, pattern of onset of AUDs relative to the onset of eating disorders remains unclear, in part because many studies have explored undifferentiated alcohol and other drug abuse. Wiseman et al.[10] compared comorbidity patterns in female patients with an eating disorder and substance dependence with patterns in individuals with an eating disorder and without substance dependence. They did not focus on alcohol only and included a broad range of substances. Of the patients with an eating disorder and substance dependence, 34.8% reported that the onset of the substance abuse preceded the onset of the eating disorder, and the remainder reported that their eating disorder preceded the onset of the substance abuse. The authors found that individuals in this group who developed substance abuse first were more likely to be dependent on a greater number of substances and were also more likely to have cluster B personality disorders compared with those who developed an eating disorder first and those with no comorbid substance use disorders. Individuals in the group who reported that the onset of eating disorders was first reported the greatest number of comorbid diagnoses and were significantly more likely to have panic disorder and social phobia compared with the other 2 groups. Although these findings are intriguing, the impact of including a broad range of substance use disorders on the results is unclear. Given that AUDs are commonly reported forms of substance abuse reported by women with bulimia, we chose to focus on alcohol abuse and dependence.

The goals of the present study were to further characterize the nature of the comorbid relationship between eating disorders and AUDs by (1) comparing the prevalence of alcohol abuse and dependence across well-defined eating disorder subtypes, (2) documenting patterns of onset of AUDs relative to eating disorders across eating disorder subtypes, (3) determining whether there are differences in comorbidity patterns and personality between individuals who experience eating disorders versus AUDs first, (4) exploring diagnostic comorbidity profiles associated with AUDs in individuals with eating disorders, and (5) exploring psychological and personality features associated with AUDs in individuals with eating disorders.

METHOD

Collaborative Arrangements

The current initiative was developed through a cooperative arrangement between the Price Foundation (Geneva, Switzerland), the University of Pittsburgh (Pittsburgh, Pa.), and other academic sites in North America and Europe. The sites of collaborative arrangement, selected on the basis of experience in the assessment of eating disorders and geographical distribution, included University of Pittsburgh (W.H.K.); Cornell University, White Plains, N.Y. (K.A.H.); University of California at Los Angeles (M.S.); University of Toronto, Toronto, Ontario, Canada (A.S.K., D.B.W.); University of Munich, Munich, Germany (M.M.F.); University of Pisa, Pisa, Italy (A.R., G.B.C., M.M.); University of North Dakota, Fargo (J.E.M.); University of Minnesota, Minneapolis (S.C.), University of Pennsylvania, Philadelphia (W.H.B.); and Harvard University, Cambridge, Mass. (P.K.). Each site obtained approval separately from its own institutional review board. All participants provided written informed consent.

Participants

Inclusion criteria for probands and affected relatives have been described in detail elsewhere.[11] The sample was ascertained on the basis of a proband with bulimia nervosa who also had at least 1 affected relative with an eating disorder. For probands, acceptance into the study required that they met the following criteria. (1) DSM-IV lifetime diagnosis of bulimia nervosa, purging type. Purging had to include regular vomiting, with other means of purging optional; binge eating and vomiting must have occurred at least twice a week for a duration of at least 6 months. (2) Age between 13 and 65 years. A current or lifetime history of anorexia nervosa was acceptable (e.g., bulimia nervosa

and anorexia nervosa). Potential probands were excluded if they had a lifetime history of any of the following: mental retardation, dementia, organic brain syndromes, psychotic disorders, Turner's syndrome, or any medical condition that could affect appetite, body weight, or eating (e.g., subjects with diabetes and thyroid conditions were excluded if the onset of the disease preceded the onset of the eating disorder). Individuals with bipolar I or bipolar II disorder were excluded only if symptoms of bulimia nervosa occurred exclusively during manic or hypomanic episodes. Probands with neurologic disorders, such as seizure disorder, were excluded unless seizures developed as a result of trauma and clearly after the onset of the eating disorder. Individuals whose premorbid weight exceeded the 95th percentile for their age and gender for a comparable epidemiologic sample[12] or whose highest lifetime body mass index (BMI) was greater than 35 kg/m^2 were excluded from the study. Of the probands included, 97.6% were white, 2.1% were Hispanic, and 0.3% were Asian.

Affected relatives were biologically related to the proband (e.g., siblings, half-siblings, cousins, aunts, uncles, grandparents, second cousins). Affected relatives were required to be between the ages of 13 and 65 years and have at least 1 of the following lifetime eating disorder diagnoses: (1) DSM-IV bulimia nervosa, purging type or nonpurging type; (2) DSM-IV anorexia nervosa, restricting type or binge-eating/purging type (criteria were modified for this study to include individuals with and without amenorrhea); and (3) eating disorder not otherwise specified (NOS)-1, defined as subthreshold anorexia nervosa or bulimia nervosa. Exclusion criteria for affected relatives included all exclusion criteria listed for the probands with the following additional criteria: (1) monozygotic twin of the proband; (2) biological parent with an eating disorder, unless there was another affected family member with whom the parent could be paired; and (3) a diagnosis of binge-eating disorder as the only lifetime eating disorder diagnosis.

The final sample for this study included 672 participants with anorexia nervosa (N = 97), bulimia nervosa (N = 282), or both anorexia nervosa and bulimia nervosa (including anorexia nervosa with binge eating) (N = 293). We refer to the latter group as ANBN for convenience. Due to a small sample size and substantial within-group heterogeneity, individuals with a primary diagnosis of eating disorder NOS were excluded.

Eating Disorder Phenotypes and General Clinical Information

Lifetime diagnoses and expanded phenotypic information about eating disorders in probands and affected relatives were characterized using a modified version of the Structured Interview for Anorexic and Bulimic Disorders.[13] In addition, eating disorder diagnoses were made using an expanded version of module H of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I)[14] to obtain information such as age at onset, severity of illness or recovery status, and number of months since last eating disorder symptoms. Additional information obtained from probands and affected relatives included gender, date of birth, height, and weight history. Body mass index was based on self-reports of height and weight. Values were calculated for the current, lifetime minimum, and lifetime maximum BMI. Participants responded to demographic questionnaires designed to elicit information about ethnicity, marital status, religious orientation, family structure, occupation, and education. Interviewer qualifications and interview procedures are described in detail elsewhere.[11]

Assessment of Comorbid Axis I and II Disorders

To characterize lifetime AUD (defined as lifetime alcohol abuse or dependence), as well as other Axis I and II disorders, participants were interviewed with the SCID-I[14] and the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II).[15] The SCID-I and SCID-II are semistructured, clinician-administered interviews designed to diagnose the major DSM-IV Axis I and II disorders and to include most of the information that is diagnostically important to researchers.

Assessment of Personality and Temperament

In addition to diagnostic information, we compared responses to a number of self-report questionnaires between participants with and without comorbid alcohol abuse or dependence. These included the Multidimensional Perfectionism Scale (MPS),[16] which assesses 6 dimensions of perfectionism in the areas of concern over mistakes, doubts about actions, personal standards, organization, perceived parental expectations, and perceived parental criticism, and the Temperament and Character Inventory (TCI),[17] which measures 7 dimensions of personality, including novelty seeking, harm avoidance, reward dependence, persistence, cooperativeness, self-directedness, and self-transcendence. To characterize impulsivity, we added the Barratt Impulsivity Scale-11 (BIS).[18] The BIS is a 30-item self-report measure of impulsiveness, currently in its 11th revision. This scale provides 3 measures of impulsivity: motor, cognitive, and nonplanning. The BIS results were available for only 30% of the participants.

Statistical Analyses

Logistic regression with generalized estimating equation (GEE) corrections for nonindependence of the sample was applied to the data (1) to compare rates of alcohol abuse and dependence across eating disorder subtypes; (2) to predict AUD from Axis I and II disorders, eating disorder diagnostic group, and the interaction of the Axis I and II disorders and eating disorder group; and (3) to

Table 1. Prevalence of Alcohol Use Disorders by Eating Disorder Diagnostic Subgroup

	Anorexia Nervosa (N = 97)		ANBN (N = 293)		Bulimia Nervosa (N = 282)		GEE Results		Eating Disorder
Alcohol Use Disorder	N	%	N	%	N	%	χ^2	p	Group Differences
Alcohol abuse	9	9.5	42	14.8	69	24.6	15.73	.0004	AN, ANBN < BN
Alcohol dependence	10	10.3	80	28.0	72	26.1	17.23	.0002	AN < ANBN, BN
Abuse and/or dependence	16	16.8	108	37.8	129	46.1	29.88	.0001	AN < ANBN, BN

Abbreviations: AN = anorexia nervosa, ANBN = anorexia nervosa with bulimia or binge eating, BN = bulimia nervosa, GEE = generalized estimating equation.

predict AUD from psychological measures (which were standardized), eating disorder diagnostic group, and the interaction between the psychological measures and the eating disorder group. The interaction terms outlined above indexed the extent to which AUD predictors varied by eating disorder subtype. These interactions were not significant for any variables, suggesting that the relation between AUDs and both the psychological measures and Axis I and II diagnoses does not differ significantly across eating disorder subgroups. Thus, the interaction was dropped from each model, although the main effects of eating disorder group were maintained. Numbers of subjects in the subgroups vary slightly across analyses due to missing data.

To investigate the effect of the psychological measures on the order of onset of AUD relative to eating disorder, logistic regressions with GEE corrections were again applied, predicting order of onset from the psychological measures. Those participants whose AUD and eating disorder onset occurred in the same year were removed from these analyses.

RESULTS

Diagnostic Groupings

The sample was divided into 3 diagnostic categories: anorexia nervosa, bulimia nervosa, and ANBN. The ANBN group comprised individuals who had a lifetime history of both anorexia nervosa and bulimia nervosa or anorexia nervosa with binge eating. We explored age differences at time of study. Age did not differ significantly across the 3 groups (anorexia nervosa, mean ± SD = 26.64 ± 9.71 years; ANBN, 29.30 ± 9.10 years; bulimia nervosa, 27.75 ± 8.98 years; NS), and there were no significant differences for AUD, eating disorder diagnosis, or the AUD-by-eating disorder interaction.

Prevalence of Alcohol Use Disorder by Eating Disorder Subtype

Table 1 presents the number and percentage of individuals in each diagnostic subgroup who met criteria for alcohol abuse or dependence. Significant differences emerged across groups. Alcohol abuse was significantly less prevalent in individuals with anorexia nervosa or ANBN than in individuals with bulimia nervosa. For alcohol dependence, the prevalence was significantly lower in individuals with anorexia nervosa than in individuals with ANBN or bulimia nervosa.

Table 2. Onset of Alcohol Use Disorders Relative to Eating Disorders[a]

Disorder Onset	Anorexia Nervosa (N = 15)	ANBN (N = 103)	Bulimia Nervosa (N = 123)
Alcohol use disorder first	5 (33.3)	35 (34.0)	42 (34.2)
Onset within same year	3 (20.0)	7 (6.8)	13 (10.6)
Eating disorder first	7 (46.7)	61 (59.2)	68 (55.3)

[a]Values shown as N (%).
Abbreviation: ANBN = anorexia nervosa with bulimia or binge eating.

Patterns of Onset of Eating Disorders and Alcohol Use Disorders

Of the 253 individuals with AUDs, 82 (32%) experienced the onset of AUDs prior to eating disorders, 136 (54%) experienced the onset of eating disorders prior to AUDs, 23 (9%) experienced the onset of both eating disorders and AUDs in the same year, and 12 women had no age recorded. Patterns of onset by eating disorder subtype are presented in Table 2. There were no significant differences in patterns of onset across eating disorder subtypes: Fisher exact test, $p < .42$. The mean ± SD ages at onset of anorexia nervosa, bulimia nervosa, and AUD were as follows: anorexia nervosa, 18.94 ± 5.97 years; bulimia nervosa, 18.49 ± 4.49 years; and AUD, 19.65 ± 5.77 years.

Features Associated With Order of Onset

We then attempted to determine whether there were any patterns of psychological features that were associated with order of onset of eating disorder and AUD. We conducted logistic regression with GEE correction, identifying the standardized psychological variables as the independent variables and order of onset as the dichotomized dependent variable. The onset of AUD first was associated with higher level of parental criticism ($\chi^2 = 5.05$, $p = .02$). No other significant associations were observed.

Axis I and II Comorbidity

Table 3 presents results of the logistic regression predicting AUDs on the basis of the presence or absence of Axis I and II disorders while controlling for the indepen-

Table 3. Logistic Regression Predicting Alcohol Use Disorders on the Basis of Axis I and II Disorders and Eating Disorder Group[a]

Diagnosis	Axis I and II Diagnoses			Eating Disorder Diagnosis, χ^{2c}
	χ^2	p[b]	OR (95% CI)[b]	
Axis I				
Major depressive disorder	7.99	.005	1.84 (1.25 to 2.70)	29.28
Generalized anxiety disorder	0.01			29.69
Obsessive-compulsive disorder	11.41	.0007	1.73 (1.27 to 2.36)	28.56
Panic disorder	1.65			28.33
Posttraumatic stress disorder	5.35	.02	1.75 (1.11 to 2.76)	32.45
Social phobia	7.61	.006	1.77 (1.20 to 2.60)	32.15
Specific phobia	5.95	.015	1.77 (1.13 to 2.77)	30.65
Axis II				
Avoidant	1.87			28.71
Obsessive-compulsive	3.07			28.51
Borderline	14.04	.0002	2.40 (1.55 to 3.70)	25.86
Cluster B	13.16	.0003	2.25 (1.48 to 3.42)	25.42
Cluster C	2.55			28.60

[a]As there were no significant alcohol use disorder–by–eating disorder interactions, these were removed from the model.
[b]Value is indicated if significant.
[c]p = .0001.

Table 4. Regression Analyses (using GEE) Predicting Alcohol Use Disorders on the Basis of Psychological and Personality Features and Eating Disorder Group

Scale	Psychological Variable			Eating Disorder Diagnosis	
	χ^2	p[a]	OR (95% CI)[a]	χ^2	p
Barratt Impulsivity Scale					
Cognitive	1.33			10.83	.0044
Motor	5.25	.02	1.52 (1.12 to 2.08)	8.31	.0157
Non-planning	1.28			9.04	.0109
Temperament and Character Inventory					
Harm avoidance	1.52			31.28	.0001
Novelty seeking	1.02			29.19	.0001
Reward dependence	0.01			29.75	.0001
Persistence	0.01			29.17	.0001
Cooperativeness	0.00			30.76	.0001
Self-directedness	3.46			29.40	.0001
Self-transcendence	4.53	.03	1.21 (1.02 to 1.43)	27.59	.0001
Multidimensional Perfectionism Scale					
Concern over mistakes	6.27	.01	1.23 (1.05 to 1.45)	29.74	.0001
Doubts about actions	4.04	.04	1.18 (1.01 to 1.39)	28.01	.0001
Personal standards	0.13			30.64	.0001
Organization	0.03			30.05	.0001
Parental criticism	16.70	.0001	1.43 (1.21 to 1.69)	30.49	.0001
Parental expectations	4.72	.03	1.20 (1.02 to 1.42)	32.58	.0001
Total perfectionism	9.63	.002	1.30 (1.10 to 1.54)	31.56	.0001

[a]Value is indicated if significant.
Abbreviation: GEE = generalized estimating equation.

dent effects of eating disorder subgroup. Alcohol use disorders were significantly associated with increased prevalence of major depressive disorder (80% of those with major depressive disorder vs. 67% of those without it had an AUD), obsessive-compulsive disorder (51% vs. 36%), posttraumatic stress disorder (19% vs. 11%), social phobia (25% vs. 17%), specific phobia (20% vs. 13%), borderline personality disorder (24% vs. 11%), and cluster B symptoms (26% vs. 13%).

Temperament and Personality Characteristics

For these analyses, we compared personality and temperament variables across individuals with and without AUDs. Those participants with AUDs scored significantly higher on the motor subscale of the BIS (mean ± SD = 23.5 ± 4.5 vs. 21.4 ± 4.2), higher on the self-transcendence scale of the TCI (16.2 ± 6.4 vs. 14.6 ± 6.7), and higher on MPS total perfectionism (95.8 ± 22.7 vs. 89.1 ± 24.3), concern over mistakes (31.1 ± 9.1 vs. 28.9 ± 10.1), doubts about actions (12.8 ± 4.1 vs. 11.8 ± 4.1), parental criticism (12.0 ± 4.7 vs. 10.2 ± 4.7), and parental expectations (14.9 ± 5.7 vs. 13.8 ± 5.9) than women without AUDs. Odds ratios and χ^2 values are presented in Table 4.

DISCUSSION

This study serves to replicate previous findings in the literature using well-defined diagnostic subgroups of individuals with eating disorders and extends the findings by addressing important unanswered questions regarding the nature of the comorbid relation between eating disorders and AUDs.

As previously found,[3,4,9] alcohol abuse or dependence was significantly less prevalent in individuals with anorexia nervosa only than in individuals with bulimia nervosa only. Although the prevalence of alcohol abuse or dependence did not differ significantly between the ANBN and bulimia nervosa groups, the rates for the ANBN group were intermediate between the rates for the anorexia nervosa and bulimia nervosa groups. This finding may be attributable to differences in baseline impulsivity between individuals with a predominantly anorectic versus predominantly bulimic symptom pattern and/or familial and genetic factors, as the prevalence of AUDs tends to be elevated in families of individuals with bulimia nervosa but not anorexia nervosa.[19]

Patterns of onset of eating disorders and AUDs did not differ across eating disorder subtypes. The most common pattern was for the onset of the eating disorder to precede the onset of the AUD. This pattern could simply reflect availability, since access to dieting or binge eating is not bound by legal age restrictions as is access to alcohol. Alternatively, this pattern could reflect the typical ages at onset of eating- and alcohol-related disorders. Another hypothesis posits that alcohol abuse or dependence could follow the onset of an eating disorder due to the increased reinforcing efficacy of alcohol once food deprivation commences. The onset of both anorexia nervosa and bulimia nervosa is

generally marked by periods of food restriction or dieting. In anorexia nervosa, these periods persist indefinitely; in bulimia nervosa, these periods can be prolonged prior to the onset of binge eating.[20–23] Animal data clearly document increased self-administration of other reinforcing substances after food deprivation and weight loss,[24–26] and these principles may be operative in humans who are also vulnerable to the reinforcing properties of alcohol,[27] although this effect has not been observed consistently in the laboratory.[28] Given the retrospective nature of this study, we were unable to obtain sufficiently reliable historical information to determine whether alcohol use did indeed emerge during periods of intense food restriction. Finally, AUDs were strongly associated with the presence of various anxiety disorders in this population, giving rise to the interpretation that AUDs may emerge in women with eating disorders due to the actual or anticipated anxiolytic effects of alcohol.

The presence of AUDs was associated with various comorbid Axis I and II disorders. Specifically, AUDs were associated with an increased risk of depression, obsessive-compulsive disorder, a variety of anxiety disorders, and cluster B symptoms and borderline personality disorder. Studies of epidemiologic samples indicate that anxiety and AUDs frequently co-occur[29–32] and that the onset of some anxiety disorders, such as social phobia, which may predispose to drinking,[33,34] often predates the onset of AUD, despite the fact that alcohol may not attenuate social anxiety symptoms.[35,36]

Substantial data now support the hypothesis that anxiety disorders are pervasive in individuals with eating disorders and that their onset most commonly predates the onset of the eating disorder.[37–40] Silberg and Bulik have identified a common genetic factor that contributes to vulnerability to early eating and anxiety disorders (J. Silberg, Ph.D.; C.M.B., manuscript submitted). We hypothesize that food restriction or dieting may be anxiolytic for individuals who are vulnerable to anorexia nervosa and may represent a primary risk and maintaining force for eating disorders.[41] Due to the pervasiveness of anxiety symptoms, alcohol use may be an additional means whereby individuals with eating disorders attempt to modulate their pervasive anxiety symptoms. These results concur with a previous investigation indicating that the presence of an anxiety disorder in women with bulimia nervosa was associated with both a history of anorexia nervosa and with an earlier age at onset of alcohol or drug use disorders.[42] It cannot be entirely ruled out, however, that the directionality of the relationship is reversed and that anxiety is a consequence of alcohol abuse.

In terms of personality profiles, after eating disorder subgroup was controlled for, the presence of AUD was associated with greater motor impulsivity, higher self-transcendence, and higher internal (concern over mistakes) and external (parental criticism and parental expectations) perfectionism scores. These findings mirror the clinical observation of both overcontrol and dyscontrol in individuals with comorbid eating disorders and AUDs. Elevated self-transcendence could reflect a greater tendency toward psychoticism or dissociative experiences or could reflect religious tendencies, possibly related to recovery attempts.

In summary, whereas prior studies have tended to focus on impulsive traits in women with bulimia and AUD, we have shown that both anxious and impulsive traits are characteristic of these individuals. Moreover, the absence of significant interactions between eating disorder subtype and AUD suggests that similar mechanisms may underlie the relation between AUDs and eating disorders across subtypes. Our sample size, however, precluded detection of significant interactions. Finally, although we did not uncover robust predictors of pattern of onset, or an explanation for why it is more typical for the onset of eating disorders to precede or coincide with the onset of AUDs, it is noteworthy that, unlike anxiety disorders, which tend to predate the onset of eating disorders,[40] AUDs tend to follow the onset of eating disorders. Further studies of predictors of the development of AUDs may assist with identifying those at risk and preventing the emergence of AUDs in individuals with eating disorders.

The results of this study must be interpreted within the context of some methodological limitations. First, the sample included only probands with bulimia nervosa who also had at least 1 affected relative with an eating disorder and their affected relatives. Cases that derive from enriched pedigrees may differ in terms of both severity and comorbidity from sporadic cases. Second, as with all studies that employ lifetime psychiatric history, our data on age at onset of eating disorders and AUDs rely on individual recall. It is unknown whether accuracy of recall of age at onset differs across disorders or whether other issues (e.g., concerns about revealing underage drinking) may influence self-report.

Countering these limitations was the fact that we were able to explore the nature of the comorbid relation between eating disorders and AUDs in a large and phenotypically well-characterized sample of individuals with eating disorders. Our careful documentation of age at onset of both AUDs and eating disorders enabled a more meticulous exploration of patterns and correlates of onset and enabled a more comprehensive comparison across eating disorder subtypes.

These data highlight the importance of continued vigilance on the part of health care professionals for the emergence of AUDs in women with bulimia nervosa as well as individuals with features of both anorexia and bulimia nervosa. Even if AUDs are not present at initial evaluation or commencement of treatment, ongoing attention to the possibility of their later emergence in individuals with

eating disorders is warranted. Particularly vulnerable are those women who manifest comorbid anxiety or depressive disorders.

Disclosure of off-label usage: The authors have determined that, to the best of their knowledge, no investigational information about pharmaceutical agents has been presented in this article that is outside U.S. Food and Drug Administration–approved labeling.

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