Substance Use Disorders in Women with Anorexia Nervosa

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ABSTRACT

Objective: We examined prevalence of substance use disorders (SUD) in women with: (1) anorexia nervosa (AN) restricting type (RAN); (2) AN with purging only (PAN); (3) AN with binge eating only (BAN); and (4) lifetime AN and bulimia nervosa (ANBN). Secondary analyses examined SUD related to lifetime purging behavior and lifetime binge eating.

Method: Participants (N = 731) were drawn from the International Price Foundation Genetic Studies.

Results: The prevalence of SUD differed across AN subtypes, with more in the ANBN group reporting SUD than those in the RAN and PAN groups. Individuals who purged were more likely to report substance use than those who did not purge. Prevalence of SUD differed across lifetime binge eating status.

Discussion: SUD are common in AN and are associated with bulimic symptomatology. Results underscore the heterogeneity in AN, highlighting the importance of screening for SUD across AN subtypes. © 2009 by Wiley Periodicals, Inc.

Keywords: eating disorders; anorexia nervosa; bulimia nervosa; drug use; alcohol related disorders; cannabis

(Int J Eat Disord 2010; 43:14–21)

Introduction

Strong evidence demonstrates that eating disorders and substance use disorders (SUD) commonly co-occur.[1–5] The prevalence of drug and alcohol abuse is approximately 50% in individuals with an eating disorder, compared with a prevalence of approximately 9% in the general population.[1] Similarly, among individuals with SUD, over 35% report having an eating disorder compared with 1–3% in the

Accepted 11 November 2008
Supported by T32MH076694 from National Institute of Health and by MH-66117 from the National Institutes of Health.
Dr. Strober is supported in part by the Franklin Mint Endowed Chair in Eating Disorders.
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Published online 3 March 2009 in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/eat.20670

general population.[1,2] However, of theoretical importance is that among persons with some form of an eating disorder, nicotine, alcohol, and illicit drug use are more common among those who binge eat.[1] Thus, inconsistencies across studies in reported rates of co-occurrence of eating disorders and SUD[3] along with inter-study differences in sample characteristics and general methodology may partly influence this variation.

The suggestion of greater liability to drug use and abuse among persons who binge eat compared with those with restricting type anorexia nervosa (AN)[2–4] is intriguing, if not counter-intuitive, in that among laboratory animals, food restriction enhances, whereas food-satiation reduces, self-administration of nearly every licit and illicit substance with abuse potential.[5] Interestingly, risk of substance abuse may nevertheless be moderated by differences in consummatory patterns across AN subtypes. Strober et al.[6] demonstrated that the 10-year prospective risk of incident cases of SUD in persons hospitalized for AN was six-times greater among individuals who reported binge eating while underweight compared with those with no history of binge eating up to the time of index hospitalization. This risk was also associated with elevated alcohol use disorder among first-degree relatives. Moreover, alcohol abuse among those who developed binge eating subsequent to weight restoration did not differ significantly from those who maintained a restricting profile throughout the study period. Bulik et al.[7] similarly reported that alcohol use disorders were significantly more prevalent among women with AN with binge eating compared with those with AN without binge eating, although this pattern of results has not been universally replicated.[8–11] Finally, little is known regarding the extent to which purging behavior, in the absence of binge eating,[12,13] is associated with risk for SUD in AN.

The motivation for the current study stems from (1) discrepancies in the extant literature regarding the frequency with which lifetime SUD co-occurs with AN; (2) the dearth of studies examining SUD across well-defined AN subtypes; (3) reported findings suggesting the need to examine the extent to which SUD risk is exclusively associated with binge eating in the low weight state[6]; and (4) the importance of examining a purging-only subtype of AN[13] with reference to SUD. To address these questions, the aims of the current study are (1) to examine the prevalence of SUD in a large sample of diagnostically well-categorized women with AN; (2) to compare the prevalence of SUD across AN subtypes; (3) to determine whether SUD are more common in those who report binge eating in the underweight state compared with those who develop binge eating at normal weight (i.e., not during episodes of AN); and, (4) to examine the specific associations of SUD with binge eating and purging. A large international collaboration involving sites in North America and Europe whose goal is to identify susceptibility genes associated with eating disorders[14] served as the sample for the current study, and subsequently represents the largest study to date examining the phenotypic comorbidity of SUD in AN.

Method

Participants

Participants were drawn from the International Price Foundation Genetic Study of AN Trios sample, which recruited male and female probands affected with AN from nine sites in North America and Europe. Self-report, clinical, and blood sample data were collected on affected probands. The study was approved by the appropriate ethical review boards and all participants completed written informed consent before participation. A detailed description of the study design is described elsewhere.[15]

For the current study, only female participants were included as the number of males was too small for independent analysis or for inclusion as a control for sex effects, resulting in a final sample size of $N = 731$. Data on eating disorders and substance use were collected from self-report and clinical assessments. Blood sample data were not used.

Measures

Demographic and Clinical Variables. Demographic information included age at time of interview, duration of eating disorders, and highest and lowest self-reported lifetime body mass index (BMI kg/m^2).

Eating Disorder Diagnosis. Modified lifetime history of eating disorders (i.e., amenorrhea criterion not required) was assessed using the Structured Inventory of Anorexia Nervosa and Bulimic Syndromes (SIAB-EX), a semi-structured clinical interview designed to establish DSM-IV and ICD-10 eating disorder diagnoses[16] and with an expanded version of Module H of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID).[17] Information regarding recovery status was obtained from the SCID and information regarding presence or absence of component features of eating disorder psychopathology (e.g., restrained eating, binge eating, purging) was obtained from the SIAB-EX.

TABLE 1. Demographic characteristics across AN subtypes

	RAN (n = 328)	PAN (n = 184)	BAN (n = 109)	ANBN (n = 110)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Age at interview (years)	24.7 (7.4)	26.4 (7.0)	27.5 (8.5)	28.5 (8.8)
Highest lifetime BMI (kg/m^2)	20.9 (2.3)	21.3 (2.4)	21.0 (2.2)	22.9 (2.1)
Lowest lifetime BMI (kg/m^2)	13.5 (1.8)	13.8 (1.9)	13.5 (2.0)	14.4 (1.8)
Eating disorder duration (years)	7.7 (6.7)	9.5 (6.7)	10.2 (8.6)	11.2 (8.9)

Participants were classified into one of four AN diagnostic subgroups based on lifetime history: (1) AN–restricting (RAN)–restricted food intake without purging behavior; (2) AN–purging (PAN)–met diagnostic criteria for AN and reported purging behavior without binge eating; (3) AN–binge eating (BAN)–met criteria for AN and reported binge eating with or without purging; and (4) AN–bulimia nervosa (ANBN)–lifetime history of AN and normal weight BN.

In addition to diagnostic subtypes, we assessed the relation between SUD and purging behavior by dividing the sample by presence or absence of lifetime purging as determined by responses to the SCID and SIAB-EX. Individuals defined as reporting lifetime purging reported vomiting, or use of enemas, ipecac, laxatives, and/or diuretics at the subthreshold (less than twice a week) or threshold level (at least twice a week for at least 3 months).

Substance Use. Lifetime alcohol abuse and dependence were assessed using the SCID.[18] Two groups were created based on DSM-IV criteria for alcohol abuse or dependence[19]: (1) no alcohol abuse/dependence; and (2) alcohol abuse/dependence.

Lifetime drug abuse and dependence were assessed using the following categories of items from the SCID: cannabis, sedatives, stimulants, opiates, hallucinogens, cocaine, and other substances. Abuse and dependence were assessed if participants reported using a substance more than 10 times per month (no duration criterion required). Three groups were created: (1) those who reported never having tried drugs or only having tried a substance once were classified in the "no use or experimenters" group; (2) those engaging in drug use more than 5 times but not meeting DSM-IV criteria for abuse/dependence were categorized in the "drug use" group; and, (3) individuals meeting criteria for drug abuse or dependence were categorized in the "drug abuse/dependence" group.

Statistical Analyses

Analyses were conducted using SAS version 9.1[20,21] with the GENMOD procedure. Age at time of interview was entered as a covariate. Prevalence of substance use was calculated across AN subtypes. Logistic regression analysis was used to test for significant differences in prevalence of substance use across AN subtypes. Substance use was examined with reference to purging status and binge eating status. All reported p-values were adjusted for multiple testing using the method of false discovery rate.[22]

Results

Demographic Variables

Table 1 describes the demographic variables across AN subtypes. Participants ranged in age from 13 to 58 years with a mean age of 26.7 years. The mean for highest BMI ranged from 20.9 to 22.9 kg/m^2 and the mean for lowest BMI ranged from 13.5 to 14.4 kg/m^2. Participants in the RAN group were the youngest at the time of interview (i.e., 24.7 years) and those in the ANBN group were the oldest at time of interview (i.e., 28.5 years). Average eating disorder duration was longest for those in the ANBN group (i.e., 11.2 years) and shortest for those in the RAN group (i.e., 7.7 years).

Eating Disorder Subtypes

The RAN group (n = 328) comprised 44.9% of the sample, the PAN group (n = 184) comprised 25.2% of the sample, 14.9% was comprised of the BAN (n = 109) group, and 15.0% of the sample was comprised of the ANBN group (n = 110).

Substance Use across AN Subtypes

Prevalence of alcohol and drug use and abuse/dependence across AN subtypes is presented in **Table 2** and, because of space limitations, **Table 3** presents only the statistically significant odds ratios for AN subtypes pairwise comparisons.

Alcohol Use. Across the total sample, 19.8% met criteria for lifetime history of alcohol abuse/dependence. Prevalence was highest in ANBN (35.5%) and lowest in RAN (13.7%).

Risk for alcohol abuse/dependence was 3.20 times greater in the ANBN group and 1.85 times greater in the BAN group than the RAN group. Risk for alcohol abuse/dependence was 2.24 times

TABLE 2. Prevalence of alcohol abuse/dependence, drug use and abuse/dependence, and drug use by drug category across an subtypes

	RAN (n = 328)	PAN (n = 184)	BAN (n = 109)	ANBN (n = 110)	χ^2 (p) df = 3
	n (%)	n (%)	n (%)	n (%)	
Alcohol Use					
No Abuse/dependence	281 (85.7)	147 (79.9)	83 (76.1)	70 (63.6)	20.29 (<.001)
Abuse/dependence	45 (13.7)	35 (19.0)	26 (23.9)	39 (35.5)	
Drug Use and Drug Abuse/Dependence					
No use/experimenters	231 (70.4)	103 (56.0)	62 (56.9)	45 (40.9)	37.62 (<.001)
Drug use	76 (23.2)	55 (29.9)	28 (25.7)	30 (27.3)	
Abuse/dependence	21 (6.4)	26 (14.1)	19 (17.4)	35 (31.8)	
Drug Use by Drug Category*					
Sedatives	10 (3.0)	13 (7.1)	13 (11.9)	17 (15.5)	16.55 (<.001)
Cannabis	93 (28.4)	64 (34.8)	40 (36.7)	60 (54.5)	20.44 (<.001)
Stimulants	9 (2.7)	11 (6.0)	12 (11.0)	19 (17.3)	23.19 (<.001)
Opioids	9 (2.7)	10 (5.4)	11 (10.1)	11 (10.0)	11.26 (<.010)
Cocaine	11 (3.4)	17 (9.2)	13 (11.9)	18 (16.4)	18.50 (<.001)
Hallucinogens	26 (7.9)	23 (12.5)	13 (11.9)	25 (22.7)	15.82 (<.002)
Other	10 (3.0)	16 (8.7)	7 (6.4)	17 (15.5)	17.96 (<.001)

* Drug use by drug category is defined as having used the substance more than once regardless of abuse or dependence. Only endorsement values are listed.

greater in the ANBN group compared with the PAN group [χ^2 (3, N = 725) = 20.29, $p < .001$].

Drug Use. Across the total sample, 25.9% reported lifetime drug use and an additional 13.8% met criteria for lifetime history of drug abuse/dependence. The RAN group had the lowest percentage of drug use (i.e., 23.2%) and drug abuse/dependence (i.e., 6.4%), the PAN group had the highest percentage of drug use (i.e., 29.9%), and the ANBN group had the highest percentage of drug abuse/dependence (31.8%; **Table 2**).

Across AN subtypes, statistically significant differences emerged in the proportion of individuals meeting criteria for drug use [χ^2 (3, N = 730) = 37.62, $p < .001$]. Individuals in the ANBN group were 1.93 times more likely to report any drug use in comparison to individuals in the RAN group. The risk for drug abuse/dependence was 6.25 times greater in individuals with ANBN, 2.84 times greater in individuals with BAN, and 2.27 times greater in individuals with PAN, compared with those in the RAN group. Also, risk for drug abuse/dependence was 2.78 and 2.17 times greater in those with ANBN compared with those with PAN and BAN, respectively.

Drug Use Category. **Table 2** presents the prevalence for each drug category across AN subtypes. Because abuse/dependence prevalence was low for the drug categories across subtypes, we combined the drug use and abuse/dependence into one group. Across AN subtypes, cannabis was the most frequently reported drug used. Analyses revealed statistically significant differences in all drug use categories across AN subtypes (**Table 2**).

TABLE 3. Statistically significant odds ratios for alcohol abuse/dependence, drug use, drug abuse/dependence, and drug use by drug category by an subtypes

Substance	AN Subtypes Comparison	Odds Ratio (95% CI)
Alcohol		
Alcohol abuse/dependence	BAN > RAN	1.85 (1.80–2.00)
Alcohol abuse/dependence	ANBN > RAN	3.20 (1.92–5.33)
Alcohol abuse/dependence	ANBN > PAN	2.24 (1.30–3.84)
Drug Use and Drug Abuse/Dependence		
Drug Use (no abuse/Dependence)	ANBN > RAN	1.93 (1.13–3.29)
Drug abuse/dependence	PAN > RAN	2.27 (1.22–4.17)
Drug abuse/dependence	BAN > RAN	2.84 (1.47–5.56)
Drug abuse/dependence	ANBN > RAN	6.25 (3.45–11.11)
Drug abuse/dependence	ANBN > PAN	2.78 (1.53–5.00)
Drug abuse/dependence	ANBN > BAN	2.17 (1.14–4.17)
Drug Use by Drug Category		
Sedatives*	BAN > RAN	3.69 (1.54–8.79)
Sedatives*	ANBN > RAN	4.72 (2.06–10.81)
Cannabis*	ANBN > RAN	2.82 (1.79–4.43)
Cannabis*	ANBN > PAN	2.17 (1.33–3.52)
Cannabis*	ANBN > BAN	2.04 (1.18–3.51)
Stimulants*	BAN > RAN	4.00 (1.62–9.34)
Stimulants*	ANBN > RAN	6.55 (2.84–15.12)
Stimulants*	ANBN > PAN	3.05 (1.38–6.74)
Opiates*	BAN > RAN	3.76 (1.50–9.39)
Opiates*	ANBN > RAN	3.64 (1.44–9.15)
Cocaine*	PAN > RAN	2.78 (1.27–6.09)
Cocaine*	BAN > RAN	3.51 (1.51–8.15)
Cocaine*	ANBN > RAN	5.89 (2.21–10.85)
Hallucinogens*	ANBN > RAN	3.51 (1.91–6.46)
Hallucinogens*	ANBN > PAN	2.09 (1.12–3.92)
Hallucinogens*	ANBN > BAN	2.19 (1.05–4.56)
Other*	PAN > RAN	2.94 (1.30–6.64)
Other*	ANBN > RAN	5.44 (2.38–12.42)
Other*	ANBN > BAN	2.62 (1.04–6.61)

* Drug use by drug category is defined as having used the substance more than once regardless of abuse or dependence.

AN subtypes pairwise comparison odds ratio by drug use categories are presented in **Table 3**. Risk for all drug use categories was greatest in the

TABLE 4. Prevalence of alcohol and drug use by purging status and odds ratios with 95% confidence intervals

	No Purging Behavior (n = 335)	Purging Behavior (n = 386)	χ^2 (p)	Odds Ratio
	n (%)	n (%)	df = 1	(95% CI)
Alcohol Use				
No abuse/dependence	287 (85.7)	290 (75.1)	9.70 (<.002)	–
Abuse/dependence	48 (14.3)	96 (24.9)		1.83 (1.24–2.70)
Drug Use				
No use/experimenters	238 (70.6)	199 (51.2)	32.25 (<.001)	–
Use	77 (22.9)	112 (28.8)		1.67* (1.18–2.37)
Abuse/dependence	22 (6.5)	78 (20.0)		3.79* (2.26–6.35)

* Comparisons are made with respect to the No use/experimenters group.
Note: Dashes indicate reference group.

TABLE 5. Prevalence of alcohol abuse/dependence, drug use group and abuse/dependence of specific drug categories across binge eating status

	No Binge Eating (n = 522)	Binge Eating at Low Weight (n = 151)	Binge Eating at Recovered Weight (n = 22)	χ^2 (p)	Odds Ratio (95% CI)
	n (%)	n (%)	n (%)	df = 12	
Alcohol Use					
No abuse/dependence	435 (84.0)	16 (72.7)	107 (71.3)	10.85 (<.005)	–
Alcohol abuse/dependence	83 (16.0)	43 (28.3)	6 (27.3)		1.99 (1.29–3.06)
Drug Use and Drug Abuse/dependence					
No use/experimenters	341 (65.3)	11 (50.0)	177 (51.0)	14.54 (<.001)	–
Use	133 (25.5)	5 (22.7)	80 (26.5)		4.03 (1.41–11.53)
Abuse/dependence	48 (9.2)	6 (27.3)	34 (22.5)		2.79 (1.67–4.07)

Note: Dashes indicate reference group.

ANBN group and lowest in the RAN group. Those in the ANBN group were 6.55 times more likely to report stimulant use, 5.89 times more likely to report cocaine use, 4.72 times more likely to report sedative use, and 5.44 times more likely to report 'other' drug use compared with those in the RAN group. See **Table 3** for a complete list of all pair wise comparisons.

Substance Use by Lifetime Purging Status

Prevalence and odds ratios of substance use by purging status are presented in **Table 4**. Lifetime purging behavior was endorsed by 53.5% of the sample. Lifetime purging was associated with greater alcohol abuse/dependence [χ^2 (1, N = 720) = 9.70, p = .002]. Those in the purging group were 1.83 times as likely to meet criteria for alcohol abuse/dependence compared with the no purging group. The relation between drug abuse/dependence and purging behavior was also statistically significant [χ^2 (1, N = 725) = 32.25, $p < .001$], with those in the purging group 1.67 times more likely to be at risk for drug use and 3.79 times more likely to meet criteria for drug abuse/dependence compared to the no purging group.

Substance Use by Lifetime Binge Eating Status

Prevalence of substance use by lifetime binge eating status is presented in **Table 5**. The relation between substance use categories and binge eating was examined by creating a binge-eating grouping variable: (1) no history of binge eating (n = 522; 71.4% of the sample); (2) history of binge eating at low weight (n = 151; 20.6% of the sample); and (3) history of binge eating only at normal weight (i.e., not during an episode of AN; n = 22; 3.0% of the sample).

Statistically significant differences [χ^2 (2, N = 689) = 10.85, p = .005] emerged across the binge eating groups for alcohol abuse/dependence. Compared with the no binge eating group, risk for alcohol abuse/dependence was 1.99 times more likely in the binge eating at low weight group. No significant differences in alcohol abuse/dependence were found for the binge eating at low weight group and the binge eating at normal weight group.

Those in the binge eating at low weight group were approximately 2.79 times more likely to report drug abuse/dependence and those in the binge eating at normal weight group were 4.03 times more likely to report drug use compared with the no history of binge eating group [χ^2 (2, N = 668) = 14.54,

$p < .001$]. There were no statistically significant differences for drug use between the two binge eating groups.

Discussion

This is the first study to examine the comorbidity between alcohol and drug use disorders and AN by investigating the prevalence of SUD across AN subtypes, by comparing individuals who report binge eating in the underweight state versus those who develop binge eating at normal weight (i.e., not during episodes of AN), and across the entire sample stratified by the presence of binge eating and purging. Our findings indicate that (1) SUD are most common among individuals with the ANBN subtype; (2) those who endorse purging behavior have higher rates of SUD compared with those who do not report purging; and (3) the prevalence of drug use differs across binge eating status.

Although our observed prevalence of SUD was higher than other AN samples,[3,9,10,23–25] which could be explained by definitional issues (i.e., we used a subthreshold, broader definition for SUD) or cross-cultural differences in SUD—our findings are consistent with several previous studies reporting that individuals with BN or with a history of bulimic symptoms during the course of AN were more likely to report SUD compared with those with RAN.[8–11,26–30] Specifically, in the current study, ANBN participants reported significantly higher levels of drug abuse/dependence than all other AN subtypes, whereas the RAN group had the lowest prevalence of drug abuse/dependence. This finding supports previous research suggesting that the ratio of alcohol abuse/dependence across a sample of inpatient females with BN, ANBN, and RAN was 9:5:1,[10] as well as findings from a community sample with Canadian adolescents reporting that binge eaters, particularly those who compensated, were more likely to report substance use.[26] Similarly, dietary restraint and bulimic symptoms in Latina adolescents were positively correlated with alcohol, tobacco and illicit drug use.[29] Our finding that greater substance use occurred in the purging group suggests that there may be meaningful characteristics associated with classification based on the presence or absence of purging.[12,13]

We also examined the relation of drug use category across AN subtypes and found differences in prevalence across AN subtypes. In the current study, the most frequently used drug was cannabis, followed by hallucinogens. These findings support previous research suggesting that greater pathological eating behavior is associated with not just alcohol and tobacco but also marijuana and other hard substances.[26] The prevalence of cannabis use being the highest is consistent with population norms[31] and previous research suggesting that cannabis is the most frequently reported illicit drug among those with restricting and binge eating/purging symptomatology.[8–11] Our findings are not surprising given that epidemiological data indicate that cannabis is the most frequently reported illicit drug.[31] What is surprising is that results from this study challenge the commonly held belief that individuals with RAN report little drug use.[3] Nonetheless it is important to note that the substance use prevalence reported in this paper may partly reflect normative experimentation with alcohol, tobacco, and illicit substances, particularly during adolescence and young adulthood. Therefore, results should be interpreted with caution.

Unexpectedly, hallucinogen use was the second most commonly reported substance used among those with RAN. Although little research exists on the relation between hallucinogen use and eating disorder symptoms, it is possible that the appetite-suppressing effect of hallucinogens,[32] along with the physiological response of changes in perceptions and thoughts which might allow one to "escape" the anxiety associated with the eating disorder, are motivating factors among those who restrict food intake. These motivations may be further encouraged given the availability of some hallucinogens on the internet.[33]

Elaborating on the association between SUD and AN within the current study, findings also suggest that a higher proportion of individuals in the BAN and ANBN groups reported sedative, stimulant and cocaine use compared with those in the RAN group, with those in the ANBN reporting the greatest use. This finding supports previous research suggesting that cocaine and amphetamine use is greater primarily in individuals with the binge eating/purging subtype of AN.[30] The use of stimulants and cocaine among the BAN and ANBN groups may be due to the appetite suppressant effects these drugs can have; thus, their use may be an effort to avoid the consequences of overeating. It has also been hypothesized that an association between SUD and eating disorders reflects an underlying influence of personality traits such as heightened impulsivity.[34,35] Our study was not, however, designed to identify mechanisms underly-

ing the relation between SUD and binge eating/purging AN.

Of final note, our findings suggest that binge eating was related to substance use under certain conditions. Risk for alcohol abuse/dependence and drug abuse/dependence was higher in those who reported binge eating at low weight relative to those who did not binge eat, but differences did not emerge between those who binge ate at low weight versus binge ate only after weight restoration. These findings are inconsistent with previous research suggesting that risk for excessive alcohol consumption is higher in individuals who exhibit binge eating at low weight compared with those who develop binge eating after restoration of normal weight.[6] This inconsistency could be due to differences across study design (i.e., longitudinal vs. cross-sectional), and to our smaller sample size. It has also been suggested that reward hypersensitivity may be a common vulnerability factor for both hazardous drinking and disordered eating.[36–38]

Limitations

Limitations to our study must be considered. First, participants are primarily of European ancestry and therefore cannot be generalized to other ancestry groups or to males. Second, a healthy control group was not available for comparison and our primary interest was examining differences in SUD across the AN subtypes. Third, we did not make distinctions between current and lifetime diagnoses for AN. It is possible that differences in substance use patterns would have emerged had such information been available. Fourth, substance use data focused on lifetime use only, not frequency and duration of use which could differentiate AN subgroups in unique ways. Additionally, the categorization of drug use is a potential limitation in that the "drug use" group included those who used a substance at least twice but not enough to meet criteria for abuse/dependence. Thus, results related to the "drug use" group need to be interpreted given this knowledge. Finally, causal conclusions pertaining to the development of either eating disorders or SUD cannot be discussed. The nature of any casual relation between AN and SUD is unknown, thus we are unable to ascertain if the eating disorder lead to SUD or if the SUD lead to eating disorder symptomatology. Further, we are not aware if certain substances were specifically used for weight-loss. Additional unexamined factors may have influenced findings. Of particular relevance are data suggesting that depression, negative affect, and anxiety are associated with eating disorders and SUD.[39,40] Future studies should consider depression, negative affect, and anxiety as each relates to the comorbidity of eating disorders and SUD.

Conclusions and Implications

Results from this and other studies highlight the need to assess alcohol and drug use behavior when screening and treating individuals with AN, particularly those with bulimic symptomatology. Our findings support previous research indicating that individuals with lifetime diagnoses of both AN and BN (ANBN group in the current analysis) and those who engage in bulimic behaviors report more alcohol abuse/dependence and drug abuse/dependence than those who only engage in restricting behaviors. These findings are of clinical importance because prior research indicates that individuals presenting with both an eating disorder and SUD may be at heightened risk for physical health complications, including increased lethality,[41] and additional psychological comorbidities.[7] Given the comorbidity between AN and SUD, particularly among those with ANBN and BAN, it would appear prudent and necessary to combine prevention and treatment efforts to better avert the emergence or advancement of these disorders.

The authors thank the Price Foundation for the support of the clinical collection of participants and support of data analysis. The authors acknowledge the staff of the Price Foundation Collaborative Group for their efforts in participant screening and clinical assessments. The authors also thank Xiaofei Mo, M.D. for her assistance in the preparation of this article. The authors are indebted to the participating families for their contribution of time and effort in support of this study.

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