Drug and Alcohol Dependence 212 (2020) 107981

Contents lists available at ScienceDirect

Drug and Alcohol Dependence

journal homepage: www.elsevier.com/locate/drugalcdep

Full length article

Eating disorders in a community-based sample of women with alcohol use disorder and nicotine dependence

Melissa A. Munn-Chernoff[a,*], Lauren R. Few[b], Camden E. Matherne[a], Jessica H. Baker[a], Vera Yu Men[c], Vivia V. McCutcheon[b], Arpana Agrawal[b], Kathleen K. Bucholz[b], Pamela A.F. Madden[b], Andrew C. Heath[b], Alexis E. Duncan[b,d]

[a] Department of Psychiatry, University of North Carolina at Chapel Hill, 101 Manning Drive, CB 7160, Chapel Hill, NC 27599 USA
[b] Department of Psychiatry, Washington University School of Medicine, 660 South Euclid, CB 8134, St. Louis, MO 63110 USA
[c] Department of Social Work and Social Administration, University of Hong Kong, Pokfulam, Hong Kong
[d] George Warren Brown School of Social Work, Washington University, One Brookings Drive, CB 1196, St. Louis, MO 63130 USA

ARTICLE INFO

Keywords:
Alcohol
Nicotine
Eating disorder symptoms
Anorexia nervosa
Bulimia nervosa
Purging disorder

ABSTRACT

Background: Studies consistently report a higher prevalence of substance use disorders (SUDs) among women with eating disorders than control women. However, limited research exists on the prevalence of eating disorder *symptoms* and diagnoses in women with SUDs, especially in community-based populations. We examined the prevalence of eating disorder symptoms and diagnosis by the presence or absence of lifetime alcohol use disorder (AUD) and/or nicotine dependence (ND) in a community-based sample of women.

Methods: 3756 women (median age = 22 years) from the Missouri Adolescent Female Twin Study completed a modified semi-structured interview assessing lifetime DSM-IV psychiatric disorders and SUDs. Logistic regression models adjusted for demographic characteristics and other psychopathology, and robust standard errors accounted for the non-independence of twin data.

Results: In general, women with comorbid AUD and ND had a higher prevalence of eating disorder symptoms and diagnoses than women with AUD or ND Only, who in turn had a higher prevalence than those without either SUD. After adjustment for covariates, women with AUD and ND had significantly greater risk of broad anorexia nervosa (RRR = 3.17; 99 % CI = 1.35, 7.44), purging disorder (2.59; 1.24, 5.43), and numerous eating disorder symptoms than women with neither disorder. Significant differences emerged between individuals with both AUD and ND versus women with AUD Only or ND Only for some eating disorder symptoms.

Conclusions: Women with lifetime AUD or ND diagnoses are at high risk for eating disorder symptoms and diagnoses, underscoring the importance of assessing eating disorder symptoms among women with these disorders.

1. Introduction

The association of alcohol abuse or dependence (alcohol use disorder; AUD) with nicotine dependence (ND) is well established. The National Epidemiologic Study of Alcohol and Related Conditions estimates that 34.5 % of adults with past 12-month DSM-IV AUD met criteria for past 12-month DSM-IV ND, and 22.8 % of those with past 12-month ND also met past 12-month criteria for AUD (Grant et al., 2004a). Eating disorders frequently co-occur with both AUD (Gadalla and Piran, 2007; Holderness et al., 1994; Hudson et al., 2007; Root et al., 2010) and ND (Anzengruber et al., 2006; Holderness et al., 1994; Solmi et al., 2016), especially in women. Indeed, AUD and ND have been most closely associated with forms of eating disorders that feature binge eating (i.e., eating a large amount of food in a short period of time while experiencing loss of control) and/or compensatory behaviors (e.g., self-induced vomiting, laxative misuse), such as bulimia nervosa (BN) and the binge-eating/purging subtype of anorexia nervosa (AN) (Anzengruber et al., 2006; Peveler and Fairburn, 1990; Schuckit et al., 1996; Smink et al., 2012). Numerous mechanisms underlying this comorbidity have been proposed, including impulsivity and trauma (Wolfe and Maisto, 2000). Recent studies indicate shared genetic risk for AUD and ND with binge eating (see Munn-Chernoff and Baker, 2016 for a review) and AN (Munn-Chernoff et al., in press), and similar patterns of brain activation between individuals with substance use

* Corresponding author.
E-mail address: melissa_chernoff@med.unc.edu (M.A. Munn-Chernoff).

https://doi.org/10.1016/j.drugalcdep.2020.107981
Received 16 October 2019; Received in revised form 17 March 2020; Accepted 22 March 2020
Available online 25 April 2020
0376-8716/

disorders (SUDs) and eating disorders have emerged (Kaye et al., 2013; Volkow et al., 2013). Finally, emotion dysregulation—the inability to effectively regulate emotional stimuli or negative affect—is present in individuals with SUDs and eating disorders, and may contribute to their comorbidity (Prefit et al., 2019; Sloan et al., 2017). Thus, understanding specific associations and, ultimately, possible mechanisms influencing the comorbidity between AUD/ND and eating disorders, will assist in refining the treatment of this comorbid presentation.

Several gaps remain in the literature on AUD, ND, and comorbid AUD and ND in relation to eating disorders. First, few studies have comprehensively examined associations of AUD or ND with *eating disorder symptoms rather than with diagnoses* (Baker et al., 2010; von Ranson et al., 2002). Although a higher prevalence of AUD and ND has been reported in individuals with BN and with the binge-eating/purging type of AN (Root et al., 2010), associations of AUD and/or ND with specific eating disorder symptoms may have gone undetected in studies solely focusing on eating disorder diagnoses. Indeed, prior studies have not included individuals with an otherwise specified feeding or eating disorder, which is the most frequent eating disorder diagnosis (Le Grange et al., 2012; Thomas et al., 2009). Second, with one exception (Sinha et al., 1996), research has only examined associations of eating disorder diagnoses and symptoms with either AUD or ND alone, independent of the other diagnosis. Given that AUD and ND frequently co-occur, it is important to understand if eating disorder diagnoses and symptoms are associated with AUD or ND specifically or with SUDs in general. Finally, prior studies have not accounted for comorbid psychopathology in examining associations of eating disorders with AUD or ND. Major depression, anxiety disorders, other SUDs (e.g., cannabis use disorder), and conduct disorder are overrepresented among individuals with eating disorders (Dani and Harris, 2005; Grant et al., 2004b,Grant et al., 2004; Hasin et al., 2007; Hudson et al., 2007; Swanson et al., 2011). Therefore, without considering comorbid psychopathology, it is difficult to determine whether associations of eating disorder symptoms/diagnoses with AUD and/or ND are specific to these SUDs or instead reflect a general propensity for people with eating disorder symptomatology to have additional psychiatric diagnoses.

Information about the co-occurrence of SUDs and eating disorders is critical for treatment and prevention efforts, since the co-occurrence of AUD/ND and eating disorders is associated with increased morbidity and mortality (Duncan et al., 2006; Franko et al., 2005, 2013), and improvement in one disorder may influence the symptom severity of the other disorder (Center on Addiction and Substance Abuse, 2003). Thus, we comprehensively explored interrelationships of AUD and ND with eating disorder symptoms and diagnoses by investigating the prevalence and risk of eating disorder symptoms and diagnoses among individuals with AUD and/or ND in a population-based sample of women. We investigated AUD and ND because they frequently co-occur (Falk et al., 2006), were legal substances in all 50 states in the United States at the time of data collection, and are the SUDs most frequently examined for their association with eating disorders (Gadalla and Piran, 2007; Solmi et al., 2016). Furthermore, we examined whether additional psychiatric and SUD diagnoses influenced the association between AUD/ND and eating disorder symptoms and diagnoses. Since a substantial proportion of individuals who present with psychiatric disorders, including SUDs and eating disorders, do not receive treatment (Grant et al., 2004b; Hasin et al., 2007; Hudson et al., 2007), using data from a community-based sample may more accurately explicate the nature of these associations and increase generalizability to a broader population. Because the majority of previous studies on this comorbidity have included young adult or adult women, our findings will allow for a comparison to and extension of prior research, providing an avenue to home in on possible mechanisms underlying SUD and eating disorder comorbidity.

2. Materials and methods

2.1. Participants

Participants were 3756 young adult female twins from the Missouri Adolescent Female Twin Study (MOAFTS) with data on lifetime DSM-IV alcohol abuse/dependence and ND (Heath et al., 2002; Waldron et al., 2013). The MOAFTS is a population-based longitudinal survey of female twins born between July 1st 1975 and June 30th 1985 in the state of Missouri to a state resident mother. Twins were recruited using a cohort sequential sampling design, with ascertainment of successive 6-month cohorts of 13-, 15-, 17-, and 19-year-old twin pairs over a two-year period and continued recruitment of 13-year-olds over several years. The MOAFTS sample is demographically representative of the Missouri population during the years the twins were born, with approximately 15 % of twins being African American and nearly all remaining twins being of European descent, as reported by the mother at the time of birth. A baseline interview was conducted with twins in 1995 (median age = 15 years; IQR: 13–17 years; range: 12–23 years). When possible, interviews were also conducted with at least one parent (usually the mother) at the time the twins entered the study. The first full-length young adult follow-up interview (Wave 4; median age = 22 years; IQR: 19–24 years; range: 18–29 years) was conducted an average of six years after the baseline assessment. Since all members of the target cohort were ≥18-years-old and study participation was no longer contingent upon parental consent at the time of the Wave 4 data collection, all twins from the original sampling frame were invited to participate, even if they had not participated at baseline. Only those twins who themselves had refused future contact or whose parents had refused all future contact with family members were excluded from being recontacted at Wave 4. A summary of response rates is provided elsewhere (Waldron et al., 2013). The Washington University School of Medicine Institutional Review Board approved the study protocol, and all twins gave verbal informed consent before study participation.

2.2. Measures

The baseline assessment did not include eating disorders; hence, we used Wave 4 data for the current analyses. A modified version of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994) was administered to participants via telephone. The SSAGA assessed alcohol and nicotine use, eating disorders, and other psychiatric disorders. In this study, lifetime assessments of DSM-IV (American Psychiatric Association, 1994) AUD and ND—the primary substance use outcomes—and eating disorder symptoms and diagnoses were included. Importantly, for specific eating disorder symptoms, this section of the interview did not include skip patterns, allowing for ample investigation of all symptoms regardless of the presence of clinical diagnosis. AN symptoms included having a lifetime body mass index (BMI) less than minimally expected (i.e., a BMI in the 10th percentile based on age- and sex-specific percentiles from the Center for Disease Control and Prevention; Kuczmarski et al., 2002); intense fear of gaining weight; weight and shape disturbance, undue influence of body weight or shape, or denial of the seriousness of the disorder; and amenorrhea. BN symptoms included ever engaging in binge eating (overeating and loss of control); purging type (i.e., self-induced vomiting and laxative and diuretic misuse) and non-purging type (i.e., strict dieting, fasting, and excessive exercise) compensatory behaviors; and undue influence of body weight and shape (see Duncan et al., 2007 and Munn-Chernoff et al., 2015 for details).

We also included the eating disorder diagnoses of narrow AN, loose AN, narrow BN, loose BN, and purging disorder (PD). Narrow AN and BN were defined as meeting all DSM-IV AN or BN diagnostic criteria, respectively. Loose AN was defined as meeting DSM-IV AN criteria except amenorrhea, which was removed from the definition of AN in the DSM-5 (American Psychiatric Association, 2013), and loose BN had

a self-reported frequency of binge eating a minimum of one day per week, consistent with the DSM-5, and compensatory behaviors reported as "sometimes" or "often" for a minimum of three months. PD, an otherwise specified feeding or eating disorder in the DSM-5, was defined as: 1) recurrent self-induced vomiting, laxative use, or diuretic use in the absence of binge eating; 2) an intense fear of weight gain or becoming fat; 3) overeating less than one day per month; and 4) no lifetime diagnosis of DSM-5 AN or BN. Binge-eating disorder was not included due to low prevalence (n = 4). Ages of onset were not available for some eating disorder symptoms and diagnoses so we could not examine temporal patterns of onset with either SUD.

Multiple covariates were included in this study. Demographic variables included age, African American race, BMI (calculated as weight [in kilograms] / height [in meters] squared), early menarche (defined as onset before age 12 years; Anderson et al., 2003), and maternal educational level (a proxy for socioeconomic status). Diagnoses, rather than symptom counts, of additional lifetime DSM-IV psychiatric disorders assessed by the interview (major depression, social phobia, panic disorder, conduct disorder, and cannabis use disorder) were included as covariates in the analyses.

2.3. Statistical analyses

Data preparation was conducted in SAS (SAS, 1999), and analyses were performed in Stata (StataCorp, 2005) using a robust variance estimator to adjust for the non-independence of twin data (Rao and Scott, 1984). Multinomial logistic regression models were used to obtain relative risk ratios (RRR) for a four-level AUD/ND dependent variable (i.e., comorbid AUD and ND [AUD + ND], AUD Only, ND Only, and neither AUD or ND [referent group]) for each eating disorder symptom and diagnosis independent variable. We tested two models for each independent variable: Model 1 was an unadjusted model, whereas Model 2 adjusted for demographic characteristics (i.e., age at interview, African American race, BMI, early menarche, and maternal educational level) and lifetime psychopathology (i.e., major depression, social phobia, panic disorder, conduct disorder, and cannabis use disorder). Post hoc tests for each model indicated whether significant differences between the three substance use groups for any eating disorder variable existed. Given the large number of tests performed, we used a threshold of $p < 0.01$ for statistical significance and reported 99 % confidence intervals (CIs) for the multinomial logistic regression models.

3. Results

Two hundred and seventy-nine (7.42 %) women met criteria for both lifetime AUD and ND (AUD + ND), 508 (13.52 %) met criteria for lifetime AUD Only, 375 (9.98 %) reported lifetime ND Only, and 2594 (69.06 %) women did not meet criteria for a lifetime diagnosis of either AUD or ND. With the exception of early onset menarche (omnibus p = 0.013; Table 1), significant differences between groups emerged for all demographic characteristics, as well as psychiatric disorders and SUDs (omnibus p-values < 0.01; Table 1).

The prevalence of all eating disorder symptoms and diagnoses differed significantly by lifetime AUD/ND group (omnibus p-values < 0.01; Table 2). For each symptom or diagnosis, the prevalence was highest among women with AUD + ND and lowest among those with neither disorder, with prevalence among those in the AUD Only and ND Only groups in between and more similar to one another than to the prevalence in the AUD + ND and no AUD or ND groups. There was large ($r \geq 0.60$) overlap of some eating disorder symptoms, and the correlations between these symptoms and the two SUDs were small to moderate, ranging from 0.09 to 0.28 (Table S1). Correlations between eating disorder diagnoses and AUD ranged from 0.17 to 0.28, and those of eating disorder diagnoses with ND ranged from 0.14 to 0.28 (Table S2).

In unadjusted multinomial logistic regression models (Table 3; Model 1), all *AN symptoms* were significantly positively associated with lifetime AUD + ND, AUD Only, and ND Only, relative to no AUD or ND. The lone exceptions were intense fear of gaining weight predicting AUD Only (RRR = 1.32, 99 % CI: 0.94, 1.86) and amenorrhea predicting ND Only (RRR = 1.64, 0.85, 3.17). Based on post hoc test results, the RRRs predicting AUD + ND were significantly greater than those predicting AUD Only for all symptoms of AN except amenorrhea and significantly greater than those for ND Only for weight/shape disturbance, undue influence or denial, which also differed significantly from that predicting AUD Only, and amenorrhea. All *BN symptoms* were significantly positively associated with AUD + ND and AUD Only, whereas these symptoms were not consistently associated with ND Only. Binge eating, laxative use, diuretic use, and excessive exercise (RRRs ranged from 1.32 to 2.04) were not associated with ND Only. Finally, for *eating disorder diagnoses*, all five disorders were significantly associated with AUD + ND, whereas loose AN was associated with AUD Only (RRR = 2.48; 1.19, 5.15) and ND Only (RRR = 2.70; 1.23, 5.95) and loose BN was associated with AUD Only (RRR = 3.08; 1.03, 9.22).

After adjusting for covariates, the RRRs for AUD + ND for all AN and BN symptoms, with the exception of binge eating and certain types of compensatory behaviors (laxative use, strict dieting, excessive exercise), were attenuated but remained statistically significant. Loose AN (RRR = 3.17; 1.35, 7.44) and PD (RRR = 2.59; 1.24, 5.43) were the only ED diagnoses significantly associated with AUD + ND after adjusting for covariates. Although not statistically significant, there was a two-fold risk of AUD + ND for narrow AN, suggesting a lack of statistical power. The RRRs for both narrow and loosely defined BN were not significant and close to the null value (1.00), indicating that there was truly no association. All AN symptoms except amenorrhea remained significantly associated with ND Only in the adjusted models; however, associations of BN symptoms and eating disorder diagnoses with ND Only were no longer significant. Only purging type compensatory behaviors (RRR = 1.80; 1.14, 2.86) and diuretic use (RRR = 2.43; 1.22, 4.85) remained significantly associated with AUD Only.

Post hoc tests of differences in RRRs predicting different SUD groups revealed significant differences for AN symptoms, BN symptoms, and eating disorder diagnoses after adjusting for covariates (Table 3, Model 2). For *AN symptoms*, there was a significantly elevated risk of reporting BMI less than minimally expected (Model 2 RRR [99 % CI]: 2.86 [1.67, 4.90] vs. 1.36 [0.84, 2.20]) and weight/shape disturbance (Model 2 RRR [99 % CI]: 2.40 [1.65, 3.49] vs. 1.32 [0.99, 1.76]) in AUD + ND versus AUD Only, as well as weight/shape disturbance in AUD + ND versus ND Only (Model 2 RRR [99 % CI]: 2.40 [1.65, 3.49] vs. 1.49 [1.09, 2.03]). For *BN symptoms*, there was a significantly increased likelihood of any compensatory behavior (Model 2 RRR [99 % CI]: 2.09 [1.38, 3.17] vs. 1.34 [0.98, 1.85]) and diuretic use in the AUD + ND versus AUD Only group (Model 2 RRR [99 % CI]: 5.39 [2.68, 10.87)] vs. 2.43 [1.22, 4.85]). Further, there was greater risk for any compensatory behavior (Model 2 RRR [99 % CI]: 2.09 [1.38, 3.17] vs. 1.21 [0.84, 1.74]), purging type compensatory behavior (Model 2 RRR [99 % CI]: 2.79 [1.64, 4.75] vs. 1.26 [0.77, 2.05]), and diuretic use (Model 2 RRR [99 % CI]: 5.39 [2.68, 10.87] vs. 1.92 [0.94, 3.96]) in AUD + ND versus ND Only groups. No significant differences between the AUD Only and ND Only groups for any eating disorder symptom or among the three substance groups for eating disorder diagnoses emerged.

To investigate the role of demographic characteristics and comorbid psychiatric disorders on the association between eating disorder symptoms/diagnoses and SUD groups, we conducted multinomial logistic regression analyses in a stepwise manner (Table S3). All demographic characteristics were initially included, followed separately by major depressive disorder, social phobia, panic disorder, and conduct disorder. The pattern of results for demographic-adjusted models remained similar to the unadjusted models, and in general, the individual inclusion of each additional psychiatric disorder did not affect the overall association between eating disorder symptoms/diagnoses and AUD and ND groups.

Table 1
Descriptive information on demographic and psychiatric and substance use variables included as covariates by lifetime alcohol use disorder (AUD) and nicotine dependence (ND) status [results shown as n (%)].

	Total Sample	AUD + ND (n = 279)	AUD Only (n = 508)	ND Only (n = 375)	No AUD or ND (n = 2594)	Omnibus p-value
Demographics						
Monozygotic	2013 (53.61)	157 (56.27)	269 (52.95)	208 (55.47)	1379 (53.18)	.717
African American	548 (14.59)	18 (6.45)	45 (8.86)	30 (8.00)	455 (17.55)	< **0.001**
Current BMI						**0.002**
Underweight	244 (6.52)	20 (7.19)	26 (5.13)	22 (5.91)	176 (6.81)	
Normal weight	2332 (62.30)	169 (60.79)	355 (70.02)	211 (56.72)	1597 (61.76)	
Overweight	677 (18.09)	53 (19.06)	80 (15.78)	68 (18.28)	476 (18.41)	
Obese class I	287 (7.64)	15 (5.40)	31 (6.11)	35 (9.41)	206 (7.97)	
Obese class II	118 (3.14)	12 (4.32)	6 (1.18)	22 (5.91)	78 (3.02)	
Obese class III	85 (2.26)	9 (3.24)	9 (1.78)	14 (3.76)	53 (2.05)	
Early menarche (before age 12)	744 (19.94)	52 (18.64)	89 (17.62)	97 (26.15)	506 (19.64)	0.013
Maternal education						< **0.001**
Less than high school	400 (10.65)	34 (12.19)	45 (8.86)	82 (21.87)	239 (9.21)	
High school or college	3269 (87.03)	239 (85.66)	454 (89.37)	282 (75.20)	2294 (88.43)	
Missing	87 (2.32)	6 (2.15)	9 (1.77)	11 (2.93)	61 (2.35)	
Psychiatric and Substance Use Disorder History (Lifetime)						
Major depression	737 (19.64)	137 (49.46)	117 (23.03)	118 (31.47)	365 (14.08)	< **0.001**
Social phobia	481 (12.81)	60 (21.51)	57 (11.22)	101 (26.93)	263 (10.14)	< **0.001**
Panic disorder	92 (2.45)	26 (9.32)	16 (3.16)	18 (4.80)	32 (1.23)	< **0.001**
Conduct disorder	76 (2.02)	25 (9.03)	20 (3.94)	13 (3.47)	18 (0.69)	< **0.001**
Cannabis use disorder	139 (3.70)	56 (20.14)	33 (6.50)	28 (7.47)	22 (0.85)	< **0.001**

Note: BMI = body mass index. Obese class I includes individuals with a BMI of 30 to < 35, obese class II includes individuals with a BMI of 35 to < 40, and obese class III includes individuals with a BMI of 40 or higher. Survey commands in Stata (StataCorp, 2005) were used for analyzing bivariate associations. Under the survey command, the Pearson chi-square statistic is corrected for the twin sampling design using the Rao and Scott (Rao and Scott, 1984) second order correction, converting the Pearson chi-square into an F statistic. Significant differences among groups are indicated by bold type.

Table 2
Number (%) of women reporting lifetime eating disorder symptoms and diagnoses by lifetime alcohol use disorder (AUD) and nicotine dependence (ND) status.

	Total Sample	AUD + ND (n = 279)	AUD Only (n = 508)	ND Only (n = 375)	No AUD or ND (n = 2594)	Omnibus p-value
Anorexia Nervosa Symptoms						
BMI less than minimally expected	336 (8.98)	54 (19.57)	52 (10.26)	53 (14.21)	177 (6.85)	< **0.001**
Intense fear of gaining weight	604 (16.11)	83 (29.96)	86 (16.93)	89 (23.73)	346 (13.36)	< **0.001**
Weight/shape disturbance, undue influence, or denial	1129 (30.17)	149 (53.79)	174 (34.32)	145 (38.87)	661 (25.57)	< **0.001**
Amenorrhea	174 (4.64)	32 (11.55)	36 (7.09)	20 (5.33)	86 (3.32)	< **0.001**
Bulimia Nervosa Symptoms						
Binge eating	106 (2.84)	15 (5.43)	23 (4.54)	14 (3.76)	54 (2.10)	< **0.001**
Any compensatory behavior	794 (21.16)	116 (41.73)	128 (25.20)	99 (26.40)	451 (17.40)	< **0.001**
Purging type	331 (8.82)	63 (22.66)	62 (12.20)	40 (10.67)	166 (6.40)	< **0.001**
Vomiting	212 (5.65)	40 (14.39)	39 (7.68)	28 (7.47)	105 (4.05)	< **0.001**
Laxative use	88 (2.34)	14 (5.04)	20 (3.94)	9 (2.40)	45 (1.74)	< **0.001**
Diuretic use	132 (3.52)	31 (11.15)	25 (4.92)	17 (4.53)	59 (2.28)	< **0.001**
Non-purging type	698 (18.59)	96 (34.53)	110 (21.65)	88 (23.47)	404 (15.58)	< **0.001**
Strict dieting	388 (10.34)	49 (17.63)	70 (13.78)	48 (12.80)	221 (8.52)	< **0.001**
Fasting	322 (8.58)	57 (20.50)	55 (10.83)	45 (12.00)	165 (6.36)	< **0.001**
Excessive exercise	408 (10.87)	51 (18.35)	69 (13.58)	45 (12.00)	243 (9.37)	< **0.001**
Undue influence of weight/shape	1052 (28.01)	124 (44.60)	155 (30.51)	136 (36.27)	637 (24.58)	< **0.001**
*Eating Disorder Diagnoses**						
Narrow anorexia nervosa	43 (1.15)	11 (4.01)	9 (1.78)	4 (1.07)	19 (0.74)	< **0.001**
Loose anorexia nervosa	100 (2.68)	22 (8.03)	20 (3.95)	16 (4.29)	42 (1.63)	< **0.001**
Narrow bulimia nervosa	26 (0.70)	6 (2.17)	6 (1.18)	3 (0.81)	11 (0.43)	**0.004**
Loose bulimia nervosa**	34 (0.91)	6 (2.18)	9 (1.78)	4 (1.08)	15 (0.58)	**0.007**
Purging disorder***	133 (3.57)	24 (8.76)	20 (3.95)	16 (4.29)	73 (2.84)	**0.001**

Note: BMI = body mass index. BMI less than minimally expected was defined as a lifetime BMI in the 10th percentile based on age- and sex-specific percentiles from the Center for Disease Control and Prevention (Kuczmarski et al., 2002). *There was an insufficient sample size of binge-eating disorder (n = 4) to examine among the substance use disorder groups. **Self-reported frequency of binge eating on at least one day per week and compensatory behaviors "sometimes" or "often" for a duration of at least three months. ***Defined as (1) recurrent self-induced vomiting, laxative use, or diuretic use in the absence of binge eating; (2) an intense fear of weight gain or becoming fat; (3) overeating < 1 day per month; and (4) no lifetime diagnosis of DSM-5 anorexia nervosa or bulimia nervosa. Survey commands in Stata (StataCorp, 2005) were used for analyzing bivariate associations. Under the survey command, the Pearson chi-square statistic is corrected for the twin sampling design using the Rao and Scott (Rao and Scott, 1984) second order correction, converting the Pearson chi-square into an F statistic. Significant differences among groups are indicated by bold type.

Table 3
Multinomial logistic regression models (RRR (99 % CI)) for associations between lifetime eating disorder symptoms and diagnoses and lifetime alcohol use disorder (AUD) and/or nicotine dependence (ND).

	Model 1[A]			Model 2[B]		
	AUD + ND	AUD Only	ND Only	AUD + ND	AUD Only	ND Only
Anorexia Nervosa Symptoms						
BMI less than minimally expected	**3.31[a]**	**1.55[a]**	**2.25**	**2.86[a]**	1.36[a]	**2.26**
	(2.11, 5.18)	**(1.01, 2.40)**	**(1.47, 3.46)**	**(1.67, 4.90)**	(0.84, 2.20)	**(1.38, 3.69)**
Intense fear of gaining weight	**2.77[a]**	1.32[a]	**2.02**	**1.93**	1.21	**1.50**
	(1.90, 4.04)	(0.94, 1.86)	**(1.42, 2.86)**	**(1.24, 3.01)**	(0.83, 1.75)	**(1.04, 2.16)**
Weight/shape disturbance, undue influence, or denial	**3.39[ab]**	**1.52[a]**	**1.85[b]**	**2.40[ab]**	1.32[a]	**1.49[b]**
	(2.43, 4.73)	**(1.16, 1.99)**	**(1.38, 2.49)**	**(1.65, 3.49)**	(0.99, 1.76)	**(1.09, 2.03)**
Amenorrhea	**3.81[b]**	**2.23**	1.64[b]	**2.06**	1.78	1.15
	(2.17, 6.69)	**(1.30, 3.80)**	(0.85, 3.17)	**(1.04, 4.08)**	(0.97, 3.27)	(0.58, 2.29)
Bulimia Nervosa Symptoms						
Binge eating	**2.69**	**2.22**	1.83	1.13	1.84	1.02
	(1.23, 5.84)	**(1.17, 4.23)**	(0.83, 4.03)	(0.45, 2.84)	(0.89, 3.82)	(0.43, 2.43)
Any compensatory behavior	**3.40[ab]**	**1.60[a]**	**1.70[b]**	**2.09[b]**	1.34	1.21[b]
	(2.38, 4.86)	**(1.19, 2.15)**	**(1.21, 2.39)**	**(1.38 3.17)**	(0.98, 1.85)	(0.84, 1.74)
Purging type	**4.28[ab]**	**2.03[a]**	**1.75[b]**	**2.79[b]**	**1.80**	1.26[b]
	(2.75, 6.67)	**(1.34, 3.09)**	**(1.09, 2.80)**	**(1.64, 4.75)**	**(1.14, 2.86)**	(0.77, 2.05)
Vomiting	**3.98[ab]**	**1.97[a]**	**1.91[b]**	**2.08**	1.58	1.21
	(2.32, 6.82)	**(1.19, 3.26)**	**(1.09, 3.36)**	**(1.11, 3.92)**	(0.90, 2.78)	(0.67, 2.21)
Laxative use	**3.00**	**2.32**	1.39	1.42	1.70	0.80
	(1.29, 7.01)	**(1.11, 4.84)**	(0.54, 3.59)	(0.53, 3.81)	(0.73, 3.96)	(0.30, 2.16)
Diuretic use	**5.39[ab]**	**2.22[a]**	2.04[b]	**5.39[ab]**	**2.43[a]**	1.92[b]
	(2.91, 9.98)	**(1.18, 4.20)**	(0.99, 4.20)	**(2.68, 10.87)**	**(1.22, 4.85)**	(0.94, 3.96)
Non-purging type	**2.86[ab]**	**1.50[a]**	**1.66[b]**	**1.63**	1.20	1.14
	(1.99, 4.11)	**(1.09, 2.05)**	**(1.17, 2.37)**	**(1.06, 2.51)**	(0.85, 1.68)	(0.78, 1.66)
Strict dieting	**2.30**	**1.72**	**1.58**	1.34	1.32	1.11
	(1.45, 3.64)	**(1.17, 2.52)**	**(1.01, 2.47)**	(0.79, 2.28)	(0.87, 1.98)	(0.69, 1.77)
Fasting	**3.80[ab]**	**1.79[a]**	**2.01[b]**	**1.92**	1.42	1.28
	(2.46, 5.84)	**(1.17, 2.72)**	**(1.27, 3.18)**	**(1.11, 3.30)**	(0.89, 2.27)	(0.79, 2.07)
Excessive exercise	**2.17**	**1.52**	1.32	1.34	1.26	0.96
	(1.38, 3.43)	**(1.04, 2.23)**	(0.83, 2.09)	(0.78, 2.28)	(0.84, 1.88)	(0.59, 1.56)
Undue influence of weight/shape	**2.47[a]**	**1.35[a]**	**1.75**	**1.80**	1.29	1.31
	(1.76, 3.46)	**(1.02, 1.77)**	**(1.28, 2.38)**	**(1.21, 2.69)**	(0.96, 1.73)	(0.94, 1.82)
*Eating Disorder Diagnoses**						
Narrow anorexia nervosa	**5.63**	2.44	1.46	2.15	1.67	0.89
	(2.02, 15.69)	(0.86, 6.93)	(0.34, 6.19)	(0.60, 7.65)	(0.52, 5.37)	(0.19, 4.21)
Loose anorexia nervosa	**5.27**	**2.48**	**2.70**	**3.17**	1.98	2.22
	(2.56, 10.81)	**(1.19, 5.15)**	**(1.23, 5.95)**	**(1.35, 7.44)**	(0.91, 4.32)	(0.97, 5.08)
Narrow bulimia nervosa	**5.18**	2.79	1.89	1.04	1.39	0.66
	(1.38, 19.37)	(0.75, 10.40)	(0.35, 10.21)	(0.17, 6.43)	(0.32, 6.14)	(0.11, 3.83)
Loose bulimia nervosa**	**3.80**	**3.08**	1.85	0.93	1.71	0.71
	(1.08, 13.36)	**(1.03, 9.22)**	(0.43, 7.96)	(0.18, 4.91)	(0.48, 6.14)	(0.15, 3.43)
Purging disorder***	**3.29[a]**	1.41[a]	1.53	**2.59**	1.24	1.21
	(1.73, 6.26)	(0.73, 2.72)	(0.75, 3.14)	**(1.24, 5.43)**	(0.60, 2.56)	(0.59, 2.50)

Note: RRR = relative risk ratios; BMI = body mass index. BMI less than minimally expected was defined as a lifetime BMI in the 10th percentile based on age- and sex-specific percentiles from the Center for Disease Control and Prevention (Kuczmarski et al., 2002). Significant RRR relative to the no AUD or ND group are indicated by bold type. [a]Across rows and within model type, AUD + ND and AUD Only groups are significantly different from each other at $p < 0.01$. [b]Across rows and within model type, AUD + ND and ND Only groups are significantly different from each other at $p < 0.01$. There were no significant differences between the AUD Only and ND Only groups. *Unfortunately, there was an insufficient sample size of binge-eating disorder ($n = 4$) to examine among the substance use disorder groups. **Self-reported frequency of binge eating on at least one day per week and compensatory behaviors "sometimes" or "often" for a duration of at least three months. ***Defined as (1) recurrent self-induced vomiting, laxative use, or diuretic use in the absence of binge eating; (2) an intense fear of weight gain or becoming fat; (3) overeating < 1 day per month; and (4) no lifetime diagnosis of DSM-5 anorexia nervosa or bulimia nervosa. [A]Unadjusted model. [B]Adjusted for age at interview, African American race, BMI, early menarche, maternal educational level, major depression, social phobia, panic disorder, conduct disorder, and cannabis use disorder.

Finally, we conducted sensitivity analyses including only individuals who had tried alcohol and nicotine in their lifetime ($N = 2569$; Tables S4-S6). In general, the pattern of associations between the four SUD groups were the same as the full sample. Notably, the overall prevalence of eating disorder symptoms and diagnoses was higher in the restricted sample than the full sample, indicating that individuals who have at least tried alcohol or nicotine may also engage in disordered eating behaviors more frequently than individuals who have not tried alcohol or nicotine in their lifetime.

4. Discussion

In this community-based sample of young adult women, women who met criteria for a lifetime diagnosis of both AUD and ND had the highest prevalence of all eating disorder symptoms and diagnoses. Conversely, women who met criteria for neither AUD nor ND had the lowest prevalence of all eating disorder symptoms and diagnoses. The prevalence of eating disorder symptoms and diagnoses among women with AUD Only was generally similar to that among women with ND Only. However, relative to no AUD or ND, AN symptoms were also associated with ND Only but not AUD Only, whereas purging-type

compensatory behaviors were associated with AUD Only but not ND Only. After adjusting for covariates, the greatest risks were for women with both AUD and ND relative to those with neither disorder, and RRRs for AUD + ND in some instances were significantly greater than the AUD Only and ND Only groups. Although these findings may reflect a general comorbidity issue, such that individuals with two SUDs could have more additional psychopathology than those with only one SUD, they also highlight the importance of screening for eating disorder symptoms and diagnoses among individuals with AUD who are also heavy smokers. This is especially critical in treatment settings, where changes in SUD symptoms may exacerbate or weaken symptoms of an eating disorder (Center on Addiction and Substance Abuse, 2003).

These findings corroborate existing research suggesting that the association between SUDs and some eating disorder pathology may be partly due to other psychiatric disorders (e.g., Baker et al., 2007; Bulik et al., 2004; Dansky et al., 2000), as seen by the attenuation of the magnitude of the RRRs after adjusting for demographic characteristics and comorbid psychopathology. For example, loosely defined AN was significantly associated with all three SUD groups before adjusting for demographic characteristics and additional psychopathology; however, after adjusting for these comorbidities, the RRRs decreased in strength and only the association with AUD + ND remained statistically significant. The same pattern held true for PD, consistent with prior work (Munn-Chernoff et al., 2015) suggesting that the elevated risk for combined AUD and ND among women with PD is independent of additional psychiatric comorbidities. The current findings further support the importance of examining compensatory behaviors as key eating disorder symptoms driving the association between SUDs and eating disorders, whereas little support for binge eating as a key symptom in this association emerged after adjusting for additional psychopathology. Although this latter finding was unexpected, findings from our unadjusted models were consistent with those of previous studies that did not adjust for comorbid psychopathology (Gadalla and Piran, 2007; Holderness et al., 1994). Thus, these results suggest that additional psychopathology does not fully account for the relationship, since some eating disorder symptoms and diagnoses remained significantly associated with AUD and ND ever after adjusting for these additional psychiatric disorders.

In general, women in the ND Only group had significantly greater risk of AN symptoms but *not* binge eating or any compensatory behaviors, the hallmark features of BN. This is consistent with research showing that women use nicotine as a means to control weight (White, 2012) and suppress appetite. Our findings are in slight contrast with a recent meta-analysis reporting an increased prevalence of lifetime nicotine use among individuals with BN or binge-eating disorder than AN (Solmi et al., 2016). The difference in results indicate that the association could be reflective of the presence of an AUD, as we found a significant association for both AUD and ND with specific BN symptoms.

This study significantly expands our understanding of the comorbid presentation of SUDs and eating disorders. Use of a female-specific sample is consistent with prior studies on this comorbidity, which allowed us to compare and expand our findings to obtain a more detailed description of associations of AUD and ND with eating disorder diagnoses and symptoms in women. We compared two SUDs to evaluate whether associations with eating disorder pathology were similar across substances. Further, we included a range of eating disorder diagnoses *as well as* symptoms, providing tailored information for prevention and treatment programs. No skip patterns were included in the diagnostic interview; thus, eating disorder symptoms were asked regardless of eating disorder diagnosis. Nonetheless, there were some limitations. Although this study was one of the largest to examine the prevalence and risk of eating disorder symptomatology and diagnoses in individuals with SUDs, we may have been underpowered to detect some significant associations. Second, because the data for this study came from a population-based sample, findings may not generalize to a clinical population, as only a fraction of persons with eating-related pathology receive treatment (Hudson et al., 2007). Similarly, these findings may not generalize to men or other age groups. Third, these data reflect lifetime symptoms and diagnoses, and temporal assessment of symptom onset is unclear. Thus, it is unknown whether the presence or severity of eating disorder symptoms fluctuated with onset of AUD or ND. Finally, we could not examine binge-eating disorder due to the small sample size (n = 4).

In sum, women who reported lifetime AUD and ND had the highest prevalence of eating disorder symptoms and diagnoses. Women with both AUD and ND had an increased risk of reporting loosely defined AN and PD, as well as other AN and BN symptoms, than control women. Women with ND only were more likely to report AN symptoms, whereas women who had AUD only were more likely to report engaging in any compensatory behaviors than women with neither AUD or ND. These findings indicate that eating disorders *and their symptoms* are prevalent among individuals with SUDs and substance-specific associations with individual eating disorder *symptoms* exist. This information is critical for treatment by reinforcing the need for assessing eating disorder pathology among individuals with an AUD or ND. For example, these findings may provide additional support for the use of Dialectical Behavior Therapy in treating comorbid AUD/ND and eating disorders. This therapy, which focuses on improving emotion regulation and distress tolerance, has been shown to be effective for SUDs (Maffei et al., 2018), eating disorders (Pisetsky et al., 2019), and possibly their comorbid presentation (Cavicchioli et al., n.d.; Courbasson et al., 2012; Grilo et al., 2002). Future studies should use alternative methods, including identifying latent dimensions of AUD, ND, and eating disorder comorbidity, to explicate which symptom profiles may co-occur, as well as how these profiles may be associated with comorbid psychiatric symptoms and personality traits associated with SUD and eating disorder comorbidity. More targeted research on eating disorder symptoms and substance-specific associations will allow clinicians and treatment providers to be vigilant for screening of eating disorders among individuals with substance use problems.

Role of funding source

This work is supported by NIH grants: K01 AA025113 (MMC); F32 AA023693 (LRF); R01 DA023668, R21 AA021235 (AA); and R01 HD049022, R01 AA009022, R01 AA017915, R01 AA021492, R37 AA007728, P50 AA011988, and K05 AA017688 (ACH). MMC was also supported by T32 AA007580 (ACH).

Contributors

Drs. Munn-Chernoff and Duncan managed the literature searches, were responsible for the study concept and design, and wrote the first draft of the manuscript. Drs. Munn-Chernoff, Few, and Duncan performed the statistical analyses. Ms. Men and Dr. McCutcheon assisted with the data analyses. Drs. Bucholz, Madden, and Heath designed the study from which data were drawn and oversaw data collection. All authors provided input on the study design and edited the manuscript. All authors have approved the final version for publication.

Declaration of competing interest

None.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.drugalcdep.2020.107981.

References

American Psychiatric Association, 1994. 4th ed. Diagnostic and Statistical Manual of Mental Disorders Vol. 4th American Psychiatric Press, Washington, DC.

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders Vol. 5th American Psychiatric Publishing, Arlington, Virginia.

Anderson, S.E., Dallal, G.E., Must, A., 2003. Relative weight and race influence average age at menarche: results from two nationally representative surveys of US girls studied 25 years apart. Pediatrics 111, 844–850.

Anzengruber, D., Klump, K.L., Thornton, L., Brandt, H., Crawford, S., Fichter, M.M., Halmi, K.A., Johnson, C., Kaplan, A.S., LaVia, M., Mitchell, J., Strober, M., Woodside, D.B., Rotondo, A., Berrettini, W.H., Kaye, W.H., Bulik, C.M., 2006. Smoking in eating disorders. Eat. Behav. 7, 291–299.

Baker, J.H., Mazzeo, S.E., Kendler, K.S., 2007. Association between broadly defined bulimia nervosa and drug use disorders: common genetic and environmental influences. Int. J. Eat. Disord. 40, 673–678.

Baker, J.H., Mitchell, K.S., Neale, M.C., Kendler, K.S., 2010. Eating disorder symptomatology and substance use disorders: prevalence and shared risk in a population based twin sample. Int. J. Eat. Disord. 43, 648–658.

Bucholz, K.K., Cadoret, R., Cloninger, C.R., Dinwiddie, S.H., Hesselbrock, V.M., Nurnberger Jr., J.I., Reich, T., Schmidt, I., Schuckit, M.A., 1994. A new, semi-structured psychiatric interview for use in genetic-linkage studies: a report on the reliability of the SSAGA. J. Stud. Alcohol 55, 149–158.

Bulik, C.M., Klump, K.L., Thornton, L., Kaplan, A.S., Devlin, B., Fichter, M.M., Halmi, K.A., Strober, M., Woodside, D.B., Crow, S., Mitchell, J.E., Rotondo, A., Mauri, M., Cassano, G.B., Keel, P.K., Berrettini, W.H., Kaye, W.H., 2004. Alcohol use disorder comorbidity in eating disorders: a multicenter study. J. Clin. Psychiatry 65, 1000–1006.

Cavicchioli, M., Ramella, P., Vassena, G., Simone, G., Prudenziati, F., Sirtori, F., Movalli, M., Maffei, C., 2020. Dialectical behaviour therapy skills training for the treatment of addictive behaviours among individuals with alcohol use disorder: the effect of emotion regulation and experiential avoidance. Am. J. Drug Alcohol Abuse. https://doi.org/10.1080/00952990.2020.1712411. (in press).

Center on Addiction and Substance Abuse, 2003. Food for Thought: Substance Abuse and Eating Disorders. Columbia University (Ed.). The National Center on Addiction and Substance Abuse at Columbia University, New York, NY, pp. 1–83.

Courbasson, C., Nishikawa, Y., Dixon, L., 2012. Outcome of dialectical behaviour therapy for concurrent eating and substance use disorders. Clin. Psychol. Psychother. 19, 434–449.

Dani, J.A., Harris, R.A., 2005. Nicotine addiction and comorbidity with alcohol abuse and mental illness. Nat. Neurosci. 8, 1465–1470.

Dansky, B.S., Brewerton, T.D., Kilpatrick, D.G., 2000. Comorbidity of bulimia nervosa and alcohol use disorders: results from the National Women's Study. Int. J. Eat. Disord. 27, 180–190.

Duncan, A.E., Bucholz, K.K., Neuman, R.J., Agrawal, A., Madden, P.A., Heath, A.C., 2007. Clustering of eating disorder symptoms in a general population female twin sample: a latent class analysis. Psychol. Med. 37, 1097–1107.

Duncan, A.E., Neuman, R.J., Kramer, J.R., Kuperman, S., Hesselbrock, V.M., Bucholz, K.K., 2006. Lifetime psychiatric comorbidity of alcohol dependence and bulimia nervosa in women. Drug Alcohol Depend. 84, 122–132.

Falk, D.E., Yi, H.Y., Hiller-Sturmhofel, S., 2006. An epidemiologic analysis of co-occurring alcohol and tobacco use and disorders: findings from the National Epidemiologic Survey on Alcohol and Related Conditions. Alcohol Res. Health 29, 162–171.

Franko, D.L., Dorer, D.J., Keel, P.K., Jackson, S., Manzo, M.P., Herzog, D.B., 2005. How do eating disorders and alcohol use disorder influence each other? Int. J. Eat. Disord. 38, 200–207.

Franko, D.L., Keshaviah, A., Eddy, K.T., Krishna, M., Davis, M.C., Keel, P.K., Herzog, D.B., 2013. A longitudinal investigation of mortality in anorexia nervosa and bulimia nervosa. Am. J. Psychiatry 170, 917–925.

Gadalla, T., Piran, N., 2007. Co-occurrence of eating disorders and alcohol use disorders in women: a meta analysis. Arch. Womens Ment. Health 10, 133–140.

Grant, B.F., Hasin, D.S., Chou, S.P., Stinson, F.S., Dawson, D.A., 2004a. Nicotine dependence and psychiatric disorders in the United States: results from the National Epidemiologic Survery on Alcohol and Related Conditions. Arch. Gen. Psychiatry 61, 1107–1115.

Grant, B.F., Stinson, F.S., Dawson, D.A., Chou, S.P., Dufour, M.C., Compton, W., Pickering, R.P., Kaplan, K., 2004b. Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Arch. Gen. Psychiatry 61, 807–816.

Grilo, C.M., Sinha, R., O'Malley, S.S., 2002. Eating Disorders and Alcohol Use Disorders. Retrieved from. National Institute on Alcohol Abuse and Alcoholism. https://pubs.niaaa.nih.gov/publications/arh26-2/151-160.htm.

Hasin, D.S., Stinson, F.S., Ogburn, E., Grant, B.F., 2007. Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Arch. Gen. Psychiatry 64, 830–842.

Heath, A.C., Howells, W., Bucholz, K.K., Glowinski, A.L., Nelson, E.C., Madden, P.A., 2002. Ascertainment of a mid-western US female adolescent twin cohort for alcohol studies: assessment of sample representativeness using birth record data. Twin Res. 5, 107–112.

Holderness, C.C., Brooks-Gunn, J., Warren, M.P., 1994. Co-morbidity of eating disorders and substance abuse review of the literature. Int. J. Eat. Disord. 16, 1–34.

Hudson, J.I., Hiripi, E., Pope Jr., H.G., Kessler, R.C., 2007. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol. Psychiatry 61, 348–358.

Kaye, W.H., Wierenga, C.E., Bailer, U.F., Simmons, A.N., Wagner, A., Bischoff-Grethe, A., 2013. Does a shared neurobiology for foods and drugs of abuse contribute to extremes of food ingestion in anorexia and bulimia nervosa? Biol. Psychiatry 73, 836–842.

Kuczmarski, R.J., Ogden, C.L., Guo, S.S., Grummer-Strawn, L.M., Flegal, K.M., Mei, Z., Wei, R., Curtin, L.R., Roche, A.F., Johnson, C.L., 2002. 2000 CDC growth charts for the United States: methods and development. Vital Health Stat. 11, 1–190.

Le Grange, D., Swanson, S.A., Crow, S.J., Merikangas, K.R., 2012. Eating disorder not otherwise specified presentation in the US population. Int. J. Eat. Disord. 45, 711–718.

Maffei, C., Cavicchioli, M., Movalli, M., Cavallaro, R., Fossati, A., 2018. Dialectical behavior therapy skills training in alcohol dependence treatment: findings based on an open trial. Subst. Use Misuse 53, 2368–2385.

Munn-Chernoff, M.A., Baker, J.H., 2016. A primer on the genetics of comorbid eating disorders and substance use disorders. Eur. Eat. Disord. Rev. 24, 91–100.

Munn-Chernoff, M.A., Johnson, E.C., Chou, Y.L., Coleman, J.R.I., Thornton, L.M., Walters, R.K., Agrawal, A., in press. Shared genetic risk between eating disorder- and substance-use-related phenotypes: evidence from genome-wide association studies. Addict. Biol. e12880. doi:https://doi.org/10.1111/adb.12880.

Munn-Chernoff, M.A., Keel, P.K., Klump, K.L., Grant, J.D., Bucholz, K.K., Madden, P.A., Heath, A.C., Duncan, A.E., 2015. Prevalence of and familial influences on purging disorder in a community sample of female twins. Int. J. Eat. Disord. 48, 601–6606.

Peveler, R., Fairburn, C., 1990. Eating disorders in women who abuse alcohol. Brit. J. Addict. 85, 1633–1638.

Pisetsky, E.M., Schaefer, L.M., Wonderlich, S.A., Peterson, C.B., 2019. Emerging psychological treatments in eating disorders. Psychiatr. Clin. North Am. 42, 219–229.

Prefit, A.B., Candea, D.M., Szentagotai-Tatar, A., 2019. Emotion regulation across eating pathology: a meta-analysis. Appetite 143, 104438.

Rao, J.N.K., Scott, A.J., 1984. On chi-squared tests for multiway contingency tables with cell proportions estimated from survey data. Ann. Stat. 12, 46–60.

Root, T.L., Pisetsky, E.M., Thornton, L., Lichtenstein, P., Pedersen, N.L., Bulik, C.M., 2010. Patterns of co-morbidity of eating disorders and substance use in Swedish females. Psychol. Med. 40, 105–115.

SAS, 1999. SAS User's Guide [computer program] Version 82. SAS Institute, Inc., Cary, NC.

Schuckit, M.A., Tipp, J.E., Anthenelli, R.M., Bucholz, K.K., Hesselbrock, V.M., Nurnberger Jr., J.I., 1996. Anorexia nervosa and bulimia nervosa in alcohol-dependent men and women and their relatives. Am. J. Psychiatry 153, 74–82.

Sinha, R., Robinson, J., Merikangas, K., Wilson, G.T., Rodin, J., O'Malley, S., 1996. Eating pathology among women with alcoholism and/or anxiety disorders. Alcohol. Clin. Exp. Res. 20, 1184–1191.

Sloan, E., Hall, K., Moulding, R., Bryce, S., Mildred, H., Staiger, P.K., 2017. Emotion regulation as a transdiagnostic treatment construct across anxiety, depression, substance, eating and borderline personality disorders: a systematic review. Clin. Psychol. Rev. 57, 141–163.

Smink, F.R., van Hoeken, D., Hoek, H.W., 2012. Epidemiology of eating disorders: incidence, prevalence and mortality rates. Curr. Psychiatry Rep. 14, 406–414.

Solmi, M., Veronese, N., Sergi, G., Luchini, C., Favaro, A., Santonastaso, P., Vancampfort, D., Correll, C.U., Ussher, M., Thapa-Chhetri, N., Fornaro, M., Stubbs, B., 2016. The association between smoking prevalence and eating disorders: a systematic review and meta-analysis. Addiction 111, 1914–1922.

StataCorp, 2005. Stata Statistical Software [computer program] Release 9. StataCorp. LP, College Station, TX.

Swanson, S.A., Crow, S.J., Le Grange, D., Swendsen, J., Merikangas, K.R., 2011. Prevalence and correlates of eating disorders in adolescents. Results from the National Comorbidity Survey Replication Adolescent Supplement. Arch. Gen. Psychiatry 68, 714–723.

Thomas, J.J., Vartanian, L.R., Brownell, K.D., 2009. The relationship between eating disorder not otherwise specified (EDNOS) and officially recognized eating disorders: meta-analysis and implications for DSM. Psychol. Bull. 135, 407–433.

Volkow, N.D., Wang, G.J., Tomasi, D., Baler, R.D., 2013. The addictive dimensionality of obesity. Biol. Psychiatry 73, 811–818.

von Ranson, K.M., Iacono, W.G., McGue, M., 2002. Disordered eating and substance use in an epidemiological sample: I. Associations within individuals. Int. J. Eat. Disord. 31, 389–403.

Waldron, M., Bucholz, K.K., Lynskey, M.T., Madden, P.A., Heath, A.C., 2013. Alcoholism and timing of separation in parents: findings in a midwestern birth cohort. J. Stud. Alcohol Drugs 74, 337–348.

White, M.A., 2012. Smoking for weight control and its associations with eating disorder symptomatology. Compr. Psychiatry 53, 403–407.

Wolfe, W.L., Maisto, S.A., 2000. The relationship between eating disorders and substance use: moving beyond co-prevalence research. Clin. Psychol. Rev. 20, 617–631.