



Associations between ADHD and eating disorders in relation to comorbid psychiatric disorders in a nationally representative sample

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ABSTRACT

The objective of this study was to examine whether previously observed associations of attention-deficit/hyperactivity disorder (ADHD) with eating disorders (EDs) are at least partially attributable to other underlying psychopathology. Data came from 4719 participants aged 18–44 years in the National Comorbidity Survey Replication and the National Survey of American Life. DSM-IV diagnoses were assessed using the World Health Organization Composite International Diagnostic Interview. Multinomial logistic regression assessed associations between DSM-IV lifetime and past-12 month diagnoses of ADHD with EDs in unadjusted models and in models adjusted for demographic variables and other psychopathology. Lifetime ADHD was strongly and significantly associated with lifetime bulimia nervosa (BN), binge eating disorder (BED), and any ED in unadjusted models, but not with anorexia nervosa or subthreshold BED. After adjusting for demographic variables and psychiatric comorbidities, all associations of lifetime ADHD with EDs were substantially attenuated, and only the association of ADHD with BN remained statistically significant. Similar results were found using past-12 month diagnoses. These results suggest that previously observed associations of ADHD with EDs might be due – at least in part – to additional psychiatric disorders that are often comorbid with both ADHD and EDs.

1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common psychiatric disorders among children and adolescents, with prevalence estimates ranging from 5.9% to 7.1% (Willcutt, 2012). As they grow older, children with ADHD are at an increased risk of developing additional psychiatric disorders (Biederman et al., 2004) including eating disorders (EDs) (Biederman et al., 2010; Yoshimasu et al., 2012). For example, Biederman et al. (2010) found that girls being treated for ADHD in childhood and adolescence were 3.5 times (95% CI: 1.6–7.3) more likely to develop an ED by young adulthood compared to girls without ADHD (Biederman et al., 2010). Similarly, using data from a population-based birth cohort, Yoshimasu et al. found that children with ADHD were 5.7 times (95% CI: 1.1–28.2) more likely than their counterparts without ADHD to have an ED diagnosis by late adolescence (Yoshimasu et al., 2012). Additionally, childhood ADHD symptomology has been found to be associated with current and later development of disordered eating behaviors, including bulimic symptoms (Mikami et al., 2008), binge eating (Bleck et al., 2015; Sonnevill et al., 2015) and restrictive eating (Bleck and DeBate, 2013; Råstam

et al., 2013). Numerous studies have found full diagnostic ADHD to be most strongly associated with ED diagnoses that feature binge eating, such as bulimia nervosa (BN) and binge eating disorder (BED) (Biederman et al., 2007, 2010; Bleck et al., 2015; Hudson et al., 2007). However, a recent meta-analysis found that, among people with ADHD, there was a similar level of risk for AN (OR: 4.28; 95%CI: 2.24–8.16) and BED (OR: 4.13; 95%CI: 3.00–5.67), and a trend toward BN having the highest risk (OR: 5.71; 95%CI: 3.56–9.16) of the three ED diagnoses (Nazar et al., 2016), with a pooled OR of 3.81 (95%CI: 2.31–6.21) for the association between ADHD and any ED or ED symptoms. The recent approval of lisdexamfetamine, a stimulant prodrug typically used to treat ADHD, for treatment of moderate to severe BED has sparked an interest in better understanding the reasons for the association of ADHD with BED and other EDs (Nazar et al., 2016).

Eating disorders, especially BN and BED, are highly comorbid with mood and anxiety disorders (Hudson et al., 2007; Kessler et al., 2013; Milos et al., 2003) as well as substance use disorders (SUDs) (Duncan et al., 2005; Holderness et al., 1994; Hudson et al., 2007) and post-traumatic stress disorder (PTSD) (Mitchell et al., 2012). Similarly, individuals with ADHD have been shown to be more likely to develop

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mood disorders, anxiety disorders, SUDs, and PTSD than those without ADHD (Adler et al., 2004; Biederman et al., 2004, 2014; Lee et al., 2011; Wilens et al., 2011; Yoshimasu et al., 2012). Previous research has found that girls with diagnoses of both ADHD and EDs had significantly higher prevalence of comorbid major depression, anxiety disorder, and disruptive behavior disorders compared with girls with ADHD who did not have an ED (Biederman et al., 2007). Similarly, Nazar et al. (2014) found that, among obese women seeking treatment for obesity or eating disorders, those with both ADHD and an ED were significantly more likely to have anxiety and substance use disorders compared with those with only EDs (Nazar et al., 2014). It is thus possible that previously observed associations between ADHD and EDs are partially or entirely due to associations of ADHD and EDs with other psychopathology; however, to our knowledge, only one study has taken other psychiatric comorbidities into account in their analyses when assessing the relationship between ADHD and EDs (Reinblatt et al., 2014). This study found that the association between ADHD and broadly defined BED remained significant after adjusting for comorbid disorders in a sample of children and adolescents receiving treatment from community mental health centers (Reinblatt et al., 2014). Given that the median ages of onset and the age of peak incidence for EDs varies according to ED type (Hudson et al., 2007), it is also important to study the role of psychiatric comorbidity in the association between ADHD and EDs among adults who are past the age of greatest risk for onset of these disorders.

An additional limitation of previous research is that the association between ADHD and EDs has primarily been assessed in clinical samples. For example, of the 24 studies that met criteria for inclusion in Nazar et al.'s 2016 meta-analysis, only 9 were from community-based samples, and a comparison of results between clinical and non-clinical samples was not assessed (Nazar et al., 2016). Considering that more than half of individuals with a history of EDs (Hudson et al., 2007) and a quarter of adolescents with ADHD (Costello et al., 2014) have never received treatment for these disorders, findings from clinical samples may not be applicable to the general population. Further limiting generalizability, the majority of these clinical studies did not include men and had few non-white participants. To our knowledge, five studies have used data from non-clinical samples to assess the association between ADHD and EDs at the diagnostic – rather than the symptom – level, none of which took comorbid psychopathology into account (Bleck et al., 2015; Brewerton and Duncan, 2016; Hudson et al., 2007; Kessler et al., 2013; Yoshimasu et al., 2012). Of these studies, two found statistically significant associations between ADHD and EDs, in general, but did not examine associations with specific ED diagnoses, and used self-reported (Bleck et al., 2015) or medical record extracted (Yoshimasu et al., 2012) diagnosis of an ED by a health care provider as the outcome, thus likely missing cases that did not seek ED treatment. The other three studies used ADHD and specific ED diagnoses derived from a structured psychiatric interview. Hudson et al. (2007) found that lifetime ADHD was significantly associated with lifetime BN and BED, but not anorexia nervosa (AN), in the National Comorbidity Survey Replication (NCS-R) (Hudson et al., 2007). Similarly, Brewerton and Duncan (2016) used the NCS-R to study the association between lifetime and past 12-month EDs and ADHD separately in men and women and found that lifetime BN and BED were associated with lifetime ADHD in both genders. In addition, they found associations of past 12-month ADHD with past 12-month BED among men and women and with past 12-month BN and lifetime AN among women only (Brewerton and Duncan, 2016). ADHD was also significantly associated with BED and BN in a study using data from the World Health Organization World Mental Health Surveys, which included the NCS-R (Kessler et al., 2013).

To address these gaps in the literature, the objective of the present study was to examine the association of ADHD with EDs before and after adjusting for other psychiatric comorbidities using nationally representative data from adults participating in the National Comorbidity Survey Replication and the National Survey of American Life.

2. Methods

2.1. Participants

Data for the current study came from two of the three surveys that comprise the Collaborative Psychiatric Epidemiology Surveys (CPES): the National Comorbidity Survey Replication (NCS-R) (Kessler et al., 2004) and the National Survey of American Life (NSAL) (Jackson et al., 2004). The surveys in the CPES used similar versions of a structured psychiatric interview and were designed to be able to combine data from each survey. The third survey, the National Latino and Asian American Study, did not include questions about ADHD and was thus not included in analyses. Briefly, the NCS-R is a nationally representative, cross-sectional survey of 9282 US adults conducted between February 2001 and April 2003 (response rate, 70.9%). Although all participants completed the core diagnostic interview, questions regarding ADHD were included in the Part II interview, which was administered to (1) those who met criteria for a disorder assessed in the core diagnostic interview (Part I), and (2) a probability subsample of respondents who did not meet criteria for any Part I disorder. Due to concerns over the validity of recall of childhood behaviors by older adults, only those participants who were under 45 years old at the time of interview were asked about ADHD symptoms ($n = 3199$). The ED diagnostic section was only administered to a random sub-sample of the Part II participants ($n = 2980$). A total of 1687 NCS-R participants who were under age 45 and had diagnostic information on both ADHD and EDs were included in the analyses.

The NSAL, conducted between February 2001 and March 2003, used a sampling scheme similar to that of the NCS-R to identify a nationally representative sample of US adults self-identifying as African American or of Caribbean descent, as well as non-Hispanic whites living in the same communities (response rate 71.5%; $n = 6197$). Assessments of ADHD and EDs were included in Part II of the interview, which was administered only to African American and Caribbean Black participants ($n = 5191$). For comparability with the NCS-R sample, only those NSAL participants who had diagnostic information on both ADHD and EDs and who were under age 45 at the time of interview were included in the current analyses ($n = 3032$).

2.2. Assessment

Both the NCS-R and NSAL assessed current and lifetime psychopathology with similar versions of the lay-administered World Health Organization Composite International Diagnostic Interview (CIDI), which generates psychiatric diagnoses according to both DSM-IV and ICD-10 criteria (Kessler and Üstün, 2004), primarily via face-to-face, computer assisted personal interviews (about 14% of NSAL participants were interviewed entirely or partially by telephone). DSM-IV criteria were used in the present study. The ED module of the interview assessed lifetime and past 12-month AN, BN, and BED. Additional composite variables were constructed for past 12-month and lifetime diagnosis of any ED. Further, variables for past 12-month and lifetime endorsement of “subthreshold BED”, defined as (a) binge eating episodes, (b) occurring at least twice a week for at least 3 months, and (c) not occurring solely during the course of AN, BN, or BED (Hudson et al., 2007) were constructed and used in analyses.

As described in more detail elsewhere (Kessler et al., 2006), the interview schedule in the NCS-R and NSAL assessed past 12-month and lifetime ADHD with the retrospective childhood ADHD assessment based on the Diagnostic Interview Schedule for DSM-IV (Robins et al., 2000). Respondents who met criteria for childhood ADHD were then asked a single question about whether they currently experienced problems with attention or hyperactivity-impulsivity. Those who endorsed this question received clinical reappraisal interviews for adult ADHD (Kessler et al., 2010), which were administered by PhD level clinical psychologists and used the Adult ADHD Clinical Diagnostic Scale (Adler and Spencer, 2004).

Additional separate variables were constructed for DSM-IV diagnoses of any mood disorder, any anxiety disorder, and any SUD, with separate variables constructed for lifetime and past 12-month diagnoses. Respondents were coded positive for any mood disorder if they met criteria for DSM-IV major depressive disorder, dysthymia, or bipolar I or II disorders. Any anxiety disorder was coded positive if a respondent met diagnostic criteria for DSM-IV panic disorder, agoraphobia without panic disorder, social phobia, generalized anxiety disorder, or separation anxiety disorder (lifetime only, as there was not information available for past 12-month separation anxiety disorder). Specific phobia was not included in the lifetime or past 12-month any anxiety disorder variable because this diagnosis was not assessed in the NSAL. Although PTSD was included in the anxiety disorder chapter in DSM-IV, it was modeled as a separate variable in the current analyses because it is no longer classified as an anxiety disorder in DSM-5 (American Psychiatric Association, 2013). Lifetime and past 12-month diagnoses of any SUD included any diagnosis of alcohol or drug abuse or dependence

2.3. Data analysis

Data analysis was conducted in Stata version 9.2 using the survey estimation (svy) command set and weights for the combined NCS-R Part II and NSAL dataset to adjust for the complex survey design of the NCS-R and NSAL. First, we used logistic regression to examine bivariate associations between ADHD (independent variable) and ED diagnostic variables (dependent variables), with analyses run separately for lifetime and past 12-month diagnoses. These unadjusted models were designated as Model 1. In Model 2, we added demographic variables (race, gender, and age) to each of the bivariate models. In Model 3, we added variables for additional comorbid psychopathology (separate variables for any mood disorder, any anxiety disorder, any SUD, and PTSD) to Model 2. In addition, since we combined data from two different studies, we tested for interactions between study (NSAL vs. NCS-R) and ADHD. Due to gender differences in the prevalence of EDs (more common among females; e.g. (Hudson et al., 2007; Swanson et al., 2011)) and ADHD (more common among males; e.g. (Willcutt, 2012)), although a recent narrative review reported that there are no significant clinical distinctions in ADHD between men and women in adults (Williamson and Johnston, 2015)). We also tested for interactions between gender and ADHD by adding interaction terms separately to Model 3 for each outcome

Since only one participant met criteria for past 12-month AN, the association between ADHD and past 12-month AN were not assessed.

3. Results

Demographic and psychiatric characteristics of the participants included in the present analysis are shown overall and by gender in Table 1. The mean age of study participants was 31 years. Fifty-two percent of participants were female, and the majority of the sample was non-Hispanic white. Four percent of participants met criteria for lifetime diagnosis of any ED, and 1.5% met criteria for any ED in past 12-months. Lifetime and past 12-month ADHD criteria were met by 7.7% and 3.7% of the study sample, respectively. With the exception of subthreshold binge eating disorder, the prevalence of all lifetime and past-12 month ED diagnoses was greater among women than among men. Although the prevalence of lifetime ADHD was greater among men (8.8% vs. 6.6%), men and women had a similar prevalence of past 12-month ADHD (3.8% and 3.6%, respectively).

The lifetime and past 12-month prevalence of comorbid disorders in the overall sample and among participants with each ED diagnosis and with ADHD is presented in Table 2 (lifetime) and Table 3 (past 12-month). The prevalence of each comorbid disorder was substantially higher in each ED category and among participants with ADHD compared to that in the overall sample. The prevalence of comorbid

Table 1

Study sample demographic and psychiatric characteristics (n = 4719). All numbers are weighted percentages.

	Total Sample	Females (52.1%)	Males (47.9%)
Age in years			
18–24	28.7	28.3	29.2
25–34	31.8	33.4	30.1
35–44	39.4	38.3	40.7
Race			
Non-Hispanic white	55.5	55.2	55.7
Non-Hispanic black	20.7	22.1	19.2
Hispanic	16.5	14.0	19.2
Other	7.4	8.8	5.9
DSM-IV eating disorder diagnoses			
Anorexia nervosa			
Lifetime	0.6	0.9	0.4
Past 12-month	< 0.1	0.0	< 0.1
Bulimia nervosa			
Lifetime	1.2	2.1	0.21
Past 12-month	0.4	0.8	< 0.1
Binge eating disorder			
Lifetime	2.5	3.4	1.5
Past 12-month	1.0	1.5	0.5
Any eating disorder			
Lifetime	4.0	5.9	2.1
Past 12-month	1.5	2.3	0.6
Subthreshold binge eating disorder			
Lifetime	1.5	0.9	2.1
Past 12-month	0.6	0.3	0.8
DSM-IV Attention-deficit/hyperactivity disorder			
Lifetime	7.7	6.6	8.8
Past 12-month	3.7	3.6	3.8

conditions by each ED category and ADHD by gender are displayed in Supplementary Tables 1 and 2.

In unadjusted models (Table 4, Model 1), lifetime ADHD was significantly and strongly associated with lifetime BN (OR: 5.78, 95% CI: 2.59–12.90), BED (OR: 2.60, 95% CI: 1.33–5.00), and any ED (OR: 3.36, 95% CI: 1.97–5.72). Although the ORs for associations with AN (OR: 2.69, 95% CI: 0.74–9.80) and subthreshold BED (OR: 1.98, 95% CI: 0.91–4.28) were of moderate magnitude, neither reached statistical significance. After adjusting for demographic variables, associations of ADHD with BN, BED, any ED, and subthreshold BED were similar to unadjusted ORs and were all statistically significant (Table 4, Model 2). Of note, although the unadjusted ORs, as well as the ORs adjusted for demographics for the association of ADHD with AN were comparable in magnitude to those for the association of ADHD with BED, those for AN were not statistically different from 1.00 (no association) as evidenced by their wide confidence intervals that included 1.00. After further adjusting for comorbid psychiatric disorders, the ORs for all ED outcomes were substantially attenuated, and only that for the association between ADHD and BN remained statistically significant (OR: 2.52, 95% CI: 1.04–6.10) (Table 4, Model 3).

ADHD in the past 12-months was strongly associated with all past 12-month ED variables in both unadjusted models and models adjusted for demographics (Table 5, Models 1 and 2, respectively). As with lifetime ED diagnoses, the strength of the association diminished considerably after further adjusting for other past 12-month psychopathology; however, the association of past 12-month ADHD with past 12-month BN (OR: 5.04, 95% CI: 1.15–22.08) and any ED (OR: 2.84, 95% CI: 1.22–6.63) remained robust and statistically significant (Table 5, Model 3). The adjusted OR for the association of ADHD with BED was weaker in magnitude compared to associations with other ED variables, and it was not statistically significant after adjusting for other 12-month psychopathology. Past 12-month subthreshold BED was not significantly associated with ADHD after further adjusting for other psychiatric diagnoses.

Table 2

Prevalence* of lifetime DSM-IV psychiatric disorders in the overall sample and among participants with lifetime DSM-IV eating disorders and attention-deficit/hyperactivity disorder (ADHD).

	Total Sample	Anorexia nervosa	Bulimia nervosa	Binge eating disorder	Any eating disorder	Subthreshold binge eating disorder	ADHD
Any substance use disorder	15.8	33.4	29.8	24.5	27.2	26.9	34.7
Any mood disorder	20.1	50.4	60.2	38.8	45.1	30.5	45.1
Any anxiety disorder	22.4	59.2	67.1	67.1	55.4	43.3	52.5
Posttraumatic stress disorder	7.2	18.1	45.1	20.3	23.6	5.8	23.5

* Weighted percentage.

There were no significant interactions of lifetime or past 12-month ADHD with gender or study (NCS-R or NSAL), indicating that the association between ADHD and the ED outcomes did not differ significantly by study or gender, which was also evidenced by the overlapping confidence intervals for females and males in predicting ED risk in gender-specific models (Supplementary Tables 3 and 4; note that there were not enough males or females with AN, or males with past 12-month BN, to perform regression models in these subgroups),

4. Discussion

To our knowledge, this is the first study to account for comorbid psychopathology when examining the association of ADHD with EDs in a general population sample of US adults. We found that although ADHD was significantly and robustly associated with BN, BED and any ED in lifetime and past 12-month models before adjusting for comorbid psychopathology, after adjusting for comorbid disorders the magnitude of these associations was substantially attenuated and in some cases no longer statistically significant. The associations that did remain statistically significant after adjusting for comorbid disorders, although reduced from unadjusted models, were still strong in magnitude. These findings suggest that underlying psychopathology may partially account for the association of ADHD with EDs. This highlights the importance of controlling for comorbid psychopathology when assessing the relationship between ADHD and EDs, as the strength of the association between these disorders may be weaker than previously thought.

As with previous studies (Biederman et al., 2004, 2010; Hudson et al., 2007; Kessler et al., 2013; Nazar et al., 2016), we found that ADHD was most strongly associated with BN, and that the association of lifetime ADHD with AN was not statistically significant, despite being of similar magnitude to that of lifetime ADHD with BED. Using data from the NCS-R, Brewerton and Duncan (2016) reported that the onset of ADHD preceded the onset of EDs in all men and women who met criteria for both diagnoses (Brewerton and Duncan, 2016). Together these findings further substantiate the premise that ADHD may be an important risk factor for the development of EDs, especially BN, given that this relationship holds true even after adjusting for other psychiatric comorbidities.

Much of the recent attention on the association of ADHD with EDs has focused on BED, with the recent approval of the stimulant prodrug, lisdexamfetamine, as mentioned earlier. Here we found that ADHD was not significantly associated with BED after adjusting for comorbid

psychopathology in either lifetime or past 12-month models, suggesting that underlying psychopathology in general may explain much of the previously observed associations. Especially because of the high comorbidity of BED, SUDS and other psychiatric disorders, health providers should therefore be cautious of treating people with BED with medications that may aggravate anxiety and/or have a high propensity for misuse or addiction.

There are several proposed biological mechanisms for the association between ADHD and EDs in general and in particular for BN. It has been suggested that attentional deficits characteristic of ADHD may influence self-image and body perceptions, leading to risk for EDs (Ortega-Roldán et al., 2014). Genetic factors are known to play important roles in both ADHD and BN (Biederman et al., 1992; Bulik and Tozzi, 2004; Sherman et al., 1997), and in particular, the dopaminergic system is known to be involved in both sets of disorders (Broft et al., 2012; LaHoste et al., 1996; Swanson et al., 2000). Dysfunction in the dopaminergic system thus might have an effect on the development of both ADHD and BN and may also account for the shared common clinical feature of impulsivity in both disorders (American Psychiatric Association, 2013), which may in turn connect the two disorders. Overlapping abnormalities in brain circuitry, particularly in reward, response inhibition, and emotional processing pathways, have been found for ADHD and binge eating behaviors (Seymour et al., 2015). In a recent review Nazar et al. (2016) observed that most current explanations for the association of ADHD with EDs rely on a single factor to explain the connection, failing to take into consideration the complexity of each disorder. We have attempted to address these complexities by taking other psychopathology into account in our models, finding that underlying psychopathology, in general, may partly explain the association between ADHD and EDs.

Similar to the findings of Nazar et al. (2016), we did not observe differences in risks for EDs between females and males, as evidenced by all non-significant interaction terms of gender x ADHD in Model 3 predicting each ED outcome, as well as by the overlapping confidence intervals in females and males, presented in supplemental analyses (Supplementary Tables 3 and 4).

Our findings should be considered in light of several limitations. First, retrospective assessment of lifetime psychiatric disorders likely underestimates their prevalence (Moffitt et al., 2010), as suggested by the results of a clinical reappraisal study of the CIDI (Haro et al., 2006). This may be particularly true for self-report of DSM-IV childhood ADHD, for which symptoms must have first occurred before the age of seven. Although questions about ADHD were only asked of participants

Table 3

Prevalence* of past 12-month DSM-IV psychiatric disorders in the overall sample and among participants with 12-month DSM-IV eating disorders and attention-deficit/hyperactivity disorder (ADHD).

	Total Sample	Anorexia nervosa	Bulimia nervosa	Binge eating disorder	Any eating disorder	Subthreshold binge eating disorder	ADHD
Any substance use disorder	5.7	–	21.3	21.4	22.2	18.3	16.7
Any mood disorder	10.3	–	62.7	30.7	39.7	9.0	41.2
Any anxiety disorder	11.6	–	59.5	34.5	42.5	30.2	55.1
Posttraumatic stress disorder	3.8	–	50.1	14.4	25.9	2.4	21.9

* Weighted percentage.

Table 4

Odds ratios and 95% confidence intervals for lifetime DSM-IV attention-deficit/hyperactivity disorder in unadjusted and adjusted logistic regression models predicting lifetime DSM-IV eating disorders and subthreshold binge eating disorder.

	Anorexia nervosa, OR (95% CI)	Bulimia nervosa, OR (95% CI)	Binge eating disorder, OR (95% CI)	Any eating disorder, OR (95% CI)	Subthreshold binge eating disorder, OR (95% CI)
Model 1 ^a	2.69 (0.74–9.80)	5.78 (2.59–12.90)	2.60 (1.33–5.09)	3.36 (1.97–5.72)	1.98 (0.91–4.28)
Model 2 ^b	2.54 (0.66–9.81)	7.30 (3.40–15.67)	2.55 (1.25–5.24)	3.50 (1.93–6.33)	2.09 (1.03–4.24)
Model 3 ^c	1.19 (0.31–4.63)	2.52 (1.04–6.10)	1.42 (0.74–2.74)	1.73 (0.99–3.01)	1.43 (0.65–3.15)

^a Unadjusted models.

^b Models adjusted for age, race, and sex.

^c Models adjusted for age, race, sex, and lifetime diagnosis of any mood disorder, any anxiety disorder, any substance use disorder, and posttraumatic stress disorder.

under the age of 45 in an attempt to minimize this risk, it is likely that some misclassification still occurred. The prevalence of any SUD may also be underestimated due to a skip in the NCS-R and NSAL interviews such that substance dependence was only assessed among individuals who endorsed at least one criterion for substance abuse.

Second, despite the large sample size, in some cases statistical power was limited, as evidenced by the broad confidence intervals for the odds ratios, due in large part to the low prevalence of individual ED diagnoses. This was particularly the case for associations of ADHD with AN, which had the lowest prevalence of all ED variables, and for which none of the associations with lifetime ADHD were statistically significant, despite being of similar magnitude to statistically significant associations of lifetime ADHD with BED in unadjusted models. Of note, it is possible that the association between ADHD and AN may differ by AN subtype. Unfortunately, we were unable to distinguish the restrictive or binge/purge subtypes of AN in our analyses due to limitations of the data collection instrument, and further subdividing the small number of participants who met criteria for AN would not likely have been fruitful given that statistical power was already low.

Third, although not strictly a limitation, it should be noted that this study used DSM-IV definitions of ADHD and EDs. As the DSM-5 ADHD criteria require that several ADHD symptoms be present before age 12, compared to age 7 in DSM-IV, more people in the study would be expected to qualify for an ADHD diagnosis under DSM-5; for example, one community-based study estimated that the prevalence of ADHD would increase from 8.9% to 11.3% using DSM-IV vs DSM-V criteria (McKeown et al., 2015). It was not possible to determine whether results would have differed if the DSM-5 definition were used because the questions that assessed ADHD symptoms specified the presence of the behaviors before age 7. Similarly, the DSM-5 criteria for BN and BED are less stringent than DSM-IV criteria, requiring that binge eating episodes occur a minimum of once a week (American Psychiatric Association, 2013), as opposed to twice a week in the DSM-IV-TR (American Psychiatric Association, 2000). Therefore, it is likely that some individuals in the sample who were coded negative for EDs may have met criteria for BN or BED under DSM-5. Since the questions in the NCS-R and NSAL regarding binge eating specified that bingeing occurred twice per week, we were unable to construct DSM-5 algorithms for ED diagnoses. Using DSM-IV criteria, however, allows us to better compare our findings with other studies that assessed the association of ADHD

with EDs using DSM-IV criteria. More so, findings are mixed regarding the effect this change in the DSM has on the prevalence of BED; while one study found that the prevalence of BED doubled when using DSM-5 compared with DSM-IV criteria in a general population sample of adults in Australia (Hay et al., 2015), another non-clinical study estimated that the prevalence of BED would increase less than two tenths of a percentage point in the NCS-R using DSM-5 criteria (Hudson et al., 2012).

Finally, this study used cross-sectional data and did not account for the chronology of the development of ADHD and EDs. Although some have posited that onset of EDs could precede ADHD, and that the effects of EDs on attention and impulsive behaviors could then lead to ADHD or ADHD-like symptoms (Duchesne et al., 2004), given that the DSM-IV ADHD criteria required onset of symptoms before age 7 and the average age of onset of EDs is in late adolescence or early adulthood, and that several studies have found that ADHD precedes the development of EDs (Biederman et al., 2007, 2010; Brewerton and Duncan, 2016; Mikami et al., 2008; Yoshimasu et al., 2012), ED onset prior to onset of ADHD is likely to be a rare occurrence.

Despite these limitations, our findings suggest that previously observed associations of ADHD with EDs may be due – at least in part – to additional psychiatric disorders that are commonly comorbid with both ADHD and EDs. In other words, ADHD may not be specifically associated with EDs in particular but with other psychiatric disorders in general. This message resonates with other studies that have emphasized the importance of adjusting for additional psychiatric disorders when assessing the comorbidity between two disorders. For example, previous studies have found that many of the significant unadjusted associations of alcohol and drug use disorder with other psychiatric disorders become nonsignificant and/or substantially attenuated after adjusting for demographic characteristics and additional comorbid psychiatric disorders (Compton et al., 2007; Hasin et al., 2007). These studies have suggested that factors common to additional psychiatric comorbidities accounted for some (and in some cases much) of the association of alcohol and drug use disorders with other diagnoses. Given that the large majority of people with EDs and ADHD have additional psychiatric diagnoses (Hudson et al., 2007; Kessler et al., 2006), and that previous research has found that patients with both ADHD and an ED are significantly more likely to have additional psychiatric diagnoses compared with patients with only ADHD or an ED

Table 5

Odds ratios and 95% confidence intervals for past 12-month DSM-IV attention-deficit/hyperactivity disorder in unadjusted and adjusted logistic regression models predicting past 12-month DSM-IV eating disorders and subthreshold binge eating disorder.

	Anorexia nervosa, OR (95% CI)	Bulimia nervosa, OR (95% CI)	Binge eating disorder, OR (95% CI)	Any eating disorder, OR (95% CI)	Subthreshold binge eating disorder, OR (95% CI)
Model 1 ^a	–	21.41 (5.47–83.71)	4.46 (1.81–10.95)	8.92 (3.87–20.56)	5.70 (1.88–17.26)
Model 2 ^b	–	28.24 (6.33–126.01)	4.53 (1.82–11.24)	9.74 (4.23–22.40)	5.55 (1.90–16.24)
Model 3 ^c	–	5.04 (1.15–22.08)	1.65 (0.67–4.04)	2.84 (1.22–6.63)	3.83 (0.94–15.67)

^a Unadjusted models.

^b Models adjusted for age, race, and sex.

^c Models adjusted for age, race, sex, and lifetime diagnosis of any mood disorder, any anxiety disorder, any substance use disorder, and posttraumatic stress disorder.

(Biederman et al., 2007; Nazar et al., 2014), it seems pivotal to take other mental disorders into account when assessing the relationship between EDs and ADHD in order to best understand the true association between these two disorders.

Conflicts of interest

None.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.psychres.2017.11.026>.

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