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ORIGINAL ARTICLE

Genetic and environmental aspects in the association between attention-deficit hyperactivity disorder symptoms and binge-eating behavior in adults: a twin study

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Background. Prior research demonstrated that attention-deficit hyperactivity disorder (ADHD) is associated with binge-eating behavior, binge-eating disorder (BED), and bulimia nervosa (BN). The aim of this study was to investigate these associations in an adult twin population, and to determine the extent to which ADHD symptoms and binge-eating behavior share genetic and environmental factors.

Methods. We used self-reports of current ADHD symptoms and lifetime binge-eating behavior and associated characteristics from a sample of over 18 000 adult twins aged 20–46 years, from the population-based Swedish Twin Registry. Mixed-effects logistic regression was used to examine the association between ADHD and lifetime binge-eating behavior, BED, and BN. Structural equation modeling was used in 13 773 female twins to determine the relative contribution of genetic and environmental factors to the association between ADHD symptoms and binge-eating behavior in female adult twins.

Results. ADHD symptoms were significantly associated with lifetime binge-eating behavior, BED, and BN. The heritability estimate for current ADHD symptoms was 0.42 [95% confidence interval (CI) 0.41–0.44], and for lifetime binge-eating behavior 0.65 (95% CI 0.54–0.74). The genetic correlation was estimated as 0.35 (95% CI 0.25–0.46) and the covariance between ADHD and binge-eating behavior was primarily explained by genetic factors (91%). Non-shared environmental factors explained the remaining part of the covariance.

Conclusions. The association between adult ADHD symptoms and binge-eating behavior in females is largely explained by shared genetic risk factors.

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Key words: ADHD, binge eating, bivariate twin analysis, heritability, twins.

Introduction

Attention-deficit hyperactivity disorder (ADHD), characterized by impairing hyperactivity/impulsivity and inattention often co-occurs with psychiatric and medical disorders (Kessler *et al.* 2006; Barkley *et al.* 2008). Epidemiological studies identified comorbidities between ADHD and binge eating across the lifespan (Cortese *et al.* 2007*a*; Reinblatt *et al.* 2015). Binge eating is a key symptom in eating disorders such as bulimia nervosa (BN), binge-purge subtype of anorexia nervosa (AN), and binge-eating disorder (BED) (American Psychiatric Association, 2013). Prospective follow-up studies implicate childhood ADHD as a risk factor for conditions involving binge eating (Sonneville *et al.* 2015), binge-purge behaviors (Bleck & DeBate, 2013), and BN (Mikami *et al.* 2008) in adolescence. Case–control studies revealed an increased risk for BN in women with ADHD, but not in children or males (Surman *et al.* 2006). Children in these studies

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were probably too young to have developed BN during the follow-up. Another study (Biederman *et al.* 2007) described increased risk for BN and AN in girls with ADHD *v.* controls. Conversely, increased risk for ADHD was found in clinical populations characterized by overeating and obesity across the lifespan, such as child psychiatric services, obese adults undergoing bariatric surgery (Cortese *et al.* 2007*a*, *b*; Davis *et al.* 2009*a*), or adults with BN (Seitz *et al.* 2013). Adult ADHD cases may differ from those with childhood onset (Moffitt *et al.* 2015), highlighting the importance of studying the association between ADHD symptoms and binge eating in adults.

ADHD and binge eating may implicate overlapping neurobehavioral circuits, involving problems with response inhibition, emotional regulation, and reward processing (Seymour *et al.* 2015). Binge eating, BN, and the binge-purge subtype of AN have been associated with behavioral impulsivity (Engel *et al.* 2005; Rosval *et al.* 2006), which is a key component in ADHD symptomatology. Genetically influenced behavioral traits such as delay aversion and low inhibitory control play a role in ADHD (Solanto *et al.* 2001; Sonuga-Barke & Fairchild, 2012) and binge eating (Davis *et al.* 2010; Seymour *et al.* 2015). Individuals with both ADHD and binge eating may represent a subgroup with specific therapeutic needs. In order to develop effective prevention and treatment strategies, it is important to determine to what extent the overlap between ADHD and binge eating reflects shared genetic and/or environmental factors. Genetic factors were established for both ADHD, with heritability estimates ranging from 30–40% for self-reported data in adults to 60–90% for childhood and adult clinical samples (Franke *et al.* 2012; Larsson *et al.* 2013; Brikell *et al.* 2015), and for binge-eating behaviors and BEDs (heritability estimated between 41% and 70%) (Bulik *et al.* 1998; Reichborn-Kjennerud *et al.* 2004; Bulik *et al.* 2010; Mitchell *et al.* 2010; Root *et al.* 2010; Trace *et al.* 2013*a*). No twin study has examined the genetic and environmental factors shared between ADHD symptoms and binge eating.

We examined the association between ADHD symptoms with lifetime binge-eating behavior, BED, and BN, based on self-reported symptoms in a large adult twin population. We evaluated the extent to which the association between ADHD symptoms and binge-eating behavior is due to genetic and environmental factors using twin methods. The hyperactive/impulsive (HI) and inattentive (IN) symptom dimensions of ADHD co-vary (Willcutt *et al.* 2012) and share some genetic factors (McLoughlin *et al.* 2007; Larsson *et al.* 2013), but specific genetic influences for each symptom dimension have been identified (McLoughlin *et al.* 2007). Therefore, we examined separately how binge-eating behavior associates with HI and IN symptom dimensions.

Methods and materials

Study population

This study used data from the national Swedish Twin Registry, the Study of Twin Adults: Genes and Environment (Lichtenstein *et al.* 2006). The regional ethics committee of Karolinska Institutet, Stockholm, Sweden approved the project. All participants provided informed consent. From the target population of 42 582 Swedish adult twins, born 1959–1985, N = 25 491 (60%) responded. Participants received personal login to the study's website, containing a questionnaire on lifestyle, physical and mental health, described in earlier publications (Lichtenstein *et al.* 2002; Furberg *et al.* 2008; Friedrichs *et al.* 2012; Trace *et al.* 2012; Capusan *et al.* 2016). Non-responders received three reminders, and were offered the alternative of a telephone interview with a trained interviewer, and an additional self-administered paper questionnaire, instead of the web page. The total study population comprised 14 184 (55.6%) women, mean age = 33.6 years (S.D. 7.6 years; range 20–46 years) and 11 307 (44.4%) men, mean age = 33.7 years (S.D. 7.6 years; range 20–46 years); 23 767 (93.2%) individuals provided data on binge eating, 18 168 (71.3%) on ADHD symptoms, and 18 029 (70.7%) on both. A standard similarity questionnaire, validated with DNA analysis (Lichtenstein *et al.* 2002; Peterson *et al.* 2016), determined zygosity. As men had low prevalence for binge-eating behavior, with only one concordant MZ pair and no concordant DZ pairs, we included only females in the twin analysis; 14 184 female twins in 10 373 pairs (3811 complete and 6562 incomplete pairs with only data from one individual in the pair available). Zygosity was not possible to establish in 411 individuals (2.9%) from 147 complete, 117 incomplete pairs. The sample included 13 773 female twins: 7328 individuals (53.2%) [4312 (31.3%) MZ and 3016 (21.9%) same sex DZ] from 3664 complete pairs and 6445 individuals (46.8%) from incomplete pairs [950 MZ (6.9%), 1073 (7.8%) same sex DZ and 4422 (32.1%) opposite sex DZ individuals].

Measures

ADHD

Current ADHD symptoms were assessed with the 18 items (nine HI and nine IN items) from the Diagnostic and Statistical Manual of Mental Disorders, Text revision (DSM-IV-TR) (American Psychiatric Association, 2000). Response options for all items were:

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0 = 'no', 1 = 'yes, to some extent', and 2 = 'yes' (online Supplementary material). Re-assessment of a subsample ($n = 54$) 2 years later with the Adult ADHD Self-Report Scale (ASRS) (Kessler *et al.* 2007) found strong a correlation, estimated to 0.63 ($p < 0.0001$) with the initial ADHD measures, indicating stability over time for ADHD symptoms (Larsson *et al.* 2013), corresponding with previous research on self-reported adult ADHD symptoms (Boomsma *et al.* 2010). Individuals with elevated ADHD scores also displayed co-morbidities similar to those found in clinical ADHD cases (Friedrichs *et al.* 2012).

For twin analysis, we used the sum of ADHD symptom scores. We also created two variables based on sum scores of the nine HI and the nine IN symptoms. For descriptive purposes, we created diagnosis-like cut-offs for ADHD using the norm-based approach proposed by Barkley *et al.* (2002), described in earlier studies (Friedrichs *et al.* 2012; Capusan *et al.* 2016). Using this method, participants scoring two standard deviations (2 S.D.) above the mean on the HI, IN, or both ADHD symptom scales were scored positive for ADHD.

Binge-eating behavior

Binge-eating behavior was assessed via self-report items based on the Structured Clinical Interview for DSM-IV-TR (SCID) (First *et al.* 2002). Questions had a 'branching' format: subsequent questions were only asked if participants answered yes to the first (gate) question/s in the section. See questions on binge eating and related disorders in online Supplementary material.

A lifetime history of binge-eating behavior was coded positive, if the participant answered yes to both having experienced eating binges and loss of control over food intake. Frequency and duration criteria were not required. We also evaluated binge-eating behavior using DSM-5 frequency and duration criteria (Trace *et al.* 2012): recurrent eating binges and loss of control at least four times/month for at least 3 months (DSM-5 binge-eating behavior).

Lifetime BED and BN were defined using self-report symptoms (SCID) based on DSM-5 criteria. BED was judged present if the participant reported binge eating at least four times/month for at least 3 months, without compensatory behaviors (self-induced vomiting, diet pills, diuretics, laxatives, exercise more than 2 h daily, not eating, or other methods to prevent weight gain when binge eating); endorsed at least three additional BED symptoms; and reported feeling distressed over binge eating. BN was defined as binge eating at least four times/month for at least 3 months, coupled with recurrent inappropriate compensatory behaviors in association with binge eating, and self-evaluation unduly influenced by body shape and weight.

Statistical analysis

Descriptive statistics were used to characterize the study population (Table 1). We used mixed-effects logistic regression, with a random effect shared between twins in the same pair, adjusted for sex and age at assessment, to calculate prevalence odds ratios (ORs) and 95% confidence intervals (CIs), as measures of association between ADHD symptoms (2 S.D. cut-off) and binge-eating behavior, BED, and BN using Stata 11.2 (StataCorp LP, College Station, Texas, USA).

We used structural equation modeling to perform maximum-likelihood model-fitting with OpenMx (Boker *et al.* 2011) for ADHD symptoms and binge-eating behavior in female twins. This allows inclusion of individuals, with information from only one twin in a pair available. In opposite sex DZ twins, males' results were set as missing. We used the full information maximum likelihood method to handle missing data. We also fitted models using female–female pairs only, with results in line with current results (online Supplementary material). Low power prevented analysis of associations of ADHD symptoms with BED and BN.

Individuals from MZ twin pairs share 100%, while DZ pairs share, on average, 50% of their segregating genes. MZ and DZ twins are assumed to share family environment equally. Higher twin correlations (within twin pair correlation for a given trait) for MZ compared with DZ twins indicate the role of additive genetic factors (A), reflecting additive effects of different alleles; MZ correlations greater than twice the DZ correlations indicate non-additive effects (dominance, D), reflecting interaction effects between alleles at the same genetic locus. DZ correlations greater than half the MZ correlations suggest an effect of shared environment (C), i.e. environmental factors common to both twins. MZ correlations lower than 1 indicate the role of non-shared environmental effects (E), i.e. environmental factors acting to make twins different, including measurement error. Cross-twin, cross-trait correlations (CTCT) – the correlation between twin 1's status on ADHD and twin 2's status on binge-eating behavior, and *vice versa* – can indicate genetic and/or environmental factors shared between the two traits. All models were adjusted for age at time of assessment, used as a covariate.

We applied structural equation modeling to estimate how much the variance of ADHD symptoms, binge-eating behavior, and the covariance between them, was explained by A, C, D, and E. Different models were fitted to the data, including models limited to

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Table 1. *Distribution of binge-eating behavior, DSM-5 binge eating, BED, and BN in the population and ADHD symptoms (norm-based 2 s.d. method) in Swedish adult twins*

	Total responders for binge-eating variables	Number who endorsed binge-eating variables *n* (%)	Total responders for ADHD symptoms and binge eating variables *N*	Positive for ADHD symptoms[1] n_1 (% of total *N*)	Positive for binge-eating variables in those positive for ADHD symptoms n_2 (% of n_1)[2]
Binge-eating behavior[3]					
Total	23 767	639 (2.69)	18 029	1575 (8.74)	113 (7.17)
Male	10 419	54 (0.52)	7244	629 (8.68)	13 (2.07)
Female	13 348	585 (4.38)	10 785	946 (8.77)	100 (10.57)
DSM-5 binge eating[4]					
Total	23 673	355 (1.50)	17 948	1559 (8.69)	58 (3.72)
Male	10 410	21 (0.20)	7238	626 (8.65)	2 (0.32)
Female	13 263	334 (2.52)	10 710	933 (8.71)	56 (6.0)
BED[5]					
Total	23 671	43 (0.18)	17 947	1559 (8.69)	7 (0.45)
Male	10 410	4 (0.04)	7238	626 (8.65)	0 (0)
Female	13 261	39 (0.29)	10 709	933 (8.71)	7 (0.75)
BN[5]					
Total	23 377	277 (1.18)	17 778	1543 (8.68)	48 (3.11)
Male	10 276	12 (0.12)	7167	617 (8.61)	2 (0.32)
Female	13 101	265 (2.02)	10 611	926 (8.73)	46 (4.97)

BED, binge-eating disorder; BN, bulimia nervosa; ADHD, attention-deficit hyperactivity disorder; 2 s.d., 2 standard deviations; DSM-5, Diagnostic and Statistical Manual of Mental Disorders, Fifth edition.

[1] ADHD symptoms, norm-based, 2 s.d. cut-off method.

[2] Per cent of those positive for ADHD symptoms.

[3] Binge eating ever, with concomitant loss of control required, no duration and frequency restrictions.

[4] Binge eating for at least 3 months, at least four times/month.

[5] BED and BN based on DSM-5 criteria.

components A, C, and E (ACE model); A, D, and E (ADE model); and A and E (AE model) (Neale & Cardon, 1992). Based on previous research and on the observed twin correlations in our sample, all fitted models included additive genetic components. Models were compared with a saturated model (i.e. all means and covariance matrices were allowed to differ for different types of twins) using a likelihood ratio χ^2 test. This test can indicate if the model fits the data significantly worse than the fully saturated model. To identify the best-fitting model, we also computed the Akaike Information Criterion (AIC). Lower AIC values indicate better quality of the model for observed data. As AIC favors parsimony, models with fewer parameters adequately explaining the data are favored (Table 2).

ADHD symptom count was a continuous variable, while binge-eating behavior was a binary variable. We used a liability-threshold approach, assuming an underlying normally distributed liability to binge-eating behavior. With this method, observed binge-eating behavior was 1 if the liability was above a threshold and 0 (no binge-eating behavior) if below. The phenotypic correlation refers to the correlation between ADHD symptoms and this underlying liability to binge-eating behavior. We used a univariate ACE model, to estimate heritability for ADHD-symptoms and binge-eating behavior and a bivariate-correlated factors model to estimate additive genetic (r_A), shared environmental (r_C), and non-shared environmental (r_E) correlations between ADHD and binge-eating behavior. Similarly, in an ADE model, we estimated r_A, dominant genetic (r_D), and r_E correlations, and in the AE model r_A and r_E. These correlations indicate the extent to which genetic and environmental influences on one measure correlate with those on the other. We also calculated the proportion of the phenotypic correlation between ADHD and binge-eating behavior explained by genetic and environmental factors. In a Cholesky decomposition (Fig. 1*a*), the ordering of the variables is important; the variable to the left is allowed to explain variance in variables to the right, but not *vice versa*. Consequently, factor A1 stands for the genetic factors for one trait (ADHD in this case), including

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Table 2. *Quantitative genetic model fitting for ADHD symptoms, binge-eating behavior in Swedish female twins (males set as missing), compared with the saturated model*

	−2×log likelihood	Degrees of freedom	AIC[1]	*p* value[2]
Univariate model				
ADHD				
Saturated	62 861.39	10 630	41 601.39	NA
ACE	62 868.67	10 636	41 596.67	0.30
ADE	62 869.16	10 636	41 597.16	0.26
AE	**62 869.16**	**10 637**	**41 595.16**	**0.35**
Binge-eating behavior				
Saturated	4534.44	13 022	−21 509.6	NA
ACE	4535.41	13 025	−21 514.6	0.81
ADE	4535.31	13 025	−21 514.7	0.83
AE	**4535.41**	**13 026**	**−21 516.6**	**0.91**
Bivariate model (ADHD and binge-eating behavior)				
Saturated	67 255.39	23 644	19 967.39	NA
ACE	67 281.18	23 658	19 965.18	0.03
ADE	67 281.94	23 658	19 965.94	0.02
AE	**67 282.07**	**23 661**	**19 960.07**	**0.06**

A, additive genetic factors; D, dominant genetic factors; C, shared environmental factors; E, non-shared environmental factors; ADHD, attention-deficit hyperactivity disorder.

[1] AIC – Akaike Information Criterion. Lowest AIC, indicating the best fitting model is highlighted in bold.

[2] *p* value (compared with the saturated model).

those shared with the other trait (binge-eating behavior). A significant path a12 will indicate shared genetic effects. A2 are the unique (residual) genetic factors for binge-eating behavior, and *vice versa* for ADHD if the order of the variables is reversed. For simplicity, Fig. 1*a* and *b* only illustrate genetic factors, but environmental factors were modelled using the same pattern.

Further we examined how binge-eating behavior was associated with HI and IN ADHD symptom dimensions, respectively, using bivariate models. In both sets of analyses, phenotypic correlations, intraclass correlations and CTCT adjusted for age at assessment were estimated, similar to the main analysis. In order to determine if genetic or environmental effects shared with binge-eating behavior were specific to HI or IN symptoms, we fitted two separate trivariate models (Cholesky decomposition): first a model to estimate phenotypic correlation between HI and binge-eating behavior when controlling for IN (partial correlation), and second the partial correlation between IN and binge-eating behavior when controlling for HI. Similarly, we estimated genetic and non-shared environmental correlations between HI and binge-eating behavior when controlling for IN and vice-versa for IN, controlling for HI. In the trivariate model, the factor A1 in Fig. 1*b* represents genetic factors in common for HI, IN, and binge-eating behavior. Factor A2 captures additional factors unique to IN and shared with binge-eating behavior. A significant path between A2 and binge-eating behavior, labeled a23 in Fig. 1*b*, indicates effects associated with IN (but not HI) shared with binge-eating behavior. Similarly, genetic effects shared between HI (but not IN) and binge-eating behavior can be calculated, setting IN first in the model.

Results

Table 1 displays descriptive statistics for binge-eating behavior in the population by sex and by ADHD symptom status. The prevalence of binge-eating behavior was low. Of 23 767 individuals providing data, 639 (2.69%), males = 54 (0.52%) and females = 585 (4.38%), reported lifetime binge-eating behavior. $N = 43$ (0.18%) endorsed symptoms meeting criteria for BED, and $n = 277$ (1.18%) for BN. Low prevalence in males decreased the population prevalence (Table 1).

Those with ADHD symptoms (2 S.D. cut-off) had significantly increased risk for binge-eating behavior [OR 3.65 (95% CI 2.72–4.91), $p < 0.001$] and DSM-5 binge-eating behavior [OR 3.01 (95% CI 2.09–4.35), $p < 0.001$] compared with those without ADHD symptoms. Both BED [OR 2.55 (95% CI 1.11–5.86), $p < 0.05$] and BN [OR 3.09 (95% CI 2.09–4.56), $p < 0.001$)] were significantly more common in adults with ADHD symptoms (online Supplementary material).

Subsequent analysis focused on binge-eating behavior in female twins only. We observed a statistically significant phenotypic correlation of 0.20 (95% CI 0.15–0.26) between ADHD symptom count and binge-eating behavior. Twin correlations and CTCT correlations indicated genetic factors contributing to ADHD symptoms, binge-eating behavior, and the covariance between the two phenotypes (online Supplementary material).

Univariate model fitting for ADHD and for binge-eating behavior indicated AE as best fitting models, not significantly different from the saturated models and with lowest AIC values (Table 2). Univariate analysis showed moderate heritability for ADHD (0.42, 95% CI 0.41–0.44) and high heritability for binge-eating behavior (0.65, 95% CI 0.54–0.74). Bivariate model fitting also indicated AE as the best fitting model. The genetic correlation was estimated at 0.28 (95% CI 0.17–0.40) and the non-shared environmental correlation at 0.10 (95% CI −0.04 to 0.24). Shared genetic factors explained 91% of the covariance between ADHD and binge-eating behavior. Non-shared environmental effects (E) accounted for

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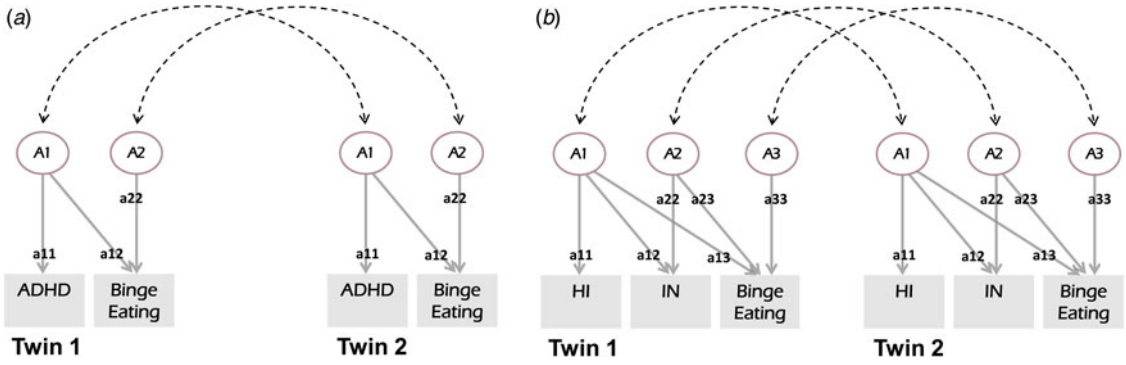

Fig. 1. (*a*) Bivariate Cholesky decomposition of genetic factors shared by ADHD and binge-eating behavior. A1 = genetic factors for ADHD and shared with binge-eating behavior (path a12), A2 = residual (unique) genetic factors for binge-eating behavior (when binge eating is set second in the model). (*b*) Trivariate Cholesky decomposition of unique genetic factors shared by IN and binge-eating behavior when controlling for common factors for HI, IN, and binge-eating behavior. A1 = factors in common between IN and HI shared with binge-eating behavior, A2 = additional factors unique to IN shared with binge-eating behavior. HI, hyperactive impulsive adult ADHD symptoms; IN, inattentive adult ADHD symptoms and binge-eating behavior (when binge eating is set second in the model); ADHD, attention-deficit hyperactivity disorder.

the remaining 9% of the covariance (Table 3). Figure 2 shows the proportion of shared *v.* residual (unique) genetic and environmental effects explaining the variability of each phenotype (ADHD respectively binge eating).

We analyzed separately how binge-eating behavior was associated with HI and IN ADHD symptom dimensions. Phenotypic correlations between binge-eating behavior and both HI (0.18, 95% CI 0.12–0.24) and IN (0.18, 95% CI 0.13–0.24) symptoms were similar. CTCT indicated shared genetic factors for both HI and IN with binge-eating behavior (online Supplementary material). The partial correlation between binge-eating behavior and the IN symptom dimension when controlling for HI (0.10, 95% CI 0.06–0.13) was stronger than the partial correlation between binge-eating behavior and the HI symptom dimension (0.03, 95% CI −0.01 to 0.07) (Table 4). Genetic correlation for the IN symptoms and binge-eating behavior remained statistically significant when controlling for factors shared with HI (0.28, 95% CI 0.13–0.42). In contrast, genetic and environmental correlations between the HI symptom dimension and binge eating attenuated substantially and became non-significant, when controlling for factors shared with IN.

Discussion

This study explored the association between ADHD symptoms in adults and lifetime binge-eating behavior in a population-based sample of twins. Shared genetic factors explained most of this association in females. Future genomic studies for ADHD and binge eating should focus on identifying such shared cross-disorder genetic risks. Better understanding of the nature of associations between ADHD and binge eating is useful when developing novel early intervention strategies and, thereby, possibly preventing the adverse correlates of binge eating, such as obesity, anxiety, depression, and suicidal risk (Davis, 2015; Welch *et al.* 2016).

Phenotypic associations

Like previous studies (Surman *et al.* 2006; Cortese *et al.* 2007*b*; Bleck & DeBate, 2013), we found ADHD symptoms in adults significantly associated with increased binge-eating behavior, as well as with BED and BN. Results are in accord with follow-up studies identifying childhood ADHD as risk factor for binge eating (Biederman *et al.* 2007; Bleck & DeBate, 2013; Sonneville *et al.* 2015). The lower prevalence of BED and BN in our population compared with other studies may partly be due to low prevalence in males and partly to geographical differences. A recent Finnish study estimated BED prevalence to 0.7% (Mustelin *et al.* 2015), closer to our results and in contrast to US data suggesting BED prevalence around 3% (Davis, 2015). Further studies are necessary to determine the prevalence of binge-eating behaviors and disorders and their association with clinically diagnosed ADHD. We found a considerable sex difference, with lower prevalence of binge-eating behaviors in men. Eating disorders are less common in men, but men may also under-report binge-eating symptoms due to feelings of shame and fear of stigmatization (Strother *et al.* 2012; MacLean *et al.* 2015). Other approaches are necessary, such as using clinical samples in primary health care and psychiatry or questionnaires adapted to how men experience eating disorders

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Table 3. *Parameter estimates (95% CI[1]) for ACE, ADE and AE univariate models for ADHD symptoms; binge-eating behavior; and from bivariate model for ADHD and binge-eating behavior (binge eating) in a population of 13 773 adult Swedish female twins (3664 complete twin pairs)*

	A2	C2	D2	E2	r_A	r_C	r_D	r_E	Bivariate[2] A2	Bivariate[2] C2	Bivariate[2] D2	Bivariate[2] E2
ACE												
ADHD	0.37 (0.24, 0.46)	0.05 (0.05, 0.05)		0.58 (0.54, 0.61)								
Binge eating	0.62 (0.25, 0.74	0.03 (0.01, 0.33)		0.35 (0.26, 0.47)								
ADHD and binge eating					**0.46 (0.15, 0.96)**	**−1 (−1 to −1)**		**0.03 (−0.08 to 0.15)**	**1.01 (0.31, 1.76)**	**−0.17 (−0.70 to 0.50)**		**0.07 (−0.19 to 0.33)**
ADE												
ADHD	0.42 (0.25, 0.46)		0.01 (0.01, 0.01)	0.58 (0.54, 0.61)								
Binge eating	0.52 (0, 0.74)		0.13 (0.00, 0.73)	0.35 (0.25, 0.46)								
ADHD and binge eating					**0.34 (1.00 to −1.00)**		**1 (−1 to 1)**	**0.04 (−0.07 to 0.15)**	**0.77 (−0.41 to 1.16)**		**0.14 (0.00, 1.35)**	**0.09 (−0.17 to 0.34)**
AE												
ADHD	0.42 (0.41, 0.44)			0.58 (0.56, 0.60)								
Binge eating	0.65 (0.54, 0.74)			0.35 (0.26, 0.46)								
ADHD and binge eating					**0.35 (0.25, 0.46)**			**0.04 (−0.07 to 0.16)**	**0.91 (0.66, 1.16)**			**0.09 (−0.16 to 0.34)**

A, additive genetic factors; D, dominant genetic factors; C, non-shared environmental factors; E, non-shared environmental factors; ADHD, attention-deficit hyperactivity disorder.

[1] 95% confidence interval.

[2]Bivariate A (bivariate heritability) refers to the amount of covariance between the two phenotypes explained by A, similarly for C, D, and E. Best fitting model for the bivariate analysis is highlighted in bold.

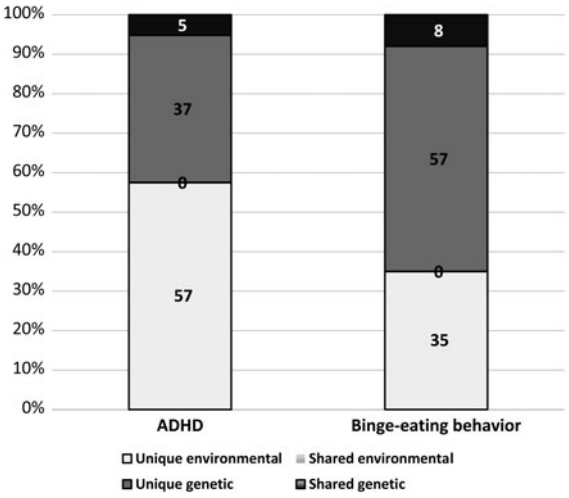

Fig. 2. Proportion of shared *v.* residual (unique) genetic and environmental effects explaining the variability of ADHD, when binge-eating behavior is set first in the model (Cholesky decomposition), respectively, of binge-eating behavior when ADHD is set first in the model. ADHD, attention-deficit hyperactivity disorder.

(Anderson & Bulik, 2004), to investigate binge eating and related problems in males.

Shared genetic and environmental factors

Our results suggest the association between ADHD symptoms and binge-eating behavior being primarily due to shared genetic factors. Part of this genetic overlap may reflect genetic risk variants with general effects cutting across traditional boundaries between neuropsychiatric traits and disorders (Pettersson *et al.* 2016). Identification of cross-disorder genetic risks is one of the challenges to understanding the etiology of neuropsychiatric disorders.

The finding of shared genetic risk factors may also partly reflect shared neurocognitive underpinnings of ADHD and binge eating (Seymour *et al.* 2015). These involve problems with executive and cognitive function, and emotional regulation, such as negative urgency (Racine *et al.* 2013). Individuals with ADHD also display deficits in inhibitory control, vigilance, planning (Carmona *et al.* 2012; Coghill *et al.* 2014), and emotional regulation (Shaw *et al.* 2014), leading to suboptimal decision-making and preference for immediate *v.* delayed rewards (Solanto *et al.* 2001; Sonuga-Barke & Fairchild, 2012). Deficits in inhibitory control and preference for immediate rewards are also exhibited in individuals with binge-eating behaviors, BN, binge-purge AN (Brogan *et al.* 2010; Wu *et al.* 2013), and BED (Davis *et al.* 2010). Dopamine (DRD2) receptor variability has been described in obese individuals with BED (Davis *et al.* 2009*b*) and in ADHD (Franke *et al.* 2012), suggesting possible genetic overlap. Also dopamine D3 receptor implicated in HI ADHD symptoms and binge eating may have a role in the association (Davis *et al.* 2009*a*).

Another mechanism may involve the addictive potential of highly palatable foods (such as sweet and fatty and salty and fatty foods) that, similarly to psychoactive substances, activate dopamine release in mesolimbic reward pathways, increasing the risk for overeating (Gearhardt *et al.* 2011*a*; Schulte *et al.* 2016). Several studies suggest an addictive dimension to obesity (Volkow *et al.* 2013) and BED (Gearhardt *et al.* 2011*b*). Common genetic factors have been identified for BN (involving binge eating) and alcoholism (Trace *et al.* 2013*b*) as well as between ADHD and alcoholism (Edwards & Kendler, 2012; Capusan *et al.* 2015). It could be speculated that shared genetic factors of ADHD symptoms and binge eating may partly reflect common genetic pathways for different addictive behaviors (Blum *et al.* 2000). Genetically influenced traits such as delay aversion and low inhibitory control were found in individuals with ADHD (Solanto *et al.* 2001; Sonuga-Barke & Fairchild, 2012) and females with binge eating and related disorders (Davis *et al.* 2010; Wu *et al.* 2013). These may be particularly detrimental in environments with pervasive food-related cues, and with highly palatable foods easily available at all times (Davis *et al.* 2009*b*; Gearhardt *et al.* 2011*b*). The role of food-related addictive behaviors in binge eating and the association with ADHD needs further exploration in clinical samples.

In addition, overlap between ADHD and binge-eating behavior may also reflect ADHD dimension-specific genetic effect. We found a small but significant genetic correlation between the IN symptoms and binge-eating behavior, even after controlling for the genetic effects shared with HI symptoms. There was however no evidence supporting specific genetic effects for the HI symptom dimension. This is somewhat surprising given that HI in girls has been associated with eating problems later in life (Mikami *et al.* 2008). In our study, we analyzed the association between ADHD symptoms in adults and binge eating. HI symptoms tend to decrease at a higher rate with age compared with IN symptoms (Biederman *et al.* 2000). We have no information on whether women reporting mainly IN symptoms, had more HI symptoms in their childhood. Future genomic studies on the association between ADHD and binge eating may benefit from including information about ADHD symptom dimensions and/or subtypes.

In the univariate analysis, 42% heritability estimate for ADHD is in line with previous twin studies based on self-report data (Boomsma *et al.* 2010; Franke *et al.* 2012), but lower than heritability estimates for

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Table 4. *Association between HI ADHD symptoms and binge-eating behavior, when controlling for IN, and associations between IN and binge-eating behaviors when controlling for HI*

	Association between HI and binge-eating behavior when controlling for IN:		Association between IN and binge-eating behavior when controlling for HI	
	Estimate	(95% CI)	Estimate	(95% CI)
Partial correlation	0.03	(−0.01 to 0.07)	0.10	(0.06–0.13)
Genetic correlation	0.03	(−0.14 to 0.18)	0.28	(0.13–0.42)
Environmental correlation	0.05	(−0.06 to 0.16)	0.00	(−0.10 to 0.11)

HI, hyperactive/impulsive ADHD symptoms; IN, inattentive ADHD symptoms; 95% CI, 95% confidence interval; ADHD, attention-deficit hyperactivity disorder.

childhood ADHD and clinically diagnosed adult ADHD (around 60–90%) (Larsson *et al.* 2014). Differences in heritability estimates have previously been attributed to rater effects (Brikell *et al.* 2015) and measurement error in self-report data (Franke *et al.* 2012). However, molecular genetic studies show similar polygenic risks (Levy *et al.* 1997; Martin *et al.* 2014) in clinical ADHD samples as for those associated with ADHD symptoms in the population, supporting the use of population samples for the study of ADHD (Faraone *et al.* 2015). For binge-eating behavior, heritability was estimated as 65%, which is higher than one Norwegian study [41% (95% CI 31–50%) (Reichborn-Kjennerud *et al.* 2004)], but in line with another study assessing binge eating in women [70% (95% CI 26–77%) (Root *et al.* 2010)].

Limitations

Presented results should be considered in the context of several limitations. Response rates of 60%, similar to other large epidemiological surveys, were relatively low. Drop-out has previously been attributed to unwillingness to answer to a survey with over 1300 questions. Drop-out analyses, described earlier (Furberg *et al.* 2008; Friedrichs *et al.* 2012; Larsson *et al.* 2013), found that non-responders did not significantly differ from responders regarding birth weight and age; however, non-responders were significantly more often male, had a parent/s born outside of Sweden, had been diagnosed with a psychiatric condition, and convicted for any type of crime. As ADHD is associated with psychiatric disorders (Kessler *et al.* 2006), and is more prevalent in prison populations (Edvinsson *et al.* 2010; Ginsberg *et al.* 2010), individuals with more severe ADHD probably did not respond to the questionnaire, limiting generalizability of our findings to the more severe end of the ADHD spectrum.

All data were based on self-reported symptoms in the general population. Self-reported ADHD symptoms show satisfactory psychometric properties (Murphy & Schachar, 2000; Sandra Kooij *et al.* 2008), and were found to be stable over time (Larsson *et al.* 2013). Information on functional impairment or childhood onset was not available. Our sample probably includes subthreshold cases, with ADHD symptoms, without fulfilling criteria for a clinical diagnosis that may be the less severe manifestations of the syndrome (Faraone *et al.* 2006).

Reliability of self-reported data on binge eating and related conditions is more unclear. An earlier twin study found low reliability and lower heritability estimates, around 50% (Bulik *et al.* 1998) for binge eating reported by just one assessment. Described results reflect an association between ADHD symptoms and binge-eating behavior, in about half of the cases outside DSM-5 BED and BN cut-offs. Given the low prevalence in our sample compared with international findings, it is likely that binge eating was underreported and our measures are conservative, probably restricting generalizability to the milder end of the spectrum in the population.

Heritability estimates were based on only eight female DZ twins concordant for binge-eating behavior, possibly affecting precision of our estimates. Despite a large study population, statistical power was too low to examine genetic and environmental aspects of the association in males, which is an important limitation and calls for future research using alternative methodologies that more accurately capture symptom patterns in males and could therefore yield different results.

Although we assess lifetime binge eating, ADHD is assessed as current symptoms. As data are cross-sectional, we are not able to draw any inference regarding whether ADHD leads to binge eating in adults. Longitudinal follow-up research found that ADHD in children and adolescents is a risk factor for later

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binge eating (Biederman *et al.* 2007; Bleck & DeBate, 2013; Sonneville *et al.* 2015). Further longitudinal clinical studies are necessary to elucidate the temporal nature of the association between ADHD and binge eating in adults.

Results may also be influenced by inherent limitations in twin studies. For instance, basic assumptions in twin studies include random mating in the population. Assortative mating in some psychiatric conditions, including ADHD (Nordsletten *et al.* 2016) may lead to underestimating heritability. Conversely, gene–environment interactions between the easy availability of highly palatable foods and genetically determined characteristics, such as reduced inhibitory control or emotional dysregulation in individuals with ADHD, cannot be excluded. In a twin study, these effects would be subsumed in the heritability estimates, possibly overestimating common genetic factors in bivariate models.

In conclusion, this study suggests that the association between adult ADHD symptoms and lifetime binge-eating behavior is primarily due to shared genetic risk factors in females. Clinicians need to be aware of these associations when assessing and managing individuals presenting with ADHD symptoms or binge-eating behavior.

Supplementary Material

The supplementary material for this article can be found at https://doi.org/10.1017/S0033291717001416.

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