Psychological Medicine (2010), **40**, 105–115. © Cambridge University Press 2009
doi:10.1017/S0033291709005662

ORIGINAL ARTICLE

Patterns of co-morbidity of eating disorders and substance use in Swedish females

T. L. Root[1], E. M. Pisetsky[1], L. Thornton[1], P. Lichtenstein[2], N. L. Pedersen[2,3] and C. M. Bulik[1,4]*

[1] *Department of Psychiatry, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA*
[2] *Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden*
[3] *Department of Psychology, University of Southern California, Los Angeles, CA, USA*
[4] *Department of Nutrition, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA*

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

Method. Female participants ($n = 13\,297$) were from the Swedish Twin Registry [Lichtenstein *et al*., *Twin Research and Human Genetics* (2006) **9**, 875–882]. Substance use was examined in four defined groups – (1) anorexia nervosa (AN); (2) bulimia nervosa (BN); (3) AN and BN (ANBN); and (4) binge eating disorder (BED) as well as a referent group without eating disorder (no ED). Secondary analyses examined differences between restricting AN (RAN) and binge and/or purge AN (ANBP).

Results. In general, eating disorders were associated with greater substance use 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 eating disorder 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. Eating disorders are associated with a range of substance use behaviors. Improved understanding of how they mutually influence risk could enhance understanding of etiology and prevention.

Received 22 August 2008; Revised 16 February 2009; Accepted 21 February 2009; First published online 20 April 2009

Key words: Alcohol, anorexia nervosa, binge-eating disorders, bulimia nervosa, tobacco.

Introduction

Approximately 50% of individuals with an eating disorder 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 at Columbia University, 2003). Of individuals with a substance-use disorder, more than 35% report some form of an eating disorder (The National Center on Addiction and Substance Abuse at Columbia University, 2003) compared with 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 eating disorders and substance use exists, prevalence varies markedly across studies (Holderness *et al*. 1994), which is partially attributable to study design and methodology. Most studies have not compared substance use across the various subtypes of eating disorders often due to small sample sizes, particularly for anorexia nervosa (AN). Additionally, most research on eating disorders and substance use has focused on alcohol, tobacco or broadly defined illicit drug use. Finally, few studies include a non-eating disorder control group, which is necessary in order to make meaningful comparisons between those with and without an eating disorder.

Substances associated with eating disorders include alcohol, tobacco, cannabis, cocaine, heroin and amphetamines (The National Center on Addiction and Substance Abuse at Columbia University, 2003; Bulik *et al*. 2004*a*, *b*; Blinder *et al*. 2006; Root *et al*. 2009) and patterns of association vary across eating disorder

* Address for correspondence: C. M. Bulik, Ph.D., Department of Psychiatry, University of North Carolina at Chapel Hill, 101 Manning Drive, CB #7160, Chapel Hill, NC 27599-7160, USA.
(Email: cbulik@med.unc.edu)

subtypes. Those who endorse binge eating, including those with bulimia nervosa (BN) and a lifetime history of both AN and BN (ANBN), tend to exhibit higher levels of licit and illicit drug use (Herzog *et al*. 1992; Wiederman & Pryor, 1996; Ross & Ivis, 1999; The National Center on Addiction and Substance Abuse at Columbia University, 2003; Hudson *et al*. 2007; Root *et al*. 2009), including use of diet pills (Reba-Harrelson *et al*. 2008), than individuals with AN or no eating disorder. For example, females with purging symptoms are more likely to report frequent alcohol use and binge drinking than females without eating disorder symptoms (Adams & Araas, 2006). Conversely, females who report alcohol problems and/or binge drinking were more likely to report recent eating disorder symptoms (Wiederman & Pryor, 1996; Field *et al*. 2002; The National Center on Addiction and Substance Abuse at Columbia University, 2003). 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*. 2004*a*). However, of those with AN, there is higher prevalence of alcohol abuse and/or dependence in the binge and/or purge subtype of AN (ANBP) than restricting AN (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 (The National Center on Addiction and Substance Abuse at Columbia University, 2003), with the heaviest illicit drug use found among females who binge and purge.

Tobacco use is also more common among individuals with eating disorders compared with those without (Anzengruber *et al*. 2006) and, as with alcohol, patterns of use vary across eating disorder category and subtype. Anzengruber *et al*. (2006) found that women with BN or ANBP had a higher prevalence of cigarette use compared with women with RAN, who had a similar prevalence as women without an eating disorder. Although cigarettes are the most studied form of tobacco, cigarette use in Sweden appears to be declining while tobacco use remains constant (Wersall & Eklund, 1998; Furberg *et al*. 2006). 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 (Lewin *et al*. 1998; Lagergren *et al*. 2000; 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 eating disorder.

Most research on substance use and eating disorders has focused on either BN or broadly defined AN with few studies comparing substance use across subtypes of AN. Root *et al*. (2009) examined substance use 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*. (2009) 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 substance use than those who did not purge. Although informative, this sample was collected for a study of the genetics of eating disorders 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 substance-use behaviors across eating disorder groups relative to individuals with no history of eating disorders in a large population-based female sample. Specifically, we: (1) compared the prevalence of substance use across four eating disorder groups relative to a non-eating disorder referent; (2) determined whether substance use, and specifically, which substances, are more common in those with ANBP compared with RAN; (3) investigated the prevalence of snus use across eating disorder groups; and (4) conducted pairwise comparisons for each substance across eating disorder groups to determine which groups report significantly more substance use and to also report effect sizes for significant comparisons. Our approach focuses on lifetime history for both eating disorders and substance use. Lifetime use models are a necessary first step in the advancement to more complex models assessing casual mechanisms (Kendler & Prescott, 1999; Eaves *et al*. 2004). 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 prerequisite to developing abuse or dependence.

Method

Participants

Participants were 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 between 1959 and 1985 (Lichtenstein *et al*. 2006). Using web-based questionnaires (or

https://doi.org/10.1017/S0033291709005662 Published online by Cambridge University Press

Table 1. *Criteria used for eating disorder diagnosis*[a]

Diagnosis	Criteria/item
Anorexia nervosa	1. BMI <18.55 kg/m^2 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 anorexia nervosa and binge and/or purge anorexia nervosa)
Bulimia nervosa	1. Recurrent episodes of binge eating 2. Recurrent inappropriate compensatory behavior in order to prevent weight gain 3. Binge eating and compensatory behaviors occur at least four times in a month 4. Self-evaluation is unduly influenced by body shape and weight 5. Disturbance does not occur exclusively during episodes of anorexia nervosa
Anorexia nervosa/bulimia nervosa	Lifetime diagnosis of anorexia nervosa and bulimia nervosa as defined above
Binge eating disorder	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 at least four times for at least 1 month (excluded duration of 6 months criteria) 4. Binge eating not associated with compensatory behaviors
Restricting anorexia nervosa	1. BMI <18.55 kg/m^2 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 anorexia nervosa	1. BMI <18.55 kg/m^2 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

BMI, Body mass index.
[a] Based on DSM-IV criteria.

computer-assisted telephone interviews for those preferring this method), data were collected on most common complex diseases including information on eating disorders and substance use. Kappa values were calculated for several components and were 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 eating disorder section was 0.76 and for the substance-use section was 0.66, 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 the low prevalence of eating disorders.

Measures

Eating disorder diagnosis

Lifetime history of broadly defined eating disorders was assessed using an expanded, online Structured Clinical Interview for the Diagnostic and Statistical Manual, 4th edition (DSM-IV) (SCID)-based instrument designed to collect detailed information about course and severity of eating disorders. Due to low prevalence using narrow definitions, we focused on broadly defined eating disorder groups.

Table 1 presents criteria used to define eating disorder groups. For the primary analyses, participants were classified into one of five groups based on lifetime history of eating disorders: (1) no eating disorder; (2) AN; (3) BN; (4) ANBN; and (5) binge eating

https://doi.org/10.1017/S0033291709005662 Published online by Cambridge University Press

Table 2. *Demographic characteristics across the four eating disorder groups and the no-eating disorder group*

	No ED ($n=12\,508$)	AN ($n=378$)	BN ($n=267$)	ANBN ($n=95$)	BED ($n=49$)
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/m²)	24.91 (4.45)	23.58 (4.24)	26.03 (5.02)	24.74 (4.91)	28.97 (7.63)
Lowest lifetime BMI (kg/m²)	19.98 (2.56)	16.45 (1.74)	19.87 (2.95)	16.16 (1.90)	20.87 (2.89)

Values are given as mean (standard deviation).

ED, Eating disorder; AN, anorexia nervosa; BN, bulimia nervosa; ANBN, lifetime history of AN and BN; BED, binge eating disorder; BMI, body mass index.

disorder (BED). For secondary analyses, the AN group was subdivided into RAN and ANBP.

Substance use

Eighteen substance-use 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 DSM-IV criteria for either abuse or dependence (APA, 2000). Binge drinking was defined as either four or more bottles of beer, four or more glasses of wine (>600 ml), or three or more shots of liquor (>180 ml) 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 smoking 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 defined as 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 substance-use 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 10 times per month; cannabis-ever). Due to low prevalence, diet pills-ever and hallucinogens 10 times per month were dropped from the analyses. Polysubstance 10 times per month was defined as having used at least two illicit substances at least 10 times per month. Polysubstance-ever was defined as ever having tried at least two illicit substances.

Statistical analyses

Prevalence was calculated for the eating disorder groups and the referent across all substance-use measures. Logistic regression analyses were conducted using generalized estimating equations (GEE) with PROC GENMOD (SAS version 9.1; SAS Institute Inc., Cary, NC, USA) to test for statistically significant differences in substance use across eating disorder groups. GEE allows for the correlated nature of the twin data to be controlled. Comparisons for each substance across pairs of eating disorders groups (e.g. AN, BN) were conducted to determine which eating disorder groups reported significantly more substance use and to report their associated effect sizes (i.e. odds ratios). In order to assess differences in substance use 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 substance-use 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 version 9.1 (SAS Institute Inc.).

Results

Of the sample, 94% reported no history of an eating disorder, 3% reported AN (RAN, $n=197$; ANBP, $n=181$), 2% BN, 1% ANBN and <1% BED (Table 2).

Demographics

Table 2 presents demographic information across the eating disorder groups and the referent group. Participants ranged in age from 20 to 47 years with a mean age of 34 years (S.D. = 7.66). The mean for highest lifetime adult body mass index (BMI) was greatest among the BED group and the mean for lowest lifetime adult BMI was lowest among the ANBN group.

https://doi.org/10.1017/S0033291709005662 Published online by Cambridge University Press

Prevalence of substance use across eating disorder groups

Table 3 presents prevalence estimates of substance use across the groups and provides odds ratios for statistically significant pairwise comparisons across eating disorder 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 (available online).

Overall, statistically significant differences for prevalence across groups were found for alcohol abuse/dependence, diet pills-weekly, cannabis 10 times per month, cannabis-ever, hallucinogens-ever, opioids 10 times per month, opioids-ever, sedatives 10 times per month, sedatives-ever, stimulants 10 times per month, stimulants-ever, polysubstance 10 times per month and polysubstance-ever. No statistically significant differences across groups were found for binge drinking, occasional smoking, regular smoking, 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 eating disorder 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 the prevalence of binge drinking.

Tobacco

No statistically significant group difference emerged for regular smoking, occasional smoking, 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 eating disorder 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 10 times per month and cannabis-ever. Individuals in the AN and BN groups were more likely to use cannabis 10 times per 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 hallucinogens-ever, 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 10 times per month and opioids-ever. Individuals in the AN group were more likely to use opioids 10 times per 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 10 times per month and sedatives-ever. The AN and BN groups were more likely to use sedatives 10 times per 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 10 times per month and stimulants-ever. The AN group was more likely to use stimulants 10 times per month relative to the referent. Those in the AN and BN groups were more likely to use stimulants-ever compared with the referent.

Polysubstance use. Statistically significant differences across groups were found for polysubstance 10 times per month and polysubstance-ever. The AN group was more likely to engage in polysubstance 10 times per 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 with the RAN group for alcohol abuse/dependence (15% *v.* 9%, $p<0.05$), diet pills-weekly (35% *v.* 9%, $p<0.01$), stimulants-ever (12% *v.* 5%, $p<0.01$) and polysubstance-ever (24% *v.* 15%, $p<0.01$). The ANBP group was 1.93 times more likely than the RAN group to have alcohol abuse/dependence [$\chi^2=4.08$, $p<0.043$, 95% confidence interval (CI) 1.02–3.66], 5.76 times more likely to use diet pills-weekly ($\chi^2=38.68$, $p<0.001$, 95% CI 3.22–10.31),

https://doi.org/10.1017/S0033291709005662 Published online by Cambridge University Press

Table 3. *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 drinking	693 (6%)	20 (5%)	20 (7%)	9 (9%)	4 (8%)		
Occasional smoking	2675 (28%)	76 (28%)	68 (33%)	20 (28%)	16 (44%)		
Regular smoking	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 10 times per 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 10 times per 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 10 times per 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 10 times per month[a]*	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 10 times per 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)*

ED, Eating disorder; AN, anorexia nervosa; BN, bulimia nervosa; ANBN, lifetime history of AN and BN; BED, binge eating disorder; CI, confidence interval.

Asterisks in the 'Substance-use item' column represent omnibus statistical significance across all groups.

[a] The BED group was removed from the analysis due to an empty cell.

* $p<0.05$, ** $p<0.01$.

https://doi.org/10.1017/S0033291709005662 Published online by Cambridge University Press

2.86 times more likely to endorse stimulants-ever ($\chi^2 = 6.87$, $p < 0.009$, 95% CI 1.28–6.38) and 1.74 times more likely to engage in polysubstance-ever ($\chi^2 = 4.40$, $p < 0.036$, 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 eating disorders in a population-based sample of Swedish women and presents novel epidemiological information on eating disorders and substance use. Three broad themes emerged from the analyses. First, consistent with previous research, the prevalence of substance use was higher in all eating disorder groups than the referent, indicating that substance use is not limited to any particular eating disorder presentation. Second, in contrast with previous studies (Anzengruber *et al*. 2006), tobacco use was not elevated in women with eating disorders relative to the referent. Third, the observation that women with AN report elevated substance-use behaviors challenges previously held beliefs that substance use is uncommon in women with AN (Wiederman & Pryor, 1996; Herzog *et al*. 2006) 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 substance use across varying presentations of AN.

Although no differences in prevalence emerged for binge drinking across the eating disorder 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*. 2004*a*). These findings could reflect the elevated relative risk of alcohol-use disorders among individuals with BN compared with AN (Kaye *et al*. 1996, 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*. 2004*a*) may also be particular risk factors for the binge/purge subtype of AN. One possible explanation is that substance use 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 effect of alcohol and other drugs is enhanced by the food deprivation associated with the illness (Carroll & Meisch, 1984; Bulik *et al*. 2004*a*). Additionally, alcohol (and drug) use may assist with the regulation of affect including prominent anxiety symptoms seen in those with AN (Godart *et al*. 2000; Bulik *et al*. 2004*a*). Overall, our finding of alcohol abuse/dependence in the AN group, particularly the ANBP group, extends a growing body of literature supporting substance-use behaviors among women with AN (von Ranson *et al*. 2002; Bulik *et al*. 2004*a*; Root *et al*. 2009).

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 eating disorder as a weight-control measure, thus resulting in no differences between groups.

Consistent with previous research (Roerig *et al*. 2003; Reba-Harrelson *et al*. 2008), diet pill use was elevated in all eating disorder 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; Reba *et al*. 2005; Fernandez-Aranda *et al*. 2006), 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 with 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 substance use and eating disorder 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 eating disorder groups reported greater use of illicit drugs and polysubstance use relative to the referent. Similar prevalences across eating disorder groups for cannabis-ever, hallucinogens-ever and stimulants-ever support previous research (Wiederman & Pryor, 1996; Root *et al*. 2009). Additionally, the BN and ANBN groups had higher risk for several illicit substances including

https://doi.org/10.1017/S0033291709005662 Published online by Cambridge University Press

cannabis, opioids, sedatives and stimulants, which supports previous research (The National Center on Addiction and Substance Abuse at Columbia University, 2003; Herzog *et al*. 2006; Hudson *et al*. 2007), 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 eating disorder 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 (Hudson *et al*. 1992; Wiederman & Pryor, 1996; Hsieh *et al*. 2005) and the attraction of cannabis and sedatives for their sedating effects (Swinbourne & Touyz, 2007).

One possible explanation for increased polysubstance use in those with eating disorders 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 eating disorders, particularly BN. Because individuals with polysubstance use have high rates of psychiatric co-morbidity (Lynskey *et al*. 2006) and often relapse after substance-abuse treatment (Marshall, 1994), replication and additional attention to polysubstance use with eating disorder populations is warranted.

Findings that illicit drug use is occurring across eating disorder 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 that these findings can be explained given genetic research associating family history (Lachman, 2006) to both substance use and eating disorders (Slof-Op 't Landt *et al*. 2005; Pinheiro *et al*. 2006; Bulik *et al*. 2006, 2007). Genetics influence liability to substance-use disorder, with research suggesting that substance use among monozygotic twin pairs is two to four times greater compared with dizygotic 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 eating disorders, family studies have demonstrated that both AN and BN tend to be increased in relatives of affected probands compared with relatives of unaffected probands (Lilenfeld *et al*. 1998; Pinheiro *et al*. 2006). Twin studies have reported heritability estimates between 33% and 84% for AN (Wade *et al*. 2000; Slof-Op 't Landt *et al*. 2005; Bulik *et al*. 2006, 2007) 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 eating disorders and substance use might not necessarily be influenced by shared genetic factors. 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 *et al*. (1998) concluded that eating disorders and alcoholism were not co-transmitted in families. Lastly, Kaye *et al*. (1996) reported that BN and substance-use disorders, including alcohol abuse and dependence, were transmitted independently. 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 co-morbid profile and familial transmission of eating disorders and substance use.

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 eating disorders or substance-use behaviors cannot be discussed. Third, additional unexamined factors may have influenced the findings, including factors that may be associated with eating disorders 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*. 2004*a*; Godart *et al*. 2006) are associated with the onset of both eating disorders and substance use. Fourth, one must consider whether any aspects of a twin population could limit generalizability of the observed prevalence of substance use and eating disorders 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 aged 16–84 years (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 poly-substance drug use in order to understand better differences in substance-use involvement across eating disorder groups. Last, the BED group is quite small in our sample (about 4%), which might be due in part to the much lower base rate of obesity in Sweden compared with the USA (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

The results of this study add to the growing literature on eating disorders and substance use by further emphasizing that eating disorders may be associated with a range of substance-use behavior. The STAGE sample used in the current study provided extensive eating disorders and substance-use phenotyping, including snus use, which has never been reported before. This study represents the first large population-based study that was able to contrast substance-use 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 substance use when examining and treating individuals with disordered eating. Although we cannot examine temporal patterns of onset, several possibilities exist, namely, the presence of an eating disorder may increase risk for substance use, substance use may increase risk for eating disorders, or a third underlying variable might increase risk for both eating disorders and substance use. Additional investigations incorporating patterns of onset will assist with determining how eating disorders and substance use mutually influence risk (Field *et al*. 2002; Stice & Shaw, 2002). Presenting comprehensive epidemiological 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 eating disorder and substance-use disorder (Keel *et al*. 2003; Franko *et al*. 2005).

Acknowledgements

T.L.R. was supported by National Institute of Health grant T32MH076694. This study was supported by grants CA-085739 (PI: P. F. Sullivan) and AI-056014 (Principal Investigator: P. F. Sullivan) from the National Institutes of Health. The STR is supported by grants from the Swedish Department of Higher Education and the Swedish Research Council.

Declaration of Interest

None.

Note

Supplementary material accompanies this paper on the Journal's website (http://journals.cambridge.org/psm).

References

Adams TB, Araas TE (2006). Purging and alcohol-related effects in college women. *International Journal of Eating Disorders* **39**, 240–244.

Anzengruber D, Klump KL, Thornton L, Brandt H, Crawford S, Fichter MM, Halmi KA, Johnson C, Kaplan AS, LaVia M, Mitchell J, Strober M, Woodside DB, Rotondo A, Berrettini WH, Kaye WH, Bulik CM (2006). Smoking in eating disorders. *Eating Behaviors* **7**, 291–299.

APA (2000). *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn, revised. American Psychiatric Press: Washington, DC.

Baker JH, Mazzeo SE, Kendler KS (2007). Association between broadly defined bulimia nervosa and drug use disorders: common genetic and environmental influences. *International Journal of Eating Disorders* **40**, 673–678.

Benjamini Y, Hochberg Y (1995). Controlling the false discovery rate: a practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society* **57**, 289–300.

Blinder BJ, Cumella EJ, Sanathara VA (2006). Psychiatric comorbidities of female inpatients with eating disorders. *Psychosomatic Medicine* **68**, 454–462.

Bulik CM, Klump KL, Thornton L, Kaplan AS, Devlin B, Fichter M, Halmi K, Strober M, Woodside DB, Crow S, Mitchell JE, Rotondo A, Mauri M, Cassano GB, Keel P, Berrettini WH, Kaye WH (2004*a*). Alcohol use disorder comorbidity in eating disorders: a multicenter study. *Journal of Clinical Psychiatry* **65**, 1000–1006.

Bulik CM, Slof S, Sullivan PF (2004*b*). Eating disorders and substance use disorders. In *Dual Diagnosis: Substance Abuse and Comorbid Medical and Psychiatric Conditions*, 2nd edn (ed. B. Rounsaville and H. Kranzler), pp. 317–348. Marcel Dekker: New York.

Bulik CM, Sullivan PF, Tozzi F, Furberg H, Lichtenstein P, Pedersen N (2006). Prevalence, heritability and prospective

risk factors for anorexia nervosa. *Archives of General Psychiatry* **63**, 305–312.

Bulik CM, Slof-Op 't Landt MC, van Furth EF, Sullivan PF (2007). The genetics of anorexia nervosa. *Annual Review of Nutrition* **27**, 263–275.

Bulik CM, Sullivan PF, Wade TD, Kendler KS (2000). Twin studies of eating disorders: a review. *International Journal of Eating Disorders* **27**, 1–20.

Carroll ME, Meisch RA (1984). Increased drug-reinforced behavior due to food deprivation. In *Advances in Behavioral Pharmacology* (ed. T. Thompson, P. B. Dews and J. E. Barrett), pp. 47–88. Academic Press: New York.

Cassin SE, von Ranson KM (2005). Personality and eating disorders: a decade in review. *Clinical Psychology Review* **25**, 895–916.

Conway KP, Kane RJ, Ball SA, Poling JC, Rounsaville BJ (2003). Personality, substance of choice, and polysubstance involvement among substance dependent patients. *Drug and Alcohol Dependence* **71**, 65–75.

Eaves L, Silberg J, Foley D, Bulik CM, Maes H, Erkanli A, Angold A, Costello EJ, Worthman C (2004). Genetic and environmental influences on the relative timing of pubertal change. *Twin Research* **7**, 471–481.

Eddy KT, Keel PK, Dorer DJ, Delinsky SS, Franko DL, Herzog DB (2002). Longitudinal comparison of anorexia nervosa subtypes. *International Journal of Eating Disorders* **31**, 191–201.

Fernandez-Aranda F, Jimenez-Murcia S, Alvarez-Moya EM, Granero R, Vallejo J, Bulik CM (2006). Impulse control disorders in eating disorders: clinical and therapeutic implications. *Comprehensive Psychiatry* **47**, 482–488.

Fernandez-Aranda F, Pinheiro AP, Tozzi F, Thornton LM, Fichter MM, Halmi KA, Kaplan AS, Klump KL, Strober M, Woodside DB, Crow S, Mitchell J, Rotondo A, Keel P, Plotnicov KH, Berrettini WH, Kaye WH, Crawford SF, Johnson C, Brandt H, La Via M, Bulik CM (2007). Symptom profile of major depressive disorder in women with eating disorders. *Australian and New Zealand Journal of Psychiatry* **41**, 24–31.

Field AE, Austin SB, Frazier AL, Gillman MW, Camargo Jr. CA, Colditz GA (2002). Smoking, getting drunk and engaging in bulimic behaviors: in which order are the behaviors adopted? *Journal of the American Academy of Child and Adolescent Psychiatry* **41**, 846–853.

Franko DL, Dorer DJ, Keel PK, Jackson S, Manzo MP, Herzog DB (2005). How do eating disorders and alcohol use disorder influence each other? *International Journal of Eating Disorders* **38**, 200–207.

Furberg H, Lichtenstein P, Pedersen NL, Bulik CM, Sullivan PF (2006). Cigarettes and oral snuff use in Sweden: prevalence and transitions. *Addiction* **101**, 1509–1515.

Godart NT, Flament MF, Lecrubier Y, Jeammet P (2000). Anxiety disorders in anorexia nervosa and bulimia nervosa: co-morbidity and chronology of appearance. *European Psychiatry* **15**, 38–45.

Godart NT, Perdereau F, Curt F, Rein Z, Lang F, Venisse JL, Halfon O, Bizouard P, Loas G, Corcos M, Jeammet P, Flament MF (2006). Is major depressive episode related to anxiety disorders in anorexics and bulimics? *Comprehensive Psychiatry* **47**, 91–98.

Herzog DB, Franko DL, Dorer DJ, Keel PK, Jackson S, Manzo MP (2006). Drug abuse in women with eating disorders. *International Journal of Eating Disorders* **39**, 364–368.

Herzog DB, Keller MB, Sacks NR, Yeh CJ, Lavori PW (1992). Psychiatric comorbidity in treatment-seeking anorexics and bulimics. *Journal of the American Academy of Child and Adolescent Psychiatry* **31**, 810–818.

Holderness CC, Brooks-Gunn J, Warren MP (1994). Co-morbidity of eating disorders and substance abuse review of the literature. *International Journal of Eating Disorders* **16**, 1–34.

Hsieh YS, Yang SF, Chiou HL, Kuo DY (2005). Transcriptional involvement of protein kinase C-alpha isozyme in amphetamine-mediated appetite suppression. *European Journal of Neurosciences* **22**, 715–723.

Hudson JI, Hiripi E, Pope Jr. HG, Kessler RC (2007). The prevalence and correlates of eating disorders in the national comorbidity survey replication. *Biological Psychiatry* **61**, 348–358.

Hudson JI, Weiss R, Pope Jr. HG, McElroy SL, Mirin SM (1992). Eating disorders in hospitalized substance abusers. *American Journal of Drug and Alcohol Abuse* **18**, 75–85.

Kaye WH, Lilenfeld LR, Plotnikov K, Merikangas KR, Nagy L, Strober M, Bulik CM, Moss H, Greeno CG (1996). Bulimia nervosa and substance dependence: association and family transmission. *Alcoholism: Clinical and Experimental Research* **20**, 878–881.

Kaye WH, Greeno CG, Moss H, Fernstrom J, Fernstrom M, Lilenfeld LR, Weltzin TE, Mann JJ (1998). Alterations in serotonin activity and psychiatric symptoms after recovery from bulimia nervosa. *Archives of General Psychiatry* **55**, 927–935.

Keel PK, Dorer DJ, Eddy KT, Franko DL, Charatan DL, Herzog DB (2003). Predictors of mortality in eating disorders. *Archives of General Psychiatry* **60**, 179–183.

Kendler KS, Prescott CA (1999). A population-based twin study of lifetime major depression in men and women. *Archives of General Psychiatry* **56**, 39–44.

Kendler KS, Walters EE, Neale MC, Kessler RC, Heath AC, Eaves LJ (1995). The structure of the genetic and environmental risk factors for six major psychiatric disorders in women: phobia, generalized anxiety disorder, panic disorder, bulimia, major depression and alcoholism. *Archives of General Psychiatry* **52**, 374–383.

Lachman HM (2006). An overview of the genetics of substance use disorders. *Current Psychiatry Reports* **8**, 133–143.

Lagergren J, Bergstrom R, Lindgren A, Nyren O (2000). The role of tobacco, snuff and alcohol use in the aetiology of cancer of the oesophagus and gastric cardia. *International Journal of Cancer* **85**, 340–346.

Lewin F, Norell SE, Johansson H, Gustavsson P, Wennerberg J, Biorklund A, Rutqvist LE (1998). Smoking tobacco, oral snuff, and alcohol in the etiology of squamous cell carcinoma of the head and neck: a population-based case-referent study in Sweden. *Cancer* **82**, 1367–1375.

Lichtenstein P, Sullivan PF, Cnattingius S, Gatz M, Johansson S, Carlstrom E, Bjork C, Svartengren M, Wolk A, Klareskog L, de Faire U, Schalling M, Palmgren J, Pedersen NL (2006). The Swedish Twin Registry in the third millennium: an update. *Twin Research and Human Genetics* **9**, 875–882.

Lilenfeld LR, Kaye WH, Greeno CG, Merikangas KR, Plotnikov K, Pollice C, Rao R, Strober M, Bulik CM, Nagy L (1998). A controlled family study of restricting anorexia and bulimia nervosa: comorbidity in probands and disorders in first-degree relatives. *Archives of General Psychiatry* **55**, 603–610.

Lynskey MT, Agrawal A, Bucholz KK, Nelson EC, Madden PA, Todorov AA, Grant JD, Martin NG, Heath AC (2006). Subtypes of illicit drug users: a latent class analysis of data from an Australian twin sample. *Twin Research and Human Genetics* **9**, 523–530.

Marshall DM (1994). Breed differences and genetic parameters for body composition traits in beef cattle. *Journal of Animal Sciences* **72**, 2745–2755.

Neovius M, Janson A, Rossner S (2006). Prevalence of obesity in Sweden. *Obesity Reviews* **7**, 1–3.

Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal K (2006). Prevalence of overweight and obesity in the United States, 1999–2004. *Journal of the American Medical Association* **295**, 1549–1555.

Osterdahl BG, Jansson C, Paccou A (2004). Decreased levels of tobacco-specific N-nitrosamines in moist snuff on the Swedish market. *Journal of Agricultural and Food Chemistry* **52**, 5085–5088.

Pinheiro AP, Sullivan PF, Bacaltchuck J, Prado-Lima PA, Bulik CM (2006). Genetics in eating disorders: extending the boundaries of research. *Brazilian Journal of Psychiatry* **28**, 218–225.

Prescott CA, Madden PA, Stallings MC (2006). Challenges in genetic studies of the etiology of substance use and substance use disorders: introduction to the special issue. *Behavior Genetics* **36**, 473–482.

Reba L, Thornton L, Tozzi F, Klump KL, Brandt H, Crawford S, Crow S, Fichter MM, Halmi KA, Johnson C, Kaplan AS, Keel P, LaVia M, Mitchell J, Strober M, Woodside DB, Rotondo A, Berrettini WH, Kaye WH, Bulik CM (2005). Relationships between features associated with vomiting in purging-type eating disorders. *International Journal of Eating Disorders* **38**, 287–294.

Reba-Harrelson L, Von Holle A, Thornton LM, Klump KL, Berrettini WH, Brandt H, Crawford S, Crow S, Fichter MM, Goldman D, Halmi KA, Johnson C, Kaplan AS, Keel P, LaVia M, Mitchell J, Plotnicov K, Rotondo A, Strober M, Treasure J, Woodside DB, Kaye WH, Bulik CM (2008). Features associated with diet pill use in individuals with eating disorders. *Eating Behaviors* **9**, 73–81.

Roerig JL, Mitchell JE, de Zwaan M, Wonderlich SA, Kamran S, Engbloom S, Burgard M, Lancaster K (2003). The eating disorders medicine cabinet revisited: a clinician's guide to appetite suppressants and diuretics. *International Journal of Eating Disorders* **33**, 443–457.

Root TL, Pinheiro AP, Thornton L, Strober M, Fernandez-Aranda F, Brandt H, Crawford S, Fichter MM, Halmi KA, Johnson C, Kaplan AS, Klump KL, La Via M, Mitchell J, Woodside DB, Rotondo A, Berrettini WH, Kaye WH, Bulik CM (2009). Substance use disorders in women with anorexia nervosa. *International Journal of Eating Disorders*. Published online: 3 March 2009. doi: 10.1002/eat.20670.

Ross HE, Ivis F (1999). Binge eating and substance use among male and female adolescents. *International Journal of Eating Disorders* **26**, 245–260.

Slof-Op 't Landt MC, van Furth EF, Meulenbelt I, Slagboom PE, Bartels M, Boomsma D, Bulik C (2005). Eating disorders: from twin studies to candidate genes and beyond. *Twin Research and Human Genetics* **16**, 467–482.

Steiger H, Bruce KR (2007). Phenotypes, endophenotypes, and genotypes in bulimia spectrum eating disorders. *Canadian Journal of Psychiatry* **52**, 220–227.

Stice E, Shaw HE (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology: a synthesis of research findings. *Journal of Psychosomatic Research* **53**, 985–993.

Strong K, Bonita R (2003). *The SuRF Report 1. Surveillance of Risk Factors Related to Noncommunicable Diseases: Current Status of Global Data*. WHO: Geneva.

Swinbourne JM, Touyz SW (2007). The co-morbidity of eating disorders and anxiety disorders: a review. *European Eating Disorders Review* **15**, 253–274.

The National Center on Addiction and Substance Abuse at Columbia University (2003). *Food for Thought: Substance Abuse and Eating Disorders*. The National Center on Addiction and Substance Abuse (CASA) at Columbia University: New York.

von Ranson KM, Iacono WG, McGue M (2002). Disordered eating and substance use in an epidemiological sample: I. Associations within individuals. *International Journal of Eating Disorders* **31**, 389–403.

Wade TD, Bulik CM, Neale M, Kendler KS (2000). Anorexia nervosa and major depression: shared genetic and environmental risk factors. *American Journal of Psychiatry* **157**, 469–471.

Wersall JP, Eklund G (1998). The decline of smoking among Swedish men. *International Journal of Epidemiology* **27**, 20–26.

Wiederman MW, Pryor T (1996). Substance use among women with eating disorders. *International Journal of Eating Disorders* **20**, 163–168.