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Patterns of Comorbidity of Eating Disorders and Substance Use in Swedish Females

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Abstract

Background—Little is known about the association of eating disorder (ED) subtypes across multiple categories of substance use (SU) in population-based samples. We examined the association between EDs and SU in a large population-based sample.

Method—Female participants (*N*=13,297) were from the Swedish Twin Registry (Lichtenstein *et al.*, 2006). SU was examined in four defined groups − (1) anorexia nervosa (AN); (2) bulimia nervosa (BN); (3) anorexia nervosa and bulimia nervosa (ANBN); and (4) binge eating disorder (BED) as well as a referent group without EDs (no ED). Secondary analyses examined differences between restricting AN (RAN) and binge and/or purge AN (ANBP).

Results—In general, EDs were associated with greater SU relative to the referent. The AN group had significantly increased odds for all illicit drugs. Significant differences emerged across the RAN and ANBP groups for alcohol abuse/dependence, diet pills, stimulants, and polysubstance use with greater use in the ANBP group. Across ED groups, (1) the BN and ANBN groups were more likely to report alcohol abuse/dependence relative to the AN group, (2) the ANBN group was more likely to report diet pill use relative to the AN, BN, and BED groups, and (3) the BN group was more likely to report diet pill use relative to the no ED, AN and BED groups.

Conclusions—EDs are associated with a range of SU behaviors. Improved understanding of how they mutually influence risk could enhance understanding of etiology and prevention.

Introduction

Approximately 50% of individuals with an eating disorder (ED) abuse or are dependent on alcohol or illicit substances compared with approximately 9% of the general population (Holderness *et al.*, 1994; The National Center on Addiction and Substance Abuse (CASA at Columbia University, 2003). Of individuals with a substance use disorder, more than 35% report some form of an ED (CASA, 2003) compared to lifetime prevalence estimates of approximately 5% for women in the United States (Hudson *et al.*, 2007). While it has been established that the co-occurrence between EDs and substance use (SU) exists, prevalence varies markedly across studies (Holderness *et al.*, 1994), which is partially attributable to study design and methodology. Most studies have not compared SU across the various subtypes of

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EDs often due to small sample sizes, particularly for anorexia nervosa (AN). Additionally, most research on EDs and SU has focused on alcohol, tobacco, or broadly defined illicit drug use. Finally, few studies include a non-ED control group, which is necessary in order to make meaningful comparisons between those with and without an ED.

Substances associated with EDs include alcohol, tobacco, cannabis, cocaine, heroin, and amphetamines (Blinder *et al.*, 2006; Bulik *et al.*, 2004a; Bulik *et al.*, 2004b; Root *et al.*, in press; CASA, 2003) and patterns of association vary across ED subtypes. Those who endorse binge eating, including those with bulimia nervosa (BN) and a lifetime history of both anorexia nervosa and bulimia nervosa (ANBN), tend to exhibit higher levels of licit and illicit drug use (Herzog *et al.*, 1992; Hudson *et al.*, 2007; Root *et al.*, in press; Ross & Ivis, 1999; CASA, 2003; Wiederman & Pryor, 1996), including use of diet pills (Reba-Harrelson *et al.*, 2008), than individuals with AN or no ED. For example, females with purging symptoms are more likely to report frequent alcohol use and binge drinking than females without ED symptoms (Adams & Araas, 2006). Conversely, females who report alcohol problems and/or binge drinking were more likely to report recent ED symptoms (Field *et al.*, 2002; CASA, 2003; Wiederman & Pryor, 1996). Further, research suggests that those with BN and those with ANBN have a higher prevalence of lifetime alcohol abuse and/or dependence than individuals with AN (Bulik *et al.*, 2004c). However, of those with AN, there is higher prevalence of alcohol abuse and/or dependence in the binge and/or purge subtype of anorexia nervosa (ANBP) than restricting anorexia nervosa (RAN; (Blinder *et al.*, 2006). Finally, those with BN are also more likely to report illicit drug use, particularly amphetamines, barbiturates, marijuana, tranquilizers, and cocaine (CASA, 2003), with the heaviest illicit drug use found among females who binge and purge.

Tobacco use is also more common among individuals with EDs compared to those without (Anzengruber *et al.*, 2006) and, as with alcohol, patterns of use vary across ED category and subtype. Anzengruber and colleagues (2006) found that women with BN or ANBP had a higher prevalence of cigarette use compared to women with RAN, who had a similar prevalence as women without an ED. Although cigarettes are the most studied form of tobacco, cigarette use in Sweden appears to be declining while tobacco use remains constant (Furberg *et al.*, 2006; Wersall & Eklund, 1998). This is partly attributable to the increased use of snus, a form of oral smokeless tobacco that has been popular in Sweden for decades, particularly among men (Furberg *et al.*, 2006), and has recently been test marketed in the United States. Snus is a less harmful and more discreet form of tobacco (Lagergren *et al.*, 2000; Lewin *et al.*, 1998; Osterdahl *et al.*, 2004) and thus may be a more appealing tobacco product. However, to date, no published data have been reported on snus use among those with an ED.

Most research on SU and EDs has focused on either BN or broadly defined AN with few studies comparing SU across subtypes of AN. Root and colleagues (Root *et al.*, in press) examined SU across several AN subtypes and reported differences in prevalence of substance use disorder across AN subtypes, with more in the ANBN group reporting a substance use disorder than those in the RAN and purging AN groups supporting previous research (Eddy *et al.*, 2002). Root et al. (Root *et al.*, in press) also report that cannabis was the most frequently used substance by women with AN, including the RAN group, and that individuals who purged were more likely to report SU than those who did not purge. Although informative, this sample was collected for a study of the genetics of EDs which recruited male and female probands affected with AN. The present study explores these issues in depth in a population-based sample.

The purpose of this study was to extend previous research by examining the prevalence of SU behaviors across ED groups relative to individuals with no history of EDs in a large population-based female sample. Specifically, we: (1) compared the prevalence of SU across four ED groups relative to a non-ED referent; (2) determined whether SU, and specifically, which

substances, are more common in those with ANBP compared to RAN; (3) investigated the prevalence of snus use across ED groups; and (4) conducted pairwise comparisons for each substance across EDs groups to determine which groups report significantly more SU and to also report effect sizes for significant comparisons. Our approach focuses on lifetime history for both EDs and SU. Lifetime use models are a necessary first step in the advancement to more complex models assessing casual mechanisms (Eaves *et al.*, 2004; Kendler & Prescott, 1999). For example, the ultimate liability to alcohol dependence in an individual who has never been exposed to alcohol is unknown. Thus, lifetime use is an important variable for any substance because it is a necessary first step in the path of developing abuse or dependence.

Method

Participants

Participants were drawn from the Screening Twin Adults: Genes and Environment (STAGE) cohort of the Swedish Twin Registry [(STR; (Lichtenstein et al., 2006), a large population-based prospective sample of Swedish twins born 1959–1985 (Lichtenstein *et al.*, 2006). Using web-based questionnaires, (or computer assisted telephone interviews for those preferring this method), data were collected on most common complex diseases including information on EDs and SU. Kappa values were calculated for several components and based on 100 respondents who were first tested using the web-based questionnaire and then retested 2–5 months later using the computer assisted telephone interview for the purpose of assessing test-retest reliability across the two methods. Kappa for the ED section was .76 and for the SU section was .66 suggesting agreement between the web-based questionnaire and the telephone interview. The current study consisted of a total of 13,297 female participants; males were not included given low prevalence of EDs

Measures

Eating Disorder Diagnosis—Lifetime history of broadly defined EDs was assessed using an expanded, on-line Structured Clinical Interview for DSM-IV (SCID) based instrument designed to collect detailed information about course and severity of EDs. Due to low prevalence using narrow definitions, we focused on broadly defined ED groups.

Table I presents criteria used to define ED groups. For the primary analyses, participants were classified into one of five groups based on lifetime history of EDs: (1) no ED; (2) AN; (3) BN; (4) ANBN; and (5) binge eating disorder (BED). For secondary analyses, the AN group was subdivided into RAN and ANBP.

Substance Use—Eighteen SU items from the SCID were included. Information on abuse/dependence was available only for alcohol. Thus, all items were based on use (not abuse or dependence) with the exception of *alcohol abuse/dependence* which required the Diagnostic and Statistical Manual, 4th edition criteria for either abuse or dependence [DSM-IV; (American Psychiatric Association, 2000)]. *Binge drink* was defined as either 4 or more bottles of beer, 4 or more glasses of wine (>60 cl), or 3 or more shots of liquor (>18 cl) at one time. *Occasional smoker* was defined as smoking less than one cigarette per day on average but more than just having tried a cigarette and includes those who report only smoking on weekends. *Regular smoker* was defined as ever having smoked at least once per day. *Occasional snus* was defined as more than just trying snus but less than once per day on average. *Regular snus* was using snus at least once per day. *Diet pills-weekly* was defined as over-the-counter and prescription diet pill use at least once per week. The remaining SU items *cannabis*, *hallucinogens*, *opioids*, *sedatives*, and *stimulants* - were categorized based on two criteria: (1) lifetime use, and (2) used more than 10 times per month. Two variables for each substance were created (e.g., *cannabis 10x/month; cannabis-ever*). Due to low prevalence, *diet pills-ever* and

hallucinogens 10x/month were dropped from the analyses. *Polysubstance 10x/month* was defined as having used at least two illicit substances at least 10x/month. *Polysubstance-ever* was defined as ever having tried at least two illicit substances.

Statistical Analyses

Prevalence was calculated for the ED groups and the referent across all SU measures. Logistic regression analyses were conducted using generalized estimating equations (GEE) with PROC GENMOD to test for statistically significant differences in SU across ED groups. GEE allows for the correlated nature of the twin data to be controlled. Comparisons for each substance across pairs of EDs groups (e.g., AN, BN) were conducted to determine which ED groups reported significantly more SU and to report their associated effect sizes (i.e., odds ratios). In order to assess differences in SU within the AN group, secondary logistic regression analyses were conducted to compare the RAN and ANBP subgroups. Given that there were 18 tests (one for each SU variable), p-values were adjusted using the method of false discovery rate [FDR; (Benjamini & Hochberg, 1995)], which controls for the expected proportion of Type I errors (i.e., rejecting the null hypothesis when it is true). It is the expected proportion of false positives (Type I) among all rejected hypotheses (Type I and Type II) at the desired significance level. For example, in our study an FDR cutoff of 0.05 was selected, implying that we allowed one expected false positive out of 20. All analyses were performed using SAS 9.1 (SAS Institute Inc., 2004).

Results

Ninety-four percent of the sample reported no history of an ED), 3% reported AN (RAN: *n*=197, ANBP:*n*=181), 2% BN, 1% ANBN, and < 1% BED (Table II).

Demographics

Table II presents demographic information across the ED groups and the referent group. Participants ranged in age from 20 to 47 years with a mean age of 34 years (*SD* = 7.66). Mean for highest lifetime adult BMI was greatest among the BED group and mean for lowest lifetime adult BMI was lowest among the ANBN group.

Prevalence of Substance Use across Eating Disorder Groups

Table III presents prevalence estimates of SU across the groups and provides odds ratios for statistically significant pairwise comparisons across ED subtypes. Due to space limitations, we only present the statistically significant pairwise comparisons, however, a complete list of all 130 pairwise comparisons can be found in the supplementary material.

Overall, statistically significant differences for prevalence across groups were found for *alcohol abuse/dependence*, *diet pills-weekly*, *cannabis 10x/month*, *cannabis-ever*, *hallucinogens-ever*, *opioids 10x/month*, *opioids-ever*, *sedatives 10x/month*, *sedatives-ever*, *stimulants 10x/month*, *stimulants-ever*, *polysubstance 10x/month and polysubstance-ever*. No statistically significant differences across groups were found for *binge drink*, *occasional smoker*, *regular smoker*, *occasional snus*, and *regular snus*.

Alcohol—The prevalence of *alcohol abuse/dependence* differed across groups with the AN, BN, and ANBN groups more likely to have had alcohol abuse/dependence relative to the referent. Across ED groups (not relative to the referent), the BN and ANBN groups were more likely to have had alcohol abuse/dependence relative to the AN group. No statistically significant group differences emerged for prevalence of *binge drink*.

Tobacco—No statistically significant group difference emerged for *regular smoker, occasional smoker, occasional snus* or *regular snus*.

Diet Pills—A statistically significant difference across groups was found for *diet pills-weekly*. The AN, BN, and ANBN groups were more likely to use *diet pills-weekly* relative to the referent. Across ED groups (not relative to the referent), (1) the ANBN group was more likely to use *diet pills-weekly* relative to the AN, BN, and BED groups, and (2) the BN group was more likely to use *diet pills-weekly* relative to the AN and BED groups.

Illicit Drugs

Cannabis—Statistically significant group differences were found for *cannabis 10x/month* and *cannabis-ever*. Individuals in the AN and BN groups were more likely to use *cannabis 10x/month* relative to the referent. Those in the AN, BN, ANBN, and BED groups were more likely to use *cannabis-ever* relative to the referent.

Hallucinogens—Statistically significant group differences emerged for the *hallucinogens-ever item* with the AN group more likely to use *hallucinogens-ever* relative to the referent.

Opioids—There was a statistically significantly difference across groups for *opioids 10x/month* and *opioids-ever*. Individuals in the AN group were more likely to use *opioids 10x/month* relative to the referent and the AN and BN groups were more likely to use *opioids-ever* relative to the referent.

Sedatives—Statistically significantly differences across groups were found for *sedatives 10x/month* and *sedatives-ever*. The AN and BN groups were more likely to use *sedative 10x/month* relative to the referent, and the AN, BN, ANBN, and BED groups were more likely to use *sedatives-ever* relative to the referent.

Stimulants—Across groups, statistically significant differences emerged for *stimulants 10x/month* and *stimulants-ever*. The AN group was more likely to use *stimulants 10x/month* relative to the referent. Those in the AN and BN groups were more likely to use *stimulants-ever* compared to the referent.

Polysubstance—Statistically significant differences across groups were found for *polysubstance 10x/month* and *polysubstance-ever*. The AN group was more likely to engage in *polysubstance 10x/month* relative to the referent, and the AN, BN, and ANBN groups were more likely to engage in *polysubstance-ever* relative to the referent.

Secondary Analyses–Substance Use for RAN and ANBP

Prevalence was statistically significantly higher in the ANBP group compared to the RAN group for *alcohol abuse/dependence (15% vs. 9%; p<.05), diet pills-weekly (35% vs. 9%; p<.01), stimulants-ever (12% vs. 5%; p<.01),* and *polysubstance-ever(24% vs. 15%; p<.01).* The ANBP group was 1.93 times more likely than the RAN group to have *alcohol abuse/dependence* (χ^2=4.08, $p\leq$.043; 95% CI: 1.02, 3.66), 5.76 times more likely to use *diet pills-weekly* (χ^2=38.68, $p\leq$.001; 95% CI: 3.22, 10.31), 2.86 times more likely to endorse *stimulants-ever* (χ^2=6.87, $p\leq$.009; 95% CI: 1.28, 6.38), and 1.74 times more likely to engage in *polysubstance-ever* (χ^2=4.40, $p\leq$.036; 95% CI: 1.04, 2.92). No statistically significant differences emerged for the remaining substances,.

Discussion

This study represents the largest and most detailed exploration to date of a wide range of substance use in EDs in a population-based sample of Swedish women and presents novel epidemiologic information on EDs and SU. Three broad themes emerged from the analyses. First, consistent with previous research, the prevalence of SU was higher in all ED groups than the referent indicating that SU is not limited to any particular ED presentation. Second, in contrast with previous studies (Anzengruber *et al.*, 2006), tobacco use was not elevated in women with EDs relative to the referent. Third, the observation that women with AN report elevated SU behaviors challenges previously held beliefs that SU is uncommon in women with AN (Herzog *et al.*, 2006; Wiederman & Pryor, 1996) and the finding that the RAN and ANBP subgroups differed on alcohol abuse/dependence, weekly diet pill use, stimulant use, and polysubstance use adds to the literature examining SU across varying presentations of AN.

Although no differences in prevalence emerged for binge drinking across the ED groups relative to the referent, those in the AN, BN, and ANBN groups were at increased risk for *alcohol abuse/dependence* relative to the referent. Observed prevalence in the BN and ANBN groups (approximately 22%) is consistent with previous research reporting prevalences of alcohol abuse or dependence of 25% for BN and 14% for ANBN (Bulik *et al.*, 2004c). These findings could reflect the elevated relative risk of alcohol use disorders among individuals with BN compared with AN (Kaye *et al.*, 1996; Kaye *et al.*, 1998). However, follow-up analyses revealing that those in the ANBP group were at elevated risk relative to the RAN group suggests that increased risk of alcohol abuse/dependence among those with bulimic symptoms or personality traits such as impulsivity (Bulik *et al.*, 2004c) may also be particular risk factors for the binge/purge subtype of AN. One possible explanation is that SU may reflect attempts to reduce negative affect (i.e., shame and guilt) associated with bingeing and purging (Stice & Shaw, 2002).

For the AN group, additional hypotheses exist. One possible explanation is that the reinforcing efficacy of alcohol and other drugs is enhanced by the food deprivation associated with the illness (Bulik *et al.*, 2004c; Carroll & Meisch, 1984). Additionally, alcohol (and drug) use may assist with the regulation of affect including prominent anxiety symptoms seen in those with AN (Bulik *et al.*, 2004c; Godart *et al.*, 2000). Overall, our finding of *alcohol abuse/dependence* in the AN group, particularly the ANBP group, extends a growing body of literature supporting SU behaviors among women with AN (Bulik *et al.*, 2004c; Root *et al.*, in press; von Ranson *et al.*, 2002).

Regarding tobacco use, in contrast to previous research (Anzengruber et al., 2006), we did not find differences across the groups in cigarette use or snus use. It is possible that as snus use increases in the female population in Sweden, differences across groups may become detectable. It is also possible that tobacco products are being used among women both with and without an ED as a weight control measure thus resulting in no differences between groups.

Consistent with previous research (Reba-Harrelson *et al.*, 2008; Roerig *et al.*, 2003), diet pill use was elevated in all ED groups. The ANBN group was at particularly elevated risk (i.e., approximately 15 times more likely than the referent). Diet pill use has been associated with purging, novelty seeking, and several Axis I and Axis II disorders (Reba-Harrelson *et al.*, 2008) which could explain why both the BN and ANBN groups, who tend to score higher on measures of novelty seeking (Cassin & von Ranson, 2005; Fernandez-Aranda *et al.*, 2006; Reba *et al.*, 2005), reported more use than the AN group.

The very high diet pill use in women with ANBN and the greater risk for diet pill use in the ANBP group compared to the RAN group are consistent with a previous report (Reba-Harrelson *et al.*, 2008) in which diet pill use was similar across purging BN, ANBN, and ANBP.

Because we were not able to assess temporal ordering of SU and ED symptoms, it is not known if the ANBN group used the diet pills during the time of their AN diagnosis, their BN diagnosis, or throughout both illnesses. We speculate that those with ANBN, who by definition have a history of low weight, may seek out more extreme weight loss measures than individuals with BN with no history of AN because they may continue to strive for previously achieved low weight.

Turning to the use of illicit drugs, all ED groups reported greater use of illicit drugs and polysubstance use relative to the referent. Similar prevalences across ED groups for *cannabis-ever*, *hallucinogens-ever*, *stimulants-ever* support previous research (Root *et al.*, in press; Wiederman & Pryor, 1996). Additionally, the BN and ANBN groups had higher risk for several illicit substances including cannabis, opioids, sedatives, and stimulants, which supports previous research (Herzog *et al.*, 2006; Hudson *et al.*, 2007; CASA, 2003), as well as for *polysubstance-ever*.

Unexpectedly, the AN group was at increased risk for all illicit drug use categories and *polysubstance-ever* relative to the referent indicating that drug use is of concern across all ED subtypes (von Ranson *et al.*, 2002). Extending the AN findings further, AN subtype comparisons revealed that *stimulants-ever* was the only illicit substance that was more frequently reported in the ANBP group relative to the RAN group, adding to the literature suggesting that illicit drug use is not limited to those with binge eating or purging subtypes of AN. The attraction of stimulant use in the AN group might in part rest with their appetite suppressant and increased metabolic effects (Hsieh *et al.*, 2005; Hudson *et al.*, 1992; Wiederman & Pryor, 1996) and the attraction of cannabis and sedatives for their sedating effects (Swinbourne & Touyz, 2007)

One possible explanation for increased polysubstance use in those with EDs is the association between polysubstance use and impulsivity (Steiger & Bruce, 2007) and novelty-seeking (Conway *et al.*, 2003) which is also elevated in individuals with EDs, particularly BN. Because individuals with polysubstance use have high rates of psychiatric comorbidity (Lynskey *et al.*, 2006) and often relapse after substance abuse treatment (Marshall, 1994), replication and additional attention to polysubstance use with ED populations is warranted.

Findings that illicit drug use is occurring across ED groups support a study in which greater pathological eating behavior was associated with not just alcohol and tobacco but also cannabis use and other illicit substances (Ross & Ivis, 1999). It is possible these findings can be explained given genetic research associating family history (Lachman, 2006) to both SU and EDs (Bulik *et al.*, 2006; Bulik *et al.*, 2007; Pinheiro *et al.*, 2006; Slof-Op 't Landt *et al.*, 2005). Genetics influence liability to SU disorder with research suggesting that SU among MZ twin pairs is two to four times greater compared to DZ twin pairs (Lachman, 2006). Heritability estimates for alcoholism are often 50% or greater, 40% to 70% for tobacco, and 25% to 80% for other substances including illicit drugs (Prescott *et al.*, 2006). Regarding EDs, family studies have demonstrated that both AN and BN tend to be increased in relatives of affected probands compared to relatives of unaffected probands (Lilenfeld *et al.*, 1998; Pinheiro *et al.*, 2006). Twin studies have reported heritability estimates between 33% and 84% for AN (Bulik *et al.*, 2006; Bulik *et al.*, 2007; Slof-Op 't Landt *et al.*, 2005; Wade *et al.*, 2000) and between 28% to 83% for BN (Bulik *et al.*, 2000) demonstrating considerable genetic effects for AN and BN. However, several lines of evidence suggest that EDs and SU might not necessarily be influenced by shared genetic factors. Kendler and colleagues (Kendler *et al.*, 1995), in a twin study of six major psychiatric illnesses, did not find a strong genetic association between BN and alcoholism. Similarly, Lilenfeld and colleagues (1998) concluded that EDs and alcoholism were not co-transmitted in families. Lastly, Kaye and colleagues (1996) reported that BN and substance use disorders, including alcohol abuse and dependence, were transmitted

independently suggesting that women with BN do not have a familial vulnerability to substance use disorders. In contrast, one study has reported a moderate genetic correlation ($r_a = 0.39$) between broadly defined BN and drug use disorders (Baker *et al.*, 2007). Additional work is required including evaluations of both genetic and environmental factors and their interactions in order to understand both the comorbid profile and familial transmission of EDs and SU.

Limitations

Limitations of our study must be considered. First, results cannot be generalized to other ancestry groups or to males. Second, because the study was not prospective, causal conclusions pertaining to the development of either EDs or SU behaviors cannot be discussed. Third, additional unexamined factors may have influenced findings including factors that may be associated with EDs and substance use disorders. Of particular relevance is research indicating that depression/negative affect (Fernandez-Aranda *et al.*, 2007) and/or anxiety (Bulik *et al.*, 2004c; Godart *et al.*, 2006) are associated with the onset of both EDs and SU. Fourth, one must consider whether any aspects of a twin population could limit generalizability of the observed prevalence of SU and EDs to non-twin samples. By virtue of socializing together, twins may be more likely to be exposed to various substances and behaviors (e.g. one twin's exposure to cigarettes could increase the likelihood of the co-twin trying a cigarette). If this were the case, then we could expect prevalences in twins that are higher than the general population. That does not appear to be the case in these data as, for example, 11% of our sample was classified as regular smokers which is below the reported national prevalence of 20% in Swedish females ages 16–84 (Strong & Bonita, 2003). Nonetheless, correlated exposure among twins and generalizability to non-twin samples is a potential weakness that must be considered when interpreting findings. Fifth, for polysubstance use, we only examined whether individuals were using more than two illicit substances either ever in their lifetime or at least 10 times per month; we did not examine which substances cluster together across individuals, nor did we include alcohol or tobacco use in these analyses. Future research would benefit from examining individuals' substances of choice for polysubstance drug use in order to understand better differences in SU involvement across ED groups. Last, the BED group is quite small in our sample (~4%) which might be due in part to the much lower base rate of obesity in Sweden compared to the U.S. (Neovius *et al.*, 2006; Ogden *et al.*, 2006). As a result of the small sample, not all analyses could be conducted with the BED group. Thus, it is important to interpret the current findings with caution.

Conclusions

Results of this study add to the growing literature on EDs and SU by further emphasizing that EDs may be associated with a range of SU behavior. The STAGE sample used in the current study provided extensive EDs and SU phenotyping, including snus use which has never been reported before. This study represents the first large population-based study that was able to contrast SU patterns across AN, BN, ANBN, and BED. Moreover, our secondary analyses allowed us to explore differences within AN subtypes in a non-treatment-seeking sample. Findings highlight the importance of screening for various types of SU when examining and treating individuals with disordered eating. Although we cannot examine temporal patterns of onset, several possibilities exist, namely, the presence of an ED may increase risk for SU, SU may increase risk for EDs, or a third underlying variable might increase risk for both EDs and SU. Additional investigations incorporating patterns of onset will assist with determining how EDs and SU mutually influence risk (Field *et al.*, 2002; Stice & Shaw, 2002). Presenting comprehensive epidemiologic data in order to characterize the sample fully was a necessary first step toward more advanced twin methodology exploring the complex genetic and environmental factors influencing liability to both traits. This is critically important given the heightened risk for physical complications, including suicide risk, among those with both an ED and substance use disorder (Franko *et al.*, 2005; Keel *et al.*, 2003).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table I

Criteria used for eating disorder diagnosis.

Diagnosis	Criteria*/Item	
Anorexia nervosa (AN)	**1**	BMI < 18.55
	2	Intense fear of gaining weight
	3	Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight
	4	Includes either subtype (i.e., restricting AN and binge and/or purge AN)
Bulimia nervosa (BN)	**1**	Recurrent episodes of binge eating
	2	Recurrent inappropriate compensatory behavior in order to prevent weight gain
	3	Binge eating and compensatory behaviors occur ≥ 4 times in a month
	4	Self-evaluation is unduly influenced by body shape and weight
	5	Disturbance does not occur exclusively during episodes of AN
Anorexia nervosa/bulimia nervosa (ANBN)	Lifetime diagnosis of AN and BN as defined above	
Binge eating disorder (BED)	**1**	Recurrent binge eating characterized by eating an unusually large amount of food in a discrete period of time, and having a sense of loss of control
	2	Marked distress regarding binge eating
	3	Binge eating occurs ≥ 4 times for at least one month (excluded duration of 6 months criteria)
	4	Binge eating not associated with compensatory behaviors
Restricting AN (RAN)	**1**	BMI < 18.55
	2	Intense fear of gaining weight
	3	Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight
	4	No bingeing or purging
Binge and/or purge AN (ANBP)	**1**	BMI < 18.55
	2	Intense fear of gaining weight
	3	Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight
	4	Bingeing and/or purging during low weight

* Based on DSM-IV criteria.

Table II

Demographic characteristics across the four eating disorder groups and the no ED Group.

	No ED (*n* = 12,508) *M* (*SD*)	**AN (*n* = 378) *M* (*SD*)**	**BN (*n* = 267) *M* (*SD*)**	**ANBN (*n* = 95) *M* (*SD*)**	**BED (*n* = 49) *M* (*SD*)**
Age at interview (years)	33.66 (7.66)	31.69 (7.59)	31.65 (7.27)	32.13 (7.23)	31.86 (7.76)
Highest lifetime BMI (kg/m^2)	24.91 (4.45)	23.58 (4.24)	26.03 (5.02)	24.74 (4.91)	28.97 (7.63)
Lowest lifetime BMI (kg/m^2)	19.98 (2.56)	16.45 (1.74)	19.87 (2.95)	16.16 (1.90)	20.87 (2.89)

Note. no ED = no eating disorder, AN = anorexia nervosa, BN = bulimia nervosa, ANBN = lifetime history of AN and BN, BED = binge eating disorder, *M* = mean, *SD* = standard deviation.

Table III

Prevalence of substance use items across eating disorder groups and odds ratios for statistically significant pairwise comparisons.

Substance Use Item	No ED *n* = 12,508	AN *n* = 378	BN *n* = 267	ANBN *n* = 95	BED *N* = 49	Pairwise Comparisons	Odds Ratio (95% CI)
Alcohol abuse/Dependence**	755 (6%)	45 (12%)	59 (22%)	21 (22%)	7 (14%)	AN > no ED	1.90 (1.35, 2.66)**
						BN > no ED	4.29 (3.13, 5.87)**
						ANBN > no ED	4.20 (2.56, 6.89)**
						BN >AN	2.26 (1.44, 3.56)**
						ANBN >AN	2.21 (1.22, 4.02)*
Binge drink	693 (6%)	20 (5%)	20 (7%)	9 (9%)	4 (8%)		
Occasional smoker	2675 (28%)	76 (28%)	68 (33%)	20 (28%)	16 (44%)		
Regular smoker	1372 (14%)	46 (17%)	36 (18%)	14 (20%)	7 (19%)		
Occasional snus	767 (8%)	27 (10%)	18 (9%)	8 (11%)	2 (6%)		
Regular snus	484 (5%)	15 (5%)	9 (4%)	7 (10%)	2 (6%)		
Diet pills-weekly**	1011 (8%)	81 (21%)	98 (37%)	56 (59%)	7 (14%)	AN > no ED	3.06 (2.37 3.95)**
						BN > no ED	6.37 (4.91, 8.27)**
						ANBN > no ED	15.36 (10.19, 23.15)**
						BN > AN	2.08 (1.46, 2.96)**
						ANBN > AN	5.02 (3.12, 8.07)**
						ANBN > BN	2.41 (1.50, 3.89)**
						BN > BED	3.53 (1.50, 8.32)**
						ANBN > BED	8.51 (3.41, 21.24)**
Cannabis 10x/month**	252 (2%)	24 (7%)	14 (6%)	6 (7%)	5 (11%)	AN > no ED	3.23 (2.12, 4.91)**
						BN > no ED	2.72 (1.54, 4.79)*
Cannabis-ever	1865 (15%)	93 (25%)	83 (31%)	25 (26%)	17 (35%)	AN > no ED	1.69 (1.13, 2.15)**
						BN > no ED	2.37 (1.82, 3.10)**
						ANBN > no ED	1.89 (1.17, 3.04)*
						BED > no ED	2.79 (1.55, 5.05)*
Hallucinogens-ever*	221 (2%)	18 (5%)	10 (4%)	5 (5%)	3 (6%)	AN > no ED	2.36 (1.40, 3.97)*
Opioids 10x/month*	216 (2%)	16 (4%)	10 (4%)	6 (7%)	1 (2%)	AN > no ED	2.52 (1.51, 4.21)*
Opioids-ever**	1416 (11%)	70 (19%)	44 (16%)	13 (14%)	9 (18%)	AN > no ED	1.71 (1.31, 2.24)**
						BN > no ED	1.52 (1.09, 2.10)*
Sedatives 10x/month**	70 (1%)	10 (3%)	8 (3%)	4 (5%)	1 (2%)	AN > no ED	5.10 (2.64, 9.89)*
						BN > no ED	5.66 (2.68, 11.97)*
Sedatives-ever**	778 (6%)	58 (15%)	42 (16%)	14 (15%)	10 (20%)	AN > no ED	2.56 (1.91, 3.44)**
						BN > no ED	2.71 (1.93, 3.80)**
						ANBN > no ED	2.61 (1.48, 4.62)*
						BED > no ED	3.62 (1.78, 7.36)*
Stimulants 10x/month*†	40 (<1%)	10 (3%)	2 (1%)	3 (3%)	0	AN > no ED	7.63 (3.74, 15.57)**
Stimulants-ever**	363 (3%)	31 (8%)	25 (9%)	7 (7%)	3 (6%)	AN > no ED	2.76 (1.88, 4.05)**
						BN > no ED	3.33 (2.20, 5.04)**
Polysubstance 10x/month**	85 (1%)	13 (3%)	6 (2%)	4 (4%)	1 (2%)	AN > no ED	5.05 (2.86, 8.91)**
Polysubstance-ever**	1129 (9%)	73 (20%)	58 (22%)	18 (19%)	10 (20%)	AN > no ED	2.24 (1.72, 2.93)**
						BN > no ED	2.67 (1.98, 3.60)**
						ANBN > no ED	2.42 (1.46, 4.03)*

Note. no ED = no eating disorder, AN = anorexia nervosa, BN = bulimia nervosa, ANBN = lifetime history of AN and BN, BED = binge eating disorder, df = degrees of freedom, CI = confidence interval;

** $p < .01$,

*$p < .05$,

†BED group removed from analysis due to an empty cell.