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Associations between Attention Deficit Hyperactivity Disorder Symptom Dimensions and Disordered Eating Symptoms in Adolescence: A Population-Based Twin Study

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Abstract

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Authors' Contributions

ZY was responsible for developing the research question, study conceptualization and design, critical review of the literature, interpretation of data, and manuscript preparation. MJQ and PSP assisted with literature search, interpretation of data, and manuscript preparation. LMT, CMB, KNJ, SY, and PL contributed to the development of the research question, study design, interpretation of data, and critical review of the manuscript. HL was involved in study design, data preparation and interpretation, and critical review of the manuscript. JHB was responsible for study design, data acquision and analysis, interpretation of data, and manuscript preparation. All authors have read and approved the current version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Conflict of Interest

KNJ owns equity shares in Sanofi and Centene Corporation. CMB is a Scientific Advisory Board member for Shire, serves as a consultant for Idorsia (consultant), is an author with and royalty recipient from Pearson, and is on the Clinical Advisory Board of Equip Health Inc. HL reports receiving grants from Shire Pharmaceuticals, personal fees from and serving as a speaker for Medice, Shire/Takeda Pharmaceuticals and Evolan Pharma AB, and sponsorship for a conference on attention-deficit/hyperactivity disorder from Shire/Takeda Pharmaceuticals and Evolan Pharma AB. All other authors report no financial interests or potential conflicts of interest.

Ethics Approval

The Swedish Twin study of CHild and Adolescent Development (TCHAD) was approved by the Ethics Committee of Karolinska Institutet, Stockholm, Sweden. This study was also approved by the Institutional Review Board of the University of North Carolina at Chapel Hill, where the data were analyzed.

Code Availability

The code used for data analysis as a part of the current study are available from the corresponding author upon reasonable request.

Although bivariate associations between attention-deficit/hyperactivity disorder (ADHD) and eating disorders in adolescent girls and boys have been previously identified, the mechanistic link underlying the symptom-level associations remains unclear. We evaluated shared genetic and environmental influences on ADHD symptoms and disordered eating in 819 female and 756 male twins from the Swedish TCHAD cohort using bivariate models. Common additive genetic and unique environmental effects accounted for majority of ADHD and disordered eating associations in a differential manner. For girls, the strongest genetic correlation was observed for cognitive/inattention problems-bulimia (.54), with genetic factors accounting for 67% of the phenotypic correlation. For boys, the strongest genetic correlations were observed for conduct problems-bulimia and hyperactivity-bulimia (~.54), accounting for 83% and 95% of the phenotypic correlation, respectively. As per our findings, the risk of comorbidity and shared genetics highlights the need for preventative measures and specialized treatment for ADHD and disordered eating in both sexes.

Keywords

Attention-deficit/hyperactivity disorder; Disordered eating; Symptom-level associations; Twin study; Population cohort; Genetics; Sex differences

Previous studies on attention deficit/hyperactivity disorder (ADHD) and eating disorders (EDs) throughout adolescence and young adulthood have reported notable comorbidity (Mikami et al. 2010; Mikami et al. 2008; Nazar et al. 2016). For instance, adolescent girls with ADHD are at almost 4-fold increased risk of developing an ED compared with those without ADHD (Biederman et al. 2007). As girls with ADHD transition into adolescence and young adulthood, 16% eventually develop an ED compared with only 5% of those without ADHD (Biederman et al. 2007). Similarly, the lifetime prevalence of ADHD in young adult women with EDs ranges from 5%−17% (Blinder et al. 2006; Wentz et al. 2005; Yates et al. 2009), which is notably higher than the global population lifetime prevalence of ADHD in children (~5%) (Sayal et al. 2018) or adults (2.2%) (Fayyad et al. 2017). Large population studies have also demonstrated a prospective relationship between ADHD and disordered eating symptoms (Bleck and DeBate 2013; Bleck et al. 2015; Mikami et al. 2010; Mikami et al. 2008; Sonneville et al. 2015; Yoshimasu et al. 2012). Specifically, binge eating, purging, and body image dissatisfaction are associated with a diagnosis of ADHD (Bleck and DeBate 2013; Mikami et al. 2008). Because disordered eating significantly increases the risk of developing an ED (Stice et al. 2008) and ED treatment outcome could be negatively affected by baseline ADHD symptoms (Testa et al. 2020), the prospective association between ADHD symptoms—even if below the threshold for diagnosis—and disordered eating could be an important indicator of future ED risk even before the onset of disordered eating symptoms.

Various hypotheses exist for how the key self-regulatory symptoms of ADHD—namely inattention and hyperactivity/impulsivity—may differentially contribute to specific disordered eating symptoms (Fleming and Levy 2002; Mikami et al. 2010; Reinblatt et al. 2014). Self-regulation problems are among the defining aspects of ADHD, and these problems negatively affect domains such as memory, attention, arousal, organizational skills,

and behavior regulation (i.e., impulsivity) (van Stralen, 2016). Importantly, similar deficits in these domains are also observed in individuals with EDs (Davis, Levitan, Smith, Tweed, & Curtis, 2006). Impulsivity could result in disinhibition and set the stage for hasty, poor decision-making involving food that could eventually lead to the development of binge eating. Indeed, impulsivity in individuals with ADHD increases the risk for binge eating and purging behaviors as well as bulimia nervosa in both sexes during adolescence (Mikami et al. 2010; Mikami et al. 2008; Wonderlich et al. 2004). On the other hand, adolescent girls with ADHD inattentive/hyperactive combined subtype are also more likely to report binge eating, purging, and body image dissatisfaction compared with girls with ADHD inattentive subtype or girls without ADHD (Mikami et al. 2008). In a national longitudinal cohort, both inattention and hyperactivity/impulsivity were also found to be associated with binge eating and purging behaviors (Bleck et al. 2015). It is possible that inattention could lead to lack of sensitivity to the internal sense of hunger, satiety, and amount of food consumed on a daily basis (Cortese et al. 2007; Fleming and Levy 2002). This potential relationship could be attributed to attentional deficits leading to a lack the organizational skills—which are required to maintain a structured meal plan—resulting in instances of significant under- or over-eating. However, the mechanism underlying these associations remains unknown.

The familial natures of ADHD and EDs, as well as many of their respective symptoms, have been well documented. Twin-based heritability estimates for disordered eating range from 40% to 60% (Munn et al. 2010; Wade et al. 1999; Yilmaz et al. 2015). To date, few studies have tested for sex differences in the magnitude of heritability estimates, but among those that have, estimates for sexes appear to be similar (e.g., after puberty) (Klump et al. 2012; Reichborn-Kjennerud et al. 2003; Reichborn-Kjennerud et al. 2004). Additionally, some variation in estimates may be present according to symptom type (e.g., primarily higher estimates in females for body shape/weight concerns) (Baker et al. 2009; Ferguson et al. 2012). The twin-based heritability estimate of ADHD in childhood is around 75% (Freitag et al. 2010; Martin et al. 2002), and heritability estimates for the inattention subtype are slightly lower for males than females (Freitag et al. 2010; Nadder et al. 2001). A 4-fold increased risk for developing an ED was reported in individuals with ADHD and their relatives compared with individuals without ADHD and their relatives, and the twin-based genetic correlations between ADHD and different ED diagnoses are between 14% and 37% (Yao et al. 2019). Additionally, ADHD polygenic score has been shown to predict binge-eating disorder in a population cohort (Hübel et al. 2021). Although sex differences appear to be present in the types of genetic factors contributing to disordered eating, there are largely common genetic effects across sexes for ADHD (Martin et al. 2018). However, the extent to which shared genetic or environmental risk factors exist for these two disorders at a symptom level (especially in the case of ADHD) is yet to be examined while being mindful of possible sex-specific effects.

The goal of the present study was to evaluate the phenotypic associations and examine the extent to which genetic and environmental factors are shared and contribute to the associations between ADHD symptom dimensions and symptoms of disordered eating in a large Swedish twin cohort of adolescent girls and boys. Based on previous literature, we hypothesized that the genetic correlations for ADHD symptom dimensions (particularly those associated with impulse dysregulation) would be stronger with behavioral disordered

eating dimensions (i.e., bulimia) than the cognitive disordered eating dimensions (i.e., drive for thinness and body dissatisfaction) (Mikami et al. 2010; Mikami et al. 2008; Wonderlich et al. 2004).

Methods

Participants

Our study sample comprises twins from the Swedish Twin study of CHild and Adolescent Development (TCHAD), which includes all twins born in Sweden between May 1985 and December 1986 (Lichtenstein et al. 2007). All twin pairs and their parents identified through the Swedish Medical Birth Registry were invited to participate. The current report includes those twins who participated in the Wave 3 assessments at which time the twins were 16–17 years old. Eighty-two percent of twins contacted at Wave 3 completed participation (Lichtenstein et al. 2007). This includes 470 monozygotic (MZ; 234 complete pairs) and 349 dizygotic (DZ; 171 complete pairs) female twins, as well as 441 MZ (214 complete pairs) and 315 DZ (153 complete pairs) male twins. Information on the family socio-economic status of participants is available in detail elsewhere (Tuvblad et al. 2006). TCHAD was approved by the Ethics Committee of Karolinska Institutet, Stockholm, Sweden. This study was also approved by the Institutional Review Board of the University of North Carolina at Chapel Hill, where the data were analyzed.

Zygosity Determination

Zygosity of each pair was determined based on computer algorithms of questionnaire responses about the twins' physical similarities and the frequency with which people confuse them with one another (Lichtenstein et al. 2007). Zygosity questions were validated by a discriminant analysis of 106 same-sex twin pairs where zygosity had been determined by typing 16 polymorphic DNA markers (Lichtenstein et al. 2007).

Measures

The Conners-Wells Adolescent Self-Report Scale: Short Form (CASS) (Conners et al. 1997), which consists of 27 items, was used to measure symptoms of ADHD. Three CASS subscales were included: conduct problems (e.g., breaks rules, trouble with police), cognitive/inattention problems (e.g., trouble organizing, poor concentration), and hyperactivity (e.g., cannot sit still, restless). Higher scores reflect elevated symptoms. All scales had acceptable reliability in the current sample (alphas = .71–.80). The conduct problems subscale score was log-transformed prior to analyses due to a positive skew. All other subscales were normally distributed.

Disordered eating symptoms were assessed using items from the drive for thinness (e.g., excessive concern with dieting, preoccupation with weight, and an extreme pursuit of thinness), bulimia (e.g., tendency toward episodes of binge eating that may be followed with the impulse to induce vomiting), and body dissatisfaction (e.g., belief that specific parts of the body are too large) subscales of the Eating Disorder Inventory-2 (EDI) (Garner 1991). To remain consistent with our previous work (Baker et al. 2009; Baker et al. 2017; Yilmaz et al. 2017), subscale scores were created by summing respective items according

to the EDI instructions. The Swedish version of the EDI has been translated and validated previously (Nevonen et al. 2006; Norring and Sohlberg 1988). Cronbach's alpha coefficients were acceptable in the sample (drive for thinness = .84, body dissatisfaction = .89, and bulimia = .64). All EDI scale scores were log-transformed prior to analysis due to a positive skew.

Body mass index (BMI) was obtained via self-reported height and weight. We used age- and sex-specific means and standard deviations (SD) from a sample of Swedish children born in 1981 to remove values 6 SD above and below the mean for height and log(weight) (Werner and Bodin 2006). We then used the World Health Organization 2007 package for R to calculate age- and sex-adjusted BMI z-scores (de Onis et al. 2007). Due to the empirical associations between BMI and disordered eating and ADHD symptoms (Waring and Lapane 2008; Westerberg-Jacobson et al. 2010), BMI was included as a covariate in twin modeling.

Statistical Analyses

For the CASS and EDI, missing data were handled as follows: subscale scores were considered missing if more than 75% of subscale items were missing. Otherwise, mean item values were imputed for the specific subscale question. This remains consistent with how we have handled missing data in previous studies (Baker et al. 2019; Baker et al. 2009).

Bivariate twin models fitted with Mx (Neale 1991) were used to decompose the covariance between disordered eating and ADHD symptoms into genetic and environmental components, controlling for the potential effect of BMI. Specifically, the classic twin design estimates the proportion of variance attributable to additive genetic effects (A; cumulative impact of many genes), common environmental effects (C; environmental factors that make twins similar), and unique environmental effects (E; environmental factors that serve to make twins dissimilar plus measurement error). The bivariate model estimates these sources of variance and also provides estimates of the genetic (r_a), common environmental (r_c), and unique environmental (r_e) correlations between ADHD and disordered eating symptoms. These correlations indicate the proportion of overlapping genetic and environmental factors between the two traits. For example, if r_a is estimated at 1.00, this suggests a 100% overlap in the genetic factors influencing ADHD and disordered eating. A raw data approach was used, which allows information from complete and incomplete pairs to be used.

The fit of the full ACE model, which estimates the proportion of variance attributable to genetic, common environmental, and unique environmental influences for each trait and the genetic, common environmental, and unique environmental correlations (indicating the respective proportions of variance that the two traits share), was compared to two nested models: the AE and CE models.[1] The AE model estimates the proportion of variance due to genetic and unique environmental influences for disordered eating and ADHD symptoms and the genetic and unique environmental correlations between the traits. The CE model allows for common and unique environmental influences and correlations to be estimated, suggesting no genetic effects on disordered eating, ADHD symptoms, or their overlap.

[1]An E model, which estimates only unique environmental variance and covariance, was not fit as—based on numerous published studies—there is substantial empirical evidence that this is a poor fitting model for both disordered eating and ADHD.

All bivariate twin models were completed separately for girls and boys; opposite-sex twin pairs were not included as we have previously shown we do not have enough power to statistically address sex differences in this sample (Baker et al. 2017). Thus, we cannot directly test the statistical significance of any differences observed between girls and boys in the twin models. First, phenotypic correlations between the disordered eating variables and the ADHD variables were calculated to inform which pairs of variables would be evaluated with bivariate modeling. Although it is possible to complete twin models with variables that have a small phenotypic association, parsing small associations into shared genetic and environmental components is limitedly informative. Thus, we required pairs of variables to have at least a moderate phenotypic correlation to be evaluated using twin modeling ($r > .20$). Similar approaches have been used previously (Baker et al. 2018; Baker et al. 2018; Baker et al. 2017; Koren et al. 2014).

Given that both disordered eating and ADHD symptoms independently show family effects (A and or C depending on age, sex, and symptom), we *a priori* defined nested submodels to apply to evaluate our aim. Nested twin models (i.e., AE and CE) were compared to the full model (i.e., ACE) using the difference in twice the negative log-likelihood of the models, which—given certain regularity conditions—is distributed as a chi-square. A significant change in chi-square indicates a worse fitting model. Model fit was also assessed with the Akaike's Information Criterion (AIC) (Akaike 1987) and the Bayesian Information Criterion (BIC) (Schwarz 1978). For both AIC and BIC, models with fewer parameters are preferable if they do not result in a significantly worse fit because they are more parsimonious. However, we present full models here as power to discern both A and C is limited.

Results

Descriptive Statistics

Mean scores for the CASS and EDI subscales are presented in Table 1. Phenotypic correlations indicated small-to-moderate associations between disordered eating symptoms and ADHD symptoms (Table 2). A Fisher r-to-z transformation indicated that there were no significant differences in the observed correlations between girls and boys. Based on the phenotypic correlations (r's > .20), the following associations were evaluated using bivariate twin modeling in girls: conduct problems-bulimia; cognitive/inattention problems-bulimia; conduct problems-body dissatisfaction; cognitive/inattention problems-body dissatisfaction; and in boys: conduct problems-bulimia; cognitive/inattention problems-bulimia; hyperactivity-bulimia; cognitive/inattention problems-body dissatisfaction.

Bivariate Twin Analysis

Cross-twin, within-trait twin correlations are provided in Table 3. These correlations provide an initial indication regarding whether genetic or environmental factors are important in risk for disordered eating and ADHD symptoms independently. Correlations were stronger for MZ twins compared with DZ twins for all symptom variables, indicating the presence of genetic effects. However, common environmental factors are also indicated for bulimia and body dissatisfaction in girls and and conduct problems and body dissatisfaction in boys as the DZ correlations are more than half the MZ correlations for the mentioned traits.

Cross-twin, cross-trait correlations were also calculated for the ADHD and disordered eating symptoms with phenotypic correlations > .20. These correlations provide an indication regarding whether each pair of symptoms share genetic or environmental factors (Table 3). Correlations that were larger for MZ twins compared with DZ twins suggest that shared genetic factors may contribute to the phenotypic associations between these specific ADHD-disordered eating symptom associations. The difference between the MZ and DZ cross-twin cross-trait correlations also indicated that common environmental factors may contribute to the associations of conduct problems and bulimia, cognitive/inattention problems and bulimia, and conduct problems and body dissatisfaction in girls. The non-significant DZ cross-twin cross-trait correlations in boys preclude hypotheses regarding the contribution of common environmental factors to these associations.

Bivariate fit results for full and nested models are presented Table 4. The AE model, estimating genetic and unique environmental effects for each phenotype and genetic and unique environmental correlations between the phenotypes, was determined by AIC and BIC to be the most parsimonious, and best-fitting model, for all bivariate models. However, power was limited to detect both A and C. Thus, any twin similarity due to C will be captured by A in an AE model, potentially inflating the genetic estimates.

Results from the full and best-fit models are presented in Table 5. Using results from the full models, heritabilities were estimated to be higher for girls for conduct problems (.50) than for boys (.36). Girls and boys had similar heritability estimates for cognitive/inattention problems (~.47) and body dissatisfaction (.36-.40 for girls and .38 for boys). Heritability for hyperactivity in boys was .46.

For girls, the largest genetic correlation was between cognitive/inattention problems and bulimia (r_a = .55) suggesting approximately 30% (r_a^2) overlap in the genetic factors responsible for these symptoms and that genetic factors accounts for about 67% of the phenotypic correlation. For boys, the genetic correlation between conduct problems and bulimia was estimated at r_a = .57, suggesting ~30% overlap. Common genetic factors account for about 83% of the phenotypic correlation.

Estimates for shared environmental correlations essentially could not be estimated as indicated by the confidence intervals that hit the bounds. Unique environmental correlations were small but significant for all pairs of symptoms in girls. However, in boys, unique environmental correlations were not significantly different from 0 for any pair of symptoms, indicating that common genetic factors account for a substantial proportion of each of the phenotypic correlations.

Discussion

Our study examined the genetic covariance of ADHD symptom dimensions and the attitudes and behaviors associated with EDs. In this population-based twin cohort, we not only replicated prior research findings showing significant associations between ADHD symptoms and disordered eating (Bleck et al. 2015; Mikami et al. 2010; Yilmaz et al. 2011), we also observed that shared genetic effects were present.

On average, similar phenotypic correlations were observed in girls and boys for all ADHD symptom and disordered eating associations. Self-regulation problems are among the defining aspects of ADHD, and these problems negatively affect domains such as memory, attention, arousal, organizational skills, and behavior regulation (i.e., impulsivity) (van Stralen, 2016). Importantly, similar deficits in these domains are also observed in individuals with EDs (Davis, Levitan, Smith, Tweed, & Curtis, 2006), which would explain the significant associations we observed across many symptoms in both sexes. However, associations between drive for thinness and ADHD symptoms and between hyperactivity and disordered eating were minimal for both boys and girls. One possible explanation is that drive for thinness as a construct may be more strongly driven by compulsive tendancies as opposed to impulsive tendencies or inattention, distinguishing it from other EDI subscales in relation to ADHD symptoms.

Results from the bivariate models showed that the bulimia subscale score had the most genetic overlap with the ADHD symptom dimensions in both sexes: cognitive/inattention problems showed the most overlap in girls and conduct problems showing the most overlap in boys. This pattern suggests that differential genetic mechanisms may underlie these associations. For example, the genetic overlap between cognitive/inattention problems and bulimia in girls may represent the inattention-based hypothesis of ADHD-ED comorbidity. More specifically, cognitive and attentional deficits have been shown to also increase risk for deficits in hunger and satiety cutes as well as disordered eating behaviors (Cortese et al. 2007; Fleming and Levy 2002). In contrast, the genetic overlap between conduct problems and bulimia in boys may align better with the impulsivity-based hypothesis, suggesting that disinhibition and poor decision-making from ADHD may influence the development of bulimia nervosa and its symptoms (Mikami et al. 2010; Mikami et al. 2008; Reinblatt et al. 2014; Wonderlich et al. 2004).

Our findings may have important biological implications for future studies. The previously observed increased risk for disordered eating resulting from ADHD may be due to genetic factors. ADHD polygenic score derived from genome-wide association studies has been shown to predict ED diagnoses, EDI scores, and disordered eating in general (Abdulkadir et al. 2022; Hübel et al. 2021; Yao et al. 2019). Furthermore, shared genetics between ADHD and disordered eating—which may be more pronounced in specific symptom pairs as per our results—could inform future subphenotypes for molecular genetic studies in an effort to reduce phenotypic heterogeneity. Findings also have notable clinical implications. Our results provide a greater understanding of how various ADHD symptoms may be used as markers of differing risk for disordered eating behaviors; these may differ between girls and boys. In order to effectively tailor prevention and treatment for adolescent girls and boys with ADHD, assessment of sex differences in symptom-based associations is critical. It is advised that parents, teachers, and health care providers display increased vigilance for the emergence of dysfunctional eating attitudes and behaviors among children and adolescents who present with ADHD symptoms, which in turn could assist with early identification of youth at risk for EDs.

The results of this study must be considered within the context of its limitations. First, we acquired ADHD symptom ratings through adolescent self-reports, and thus there is no

behavioral data that might be applicable to a clinical population. Second, the hyperactivity scale does not fully incorporate impulsivity, which might play a role in the limited association observed between hyperactivity and disordered eating symptoms. However, the CASS is regarded as a reliable and valid scale to evaluate ADHD symptoms in adolescents (Conners et al. 1997). Third, capturing heritability at a specific point in time may not be informative for other stages of development. The genetic influence for disordered eating for girls, for instance, has been shown to increase in adolescence (Fairweather-Schmidt and Wade 2015; Klump 2013), emphasizing the need for longitudinal data to elucidate the heritability of these symptoms. Fourth, there were minimal sex differences on the EDI bulimia subscale, and the mean scores for girls were higher than those for boys on CASS ADHD subscales except conduct problems. Of note, the mean bulimia subscale score was relatively low for girls, which is in line with previous findings that the incidence of bulimia nervosa is lower in Scandinavian registers than the indicated lifetime prevalence of the disorder (Larsen et al. 2021; Schaumberg et al. 2019). Of note, the alpha reliability for bulimia was also the lowest among all EDI subscales (.64). In the case of ADHD, the literature shows that ADHD is underdiagnosed in girls and symptoms are not always recognized due to the misconception that ADHD only affects boys, as well as the possibility that girls mask their ADHD symptoms better and function at a higher level than boys (Quinn and Madhoo 2014; Skogli et al. 2013). Another related explanation is that girls may be more aware of the cognitive differences they experience compared to their female peers when it comes to ADHD symptoms due to perception, social expectations, and conditioning (Quinn and Wigal 2004), which may have resulted in girls rating items pertaining to ADHD symptoms higher than boys in our study. Finally, our sample size was modest, likely limiting statistical power, and it may not have been large enough to significally detect both A and C in the final, best-fit models as the power to detect common environment is more limited.

Despite these limitations, this study has several notable strengths. First, our study utilized a population-based twin design with high response rates. Second, we examined different symptom dimensions for both disordered eating and ADHD. Third, the inclusion of girls and boys allowed for a careful evaluation of sex-specific associations. Fourth, the low psychostimulant prescription use for ADHD treatment in Sweden during the mid-to-late 1990s (Ekman and Gustafsson 2000) suggests that markedly few children and adolescents in our sample used psychostimulant medications. Fifth, we incorporated BMI as a covariate in the examination of disordered eating and ADHD symptoms since high BMI is a risk factor for disordered eating attitudes and behaviors (Westerberg-Jacobson et al. 2010), and genome-wide association studies have revealed a positive genetic correlation between ADHD and BMI (Demontis et al. 2019; Hübel et al. 2019). Additionally, the presence of an ADHD diagnosis is associated with higher BMI (Cortese et al. 2016), which may become stronger during adolescence compared to childhood (Nigg et al. 2016). Furthermore, individuals with ADHD who are not taking psychostimulants—as likely is the case in our study sample—have higher BMIs than individuals with ADHD taking psychostimulants (Waring and Lapane 2008).

In summary, our study provides evidence for shared genetic factors having a significant influence on the association between ADHD symptoms and disordered eating attitudes and behaviors. To advance prevention and treatment programs for individuals at risk for

these two comorbid psychiatric disorders, research on sex-differences at the symptom level must be prioritized. The risk of comorbidity and shared genetic effects emphasizes the prioritization of preventative measures and specialized treatment for both girls and boys.

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Availability of Data and Material

The datasets generated during and/or analysed during the current study are available from the corresponding author upon reasonable request.

References

Abdulkadir M, Hübel C, Herle M, Loos RJF, Breen G, Bulik CM, Micali N (2022) Eating disorder symptoms and their associations with anthropometric and psychiatric polygenic scores. Eur Eat Disord Rev 30(3):221–236 [PubMed: 35178801]

Akaike H (1987) Factor analysis and AIC. Psychometrika 52:317–332

Baker JH, Brosof LC, Munn-Chernoff MA, Lichtenstein P, Larsson H, Maes HH, Kendler KS (2018) Associations Between Alcohol Involvement and Drive for Thinness and Body Dissatisfaction in Adolescent Twins: A Bivariate Twin Study. Alcohol Clin Exp Res 42(11):2214–2223 [PubMed: 30252141]

Baker JH, Higgins Neyland MK, Thornton LM, Runfola CD, Larsson H, Lichtenstein P, Bulik C (2019) Body dissatisfaction in adolescent boys. Dev Psychol 55(7):1566–1578 [PubMed: 30985163]

Baker JH, Johnson NK, Munn-Chernoff MA, Lichtenstein P, Larsson H, Maes HH, Kendler KS (2018) Illicit Drug Use, Cigarette Smoking, and Eating Disorder Symptoms: Associations in an Adolescent Twin Sample. J Stud Alcohol Drugs 79(5):720–724 [PubMed: 30422785]

Baker JH, Maes HH, Lissner L, Aggen SH, Lichtenstein P, Kendler KS (2009) Genetic risk factors for disordered eating in adolescent males and females. J Abnorm Psychol 118(3):576–586 [PubMed: 19685954]

Baker JH, Munn-Chernoff MA, Lichtenstein P, Larsson H, Maes H, Kendler KS (2017) Shared familial risk between bulimic symptoms and alcohol involvement during adolescence. J Abnorm Psychol 126(5):506–518 [PubMed: 28691841]

Biederman J, Ball SW, Monuteaux MC, Surman CB, Johnson JL, Zeitlin S (2007) Are girls with ADHD at risk for eating disorders? Results from a controlled, five-year prospective study. J Dev Behav Pediatr 28(4):302–307 [PubMed: 17700082]

Bleck J, DeBate RD (2013) Exploring the co-morbidity of attention-deficit/hyperactivity disorder with eating disorders and disordered eating behaviors in a nationally representative community-based sample. Eat Behav 14(3):390–393 [PubMed: 23910787]

Bleck J, DeBate RD, Olivardia R (2015) The Comorbidity of ADHD and Eating Disorders in a Nationally Representative Sample. J Behav Health Serv Res 42(4):437–451 [PubMed: 25007864]

Blinder BJ, Cumella EJ, Sanathara VA (2006) Psychiatric comorbidities of female inpatients with eating disorders. Psychosom Med 68(3):454–462 [PubMed: 16738079]

Conners CK, Wells KC, Parker JD, Sitarenios G, Diamond JM, Powell JW (1997) A new self-report scale for assessment of adolescent psychopathology: factor structure, reliability, validity, and diagnostic sensitivity. J Abnorm Child Psychol 25(6):487–497 [PubMed: 9468109]

Cortese S, Bernardina BD, Mouren MC (2007) Attention-deficit/hyperactivity disorder (ADHD) and binge eating. Nutr Rev 65(9):404–411 [PubMed: 17958207]

Cortese S, Moreira-Maia CR, St Fleur D, Morcillo-Peñalver C, Rohde LA, Faraone SV (2016) Association Between ADHD and Obesity: A Systematic Review and Meta-Analysis. Am J Psychiatry 173(1):34–43 [PubMed: 26315982]

de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J (2007) Development of a WHO growth reference for school-aged children and adolescents. Bull World Health Organ 85(9):660–667 [PubMed: 18026621]

Demontis D, Walters RK, Martin J, Mattheisen M, Als TD, Agerbo E, Baldursson G, Belliveau R, Bybjerg-Grauholm J, Baekvad-Hansen M, Cerrato F, Chambert K, Churchhouse C, Dumont A, Eriksson N, Gandal M, Goldstein JI, Grasby KL, Grove J, Gudmundsson OO, Hansen CS, Hauberg ME, Hollegaard MV, Howrigan DP, Huang H, Maller JB, Martin AR, Martin NG, Moran J, Pallesen J, Palmer DS, Pedersen CB, Pedersen MG, Poterba T, Poulsen JB, Ripke S, Robinson EB, Satterstrom FK, Stefansson H, Stevens C, Turley P, Walters GB, Won H, Wright MJ, Andreassen OA, Asherson P, Burton CL, Boomsma DI, Cormand B, Dalsgaard S, Franke B, Gelernter J, Geschwind D, Hakonarson H, Haavik J, Kranzler HR, Kuntsi J, Langley K, Lesch KP, Middeldorp C, Reif A, Rohde LA, Roussos P, Schachar R, Sklar P, Sonuga-Barke EJS, Sullivan PF, Thapar A, Tung JY, Waldman ID, Medland SE, Stefansson K, Nordentoft M, Hougaard DM, Werge T, Mors O, Mortensen PB, Daly MJ, Faraone SV, Borglum AD, Neale BM (2019) Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. Nat Genet 51(1):63–75 [PubMed: 30478444]

Ekman JT, Gustafsson PA (2000) Stimulants in AD/HD, a controversial treatment only in Sweden? Eur Child Adolesc Psychiatry 9(4):312–313 [PubMed: 11202108]

Fairweather-Schmidt AK, Wade TD (2015) Changes in genetic and environmental influences on disordered eating between early and late adolescence: a longitudinal twin study. Psychol Med 45(15):3249–3258 [PubMed: 26134758]

Fayyad J, Sampson NA, Hwang I, Adamowski T, Aguilar-Gaxiola S, Al-Hamzawi A, Andrade LH, Borges G, de Girolamo G, Florescu S, Gureje O, Haro JM, Hu C, Karam EG, Lee S, Navarro-Mateu F, O'Neill S, Pennell BE, Piazza M, Posada-Villa J, Ten Have M, Torres Y, Xavier M, Zaslavsky AM, Kessler RC (2017) The descriptive epidemiology of DSM-IV Adult ADHD in the World Health Organization World Mental Health Surveys. Atten Defic Hyperact Disord 9(1):47–65 [PubMed: 27866355]

Ferguson CJ, Munoz ME, Winegard B, Winegard B (2012) The influence of heritability, neuroticism, maternal warmth and media use on disordered eating behaviors: a prospective analysis of twins. Psychiatr Q 83(3):353–360 [PubMed: 22278805]

Fleming J, Levy L (2002) Eating disorders in women with AD/HD. In: Quinn PO, Nadeau KG (eds) Gender issues and AD/HD: research, diagnosis, and treatment. Advantage Books, Silver Spring, MD, pp 411

Freitag CM, Rohde LA, Lempp T, Romanos M (2010) Phenotypic and measurement influences on heritability estimates in childhood ADHD. Eur Child Adolesc Psychiatry 19(3):311–323 [PubMed: 20213230]

Garner D (1991) Eating Disorders Inventory-2: Professional Manual. Psychological Assessment Resources, Odessa, FL

Hübel C, Abdulkadir M, Herle M, Loos RJF, Breen G, Bulik CM, Micali N (2021) One size does not fit all. Genomics differentiates among anorexia nervosa, bulimia nervosa, and binge-eating disorder. Int J Eat Disord 54(5):785–793 [PubMed: 33644868]

Hübel C, Gaspar HA, Coleman JRI, Hanscombe KB, Purves K, Prokopenko I, Graff M, Ngwa JS, Workalemahu T, O'Reilly PF, Bulik CM, Breen G (2019) Genetic correlations of psychiatric traits with body composition and glycemic traits are sex- and age-dependent. Nat Commun 10(1):5765 [PubMed: 31852892]

Klump KL (2013) Puberty as a critical risk period for eating disorders: a review of human and animal studies. Horm Behav 64(2):399–410 [PubMed: 23998681]

Klump KL, Culbert KM, Slane JD, Burt SA, Sisk CL, Nigg JT (2012) The effects of puberty on genetic risk for disordered eating: evidence for a sex difference. Psychol Med 42(3):627–637 [PubMed: 21854699]

Koren R, Munn-Chernoff MA, Duncan AE, Bucholz KK, Madden PA, Heath AC, Agrawal A (2014) Is the relationship between binge eating episodes and personality attributable to genetic factors? Twin Res Hum Genet 17(2):65–71 [PubMed: 24423627]

Larsen JT, Bulik CM, Thornton LM, Koch SV, Petersen L (2021) Prenatal and perinatal factors and risk of eating disorders. Psychol Med 51(5):870–880 [PubMed: 31910913]

Lichtenstein P, Tuvblad C, Larsson H, Carlstrom E (2007) The Swedish Twin study of CHild and Adolescent Development: the TCHAD-study. Twin Res Hum Genet 10(1):67–73

Martin J, Walters RK, Demontis D, Mattheisen M, Lee SH, Robinson E, Brikell I, Ghirardi L, Larsson H, Lichtenstein P, Eriksson N; 23andMe Research Team; Psychiatric Genomics Consortium: ADHD Subgroup; iPSYCH–Broad ADHD Workgroup, Werge T, Mortensen PB, Pedersen MG, Mors O, Nordentoft M, Hougaard DM, Bybjerg-Grauholm J, Wray NR, Franke B, Faraone SV, O'Donovan MC, Thapar A, Børglum AD, Neale BM (2018) A Genetic Investigation of Sex Bias in the Prevalence of Attention-Deficit/Hyperactivity Disorder. Biol Psychiatry 83(12):1044–1053 [PubMed: 29325848]

Martin N, Scourfield J, McGuffin P (2002) Observer effects and heritability of childhood attention-deficit hyperactivity disorder symptoms. Br J Psychiatry 180260–265

Mikami AY, Hinshaw SP, Arnold LE, Hoza B, Hechtman L, Newcorn JH, Abikoff HB (2010) Bulimia nervosa symptoms in the multimodal treatment study of children with ADHD. Int J Eat Disord 43(3):248–259 [PubMed: 19378318]

Mikami AY, Hinshaw SP, Patterson KA, Lee JC (2008) Eating pathology among adolescent girls with attention-deficit/hyperactivity disorder. J Abnorm Psychol 117(1):225–235 [PubMed: 18266500]

Munn MA, Stallings MC, Rhee SH, Sobik LE, Corley RP, Rhea SA, Hewitt JK (2010) Bivariate analysis of disordered eating characteristics in adolescence and young adulthood. Int J Eat Disord 43(8):751–761 [PubMed: 20957703]

Nadder TS, Silberg JL, Rutter M, Maes HH, Eaves LJ (2001) Comparison of multiple measures of ADHD symptomatology: a multivariate genetic analysis. J Child Psychol Psychiatry 42(4):475–486 [PubMed: 11383963]

Nazar BP, Bernardes C, Peachey G, Sergeant J, Mattos P, Treasure J (2016) The risk of eating disorders comorbid with attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. Int J Eat Disord 49(12):1045–1057 [PubMed: 27859581]

Neale M (1991) Statistical Modelling with Mx. Box 980710, Richmond VA 23298: Department of Psychiatry, Richmond, VA

Nevonen L, Clinton D, Norring C (2006) Validating the EDI-2 in three Swedish female samples: eating disorders patients, psychiatric outpatients and normal controls. Nord J Psychiatry 60(1):44–50 [PubMed: 16500799]

Nigg JT, Johnstone JM, Musser ED, Long HG, Willoughby MT, Shannon J (2016) Attention-deficit/hyperactivity disorder (ADHD) and being overweight/obesity: New data and meta-analysis. Clin Psychol Rev 4367–79

Norring C, Sohlberg S (1988) Eating Disorder Inventory in Sweden: description, cross-cultural comparison, and clinical utility. Acta Psychiatr Scand 78(5):567–575 [PubMed: 3232534]

Quinn P, Wigal S (2004) Perceptions of girls and ADHD: results from a national survey. MedGenMed 6(2):2

Quinn PO, Madhoo M (2014) A review of attention-deficit/hyperactivity disorder in women and girls: uncovering this hidden diagnosis. Prim Care Companion CNS Disord 16(3): PCC.13r01596

Reichborn-Kjennerud T, Bulik CM, Kendler KS, Røysamb E, Maes H, Tambs K, Harris JR (2003) Gender differences in binge-eating: a population-based twin study. Acta Psychiatr Scand 108(3):196–202 [PubMed: 12890274]

Reichborn-Kjennerud T, Bulik CM, Tambs K, Harris JR (2004) Genetic and environmental influences on binge eating in the absence of compensatory behaviors: a population-based twin study. Int J Eat Disord 36(3):307–314 [PubMed: 15478129]

Reinblatt SP, Leoutsakos JM, Mahone EM, Forrester S, Wilcox HC, Riddle MA (2014) Association between binge eating and attention-deficit/hyperactivity disorder in two pediatric community mental health clinics. Int J Eat Disord 48(5):505–511 [PubMed: 25130278]

Sayal K, Prasad V, Daley D, Ford T, Coghill D (2018) ADHD in children and young people: prevalence, care pathways, and service provision. Lancet Psychiatry 5(2):175–186 [PubMed: 29033005]

Schaumberg K, Jangmo A, Thornton LM, Birgegård A, Almqvist C, Norring C, Larsson H, Bulik CM (2019) Patterns of diagnostic transition in eating disorders: a longitudinal population study in Sweden. Psychol Med 49(5):819–827 [PubMed: 29911514]

Schwarz G (1978) Estimating the dimension of a model. Ann Stat 6(2):461–464

Skogli EW, Teicher MH, Andersen PN, Hovik KT, Øie M (2013) ADHD in girls and boys--gender differences in co-existing symptoms and executive function measures. BMC Psychiatry 13298

Sonneville KR, Calzo JP, Horton NJ, Field AE, Crosby RD, Solmi F, Micali N (2015) Childhood hyperactivity/inattention and eating disturbances predict binge eating in adolescence. Psychol Med 45(12):2511–2520 [PubMed: 26098685]

Stice E, Davis K, Miller NP, Marti CN (2008) Fasting increases risk for onset of binge eating and bulimic pathology: a 5-year prospective study. J Abnorm Psychol 117(4):941–946 [PubMed: 19025239]

Testa G, Baenas I, Vintró-Alcaraz C, Granero R, Agüera Z, Sánchez I, Riesco N, Jiménez-Murcia S, Fernández-Aranda F (2020) Does ADHD Symptomatology Influence Treatment Outcome and Dropout Risk in Eating Disorders? A Longitudinal Study. J Clin Med 20;9(7):2305

Tuvblad C, Grann M, Lichtenstein P (2006) Heritability for adolescent antisocial behavior differs with socioeconomic status: gene-environment interaction. J Child Psychol Psychiatry 47(7):734–743 [PubMed: 16790008]

Wade T, Martin NG, Neale MC, Tiggemann M, Treloar SA, Bucholz KK, Madden PA, Heath AC (1999) The structure of genetic and environmental risk factors for three measures of disordered eating. Psychol Med 29(4):925–934 [PubMed: 10473319]

Waring ME, Lapane KL (2008) Overweight in children and adolescents in relation to attention-deficit/hyperactivity disorder: results from a national sample. Pediatrics 122(1):e1–6 [PubMed: 18595954]

Wentz E, Lacey JH, Waller G, Rastam M, Turk J, Gillberg C (2005) Childhood onset neuropsychiatric disorders in adult eating disorder patients. A pilot study. Eur Child Adolesc Psychiatry 14(8):431–437 [PubMed: 16341499]

Werner B, Bodin L (2006) Growth from birth to age 19 for children in Sweden born in 1981: descriptive values. Acta Paediatr 95(5):600–613 [PubMed: 16825142]

Westerberg-Jacobson J, Edlund B, Ghaderi A (2010) Risk and protective factors for disturbed eating: a 7-year longitudinal study of eating attitudes and psychological factors in adolescent girls and their parents. Eat Weight Disord 15(4):e208–218 [PubMed: 21406944]

Wonderlich SA, Connolly KM, Stice E (2004) Impulsivity as a risk factor for eating disorder behavior: assessment implications with adolescents. Int J Eat Disord 36(2):172–182 [PubMed: 15282687]

Yao S, Kuja-Halkola R, Martin J, Lu Y, Lichtenstein P, Norring C, Birgegård A, Yilmaz Z, Hübel C, Watson H, Baker J, Almqvist C, Thornton LM, Magnusson PK, Bulik CM, Larsson H (2019) Associations Between Attention-Deficit/Hyperactivity Disorder and Various Eating Disorders: A Swedish Nationwide Population Study Using Multiple Genetically Informative Approaches. Biol Psychiatry 86(8):577–586 [PubMed: 31301758]

Yates WR, Lund BC, Johnson C, Mitchell J, McKee P (2009) Attention-deficit hyperactivity symptoms and disorder in eating disorder inpatients. Int J Eat Disord 42(4):375–378 [PubMed: 19040267]

Yilmaz Z, Hardaway JA, Bulik CM (2015) Genetics and epigenetics of eating disorders. Adv Genomics Genet 5:131–150 [PubMed: 27013903]

Yilmaz Z, Javaras KN, Baker JH, Thornton LM, Lichtenstein P, Bulik CM, Larsson H (2017) Association Between Childhood to Adolescent Attention Deficit/Hyperactivity Disorder Symptom Trajectories and Late Adolescent Disordered Eating. J Adolesc Health 61(2):140–146 [PubMed: 28734322]

Yilmaz Z, Kaplan AS, Zai CC, Levitan RD, Kennedy JL (2011) COMT Val158Met variant and functional haplotypes associated with childhood ADHD history in women with bulimia nervosa. Prog Neuropsychopharmacol Biol Psychiatry 35(4):948–952 [PubMed: 21300128]

Yoshimasu K, Barbaresi WJ, Colligan RC, Voigt RG, Killian JM, Weaver AL, Katusic SK (2012) Childhood ADHD is strongly associated with a broad range of psychiatric disorders during adolescence: a population-based birth cohort study. Journal of Child Psychology and Psychiatry 53(10):1036–1043 [PubMed: 22647074]

Table 1.

Mean (SD) Scores for ADHD and Disordered Eating Symptoms

	Girls (*n*=744–789)	**Boys (*n*=694–726)**
CASS Conduct Problems	2.00 (2.20)	2.25 (2.60)
CASS Cognitive/Inattention Problems	4.00 (3.30)	3.32 (3.10)
CASS Hyperactivity	4.00 (3.50)	3.55 (3.42)
EDI Drive for Thinness	2.90 (4.17)	.90 (1.74)
EDI Bulimia	.51 (1.30)	.50 (1.20)
EDI Body Dissatisfaction	5.60 (6.00)	2.10 (3.60)
Body mass index	20.50 (3.00)	20.90 (2.80)

Note. Sample sizes differ across analyses due to missing data.

CASS = Conners-Wells Adolescent Self-Report Scale: Short Form; EDI = Eating Disorder Inventory-2; SD = Standard Deviation.

Table 2.

ADHD and Disordered Eating Symptoms Phenotypic Correlations in Girls and Boys

	Drive for Thinness		Bulimia		Body Dissatisfaction	
	Girls (*n*=777)	**Boys (*n*=723)**	**Girls (*n*=775)**	**Boys (*n*=719)**	**Girls (*n*=777)**	**Boys (*n*=725)**
Conduct Problems	.14**	.04 *	.24**	.25**	.23**	.20**
Cognitive/Inattention Problems	.13**	.05	.30**	.22**	.29**	.22**
Hyperactivity	.10**	.06*	.19**	.22**	.16**	.11**

Notes. Fischer r-to-z transformation significant at $p < .05$ (two-tailed) indicating significant difference in correlation between girls and boys. Subscale scores log-transformed prior to analysis. Sample sizes differ across analyses due to missing data.

* $p < .05$;

** $p < .01$

Table 3.

Twin Correlations (95% Confidence Intervals) Between ADHD Symptoms and Disordered Eating

	Girls		Boys	
	MZ *n*=440–444	**DZ *n*=310–326**	**MZ *n*=400–404**	**DZ n=289–292**
Cross-Twin, Within-Trait Correlations				
Conduct Problems	.57 (.50, .64)	.24 (.13, .36)	.42 (.34, .50)	.24 (.13, .34)
Cognitive/Inattention Problems	.48 (.40, .56)	.24 (.13, .35)	.51 (.43, .60)	.10 (.01, .20)
Hyperactivity	.53 (.44, .62)	.14 (.03, .25)	.48 (.40, .56)	.11 (−.01, .22)
Bulimia	.31 (.18, .43)	.22 (.10, .35)	.36 (.23, .50)	.15 (.01, .29)
Body Dissatisfaction	.64 (.60, .70)	.33 (.23, .43)	.44 (.34, .53)	.26 (.13, .40)
Cross-Twin Cross-Trait Correlations				
Conduct Problems-Bulimia	.14 (.04, .25)	.21 (.10, .32)	.20 (.10, .30)	.09 (−.02, .22)
Cognitive/Inattention Problems-Bulimia	.20 (.08, .28)	.16 (.04, .28)	.15 (.04, .27)	.10 (−.02, .22)
Hyperactivity-Bulimia	--	--	.22 (.11, .32)	.08 (−.04, .19)
Conduct Problems-Body Dissatisfaction	.14 (.05, .24)	.14 (.04, .25)	--	--
Cognitive/Inattention Problems-Body Dissatisfaction	.18 (.08, .27)	.09 (−.02, .20)	.13 (.03, .23)	.09 (−.03, .19)

Note. Sample sizes differ across analyses due to missing data. MZ = Monozygotic; DZ = Dizygotic.

Table 4.

Bivariate Twin Model Fitting Results between ADHD Symptoms and Disordered Eating

Model	−2lnL	df	χ^2 diff (p)	AIC	BIC
Girls					
Conduct Problems-Bulimia					
ACE	2270.04	1358	--	−446.00	−2836.70
AE	**2271.05**	**1361**	**1.01 (.80)**	**−451.00**	**−2845.00**
CE	2290.12	1361	20.00 (<.01)	−431.90	−2835.41
Cognitive/Inattention Problems-Bulimia					
ACE	4335.258	1358	--	1619.258	−1804.10
AE	**4335.287**	**1361**	**.029 (.99)**	**1613.287**	**−1813.00**
CE	4349.995	1361	14.736 (.002)	1627.995	−1805.50
Conduct Problems-Body Dissatisfaction					
ACE	3189.00	1360	--	470.00	−2383.00
AE	**3190.20**	**1363**	**1.05 (.80)**	**464.20**	**−2391.23**
CE	3214.20	1363	25.10 (<.01)	488.20	−2380.00
Cognitive/Inattention Problems-Body Dissatisfaction					
ACE	5250.80	1360	--	2530.80	−1352.14
AE	**5252.60**	**1363**	**2.00 (.61)**	**2526.60**	**−1360.01**
CE	5270.33	1363	20.00 (<.01)	2544.00	−1351.15
Boys					
Conduct Problems-Bulimia					
ACE	2129.589	1272	--	−414.411	−2612.00
AE	**2129.647**	**1275**	**.059 (.99)**	**−420.353**	**−2620.40**
CE	2136.953	1275	7.365 (.06)	−413.047	−2617.0
Cognitive/Inattention Problems-Bulimia					
ACE	3980.00	1272	--	1434.70	−1687.22
AE	**3980.00**	**1275**	**0 (0)**	**1428.70**	**−1696.00**
CE	4000.00	1275	18.00 (<.01)	1446.33	−1687.10
Hyperactivity-Bulimia					
ACE	4107.50	1274	--	1560.00	−1629.00
AE	**4107.50**	**1277**	**0 (0)**	**1553.50**	**−1637.30**
CE	4122.44	1277	15.00 (<.01)	1570.00	−1630.00
Cognitive/Inattention Problems-Body Dissatisfaction					
ACE	4762.80	1277	--	2208.80	−1309.63
AE	**4762.81**	**1277**	**.06 (.99)**	**2202.81**	**−1318.30**
CE	4781.34	1280	19.00 (<.01)	2221.34	−1310.0

Best-fit model shown in bold. −2lnL = twice the negative log-likelihood; df = degrees of freedom; χ^2 diff (p) = χ^2 difference from full model; AIC = Akaike's Information Criterion; BIC = Bayesian Information Criterion; A = additive genetic; C = common environment; E = unique environment.

Table 5.

Bivariate Model Parameter Estimates, Correlations, and Confidence Intervals for Full Model (ACE Model) Best-fit Models (AE Model [*]) between and ADHD and Disordered Eating Symptoms Adjusted for Body Mass Index

	Girls								
	ADHD Symptom			**Disordered Eating**			**Correlations**		
	a^2	**c^2**	**e^2**	**a^2**	**c^2**	**e^2**	**r_a (95% CI)**	**r_c (95% CI)**	**r_e (95% CI)**
Conduct Problems-Bulimia									
Full Model	.50 (.26, .64)	.07 (0, .26)	.44 (.35, .54)	.15 (0, .38)	.14 (0, .33)	.73 (.62, .85)	.24 (−1, 1)	1 (−.61, 1)	.18 (.05, .31)
Best Fit Model	.56 (.46, .65)	--	.44 (.35, .54)	.28 (.16, .40)	--	.72 (.61, .84)	.41 (.19, .63)	--	.18 (.04, .30)
Conduct Problems-Body Dissatisfaction									
Full Model	.56 (.37, .65)	.01 (0, .16)	.43 (.35, .53)	.40 (.10, .63)	.15 (0, .43)	.45 (.37, .55)	.34 (−.10, .80)	1 (−1, 1)	.11 (−.03, .24)
Best Fit Model	.57 (.47, .65)	--	.43 (.35, .53)	.56 (.47, .64)	--	.44 (.36, .53)	.36 (.20, .50)	--	.10 (−.03, .23)
Cognitive/Inattention Problems-Bulimia									
Full Model	.48 (.27, .57)	.01 (0, .16)	.52 (.42, .63)	.25 (0, .40)	.03 (0, .30)	.71 (.60, .84)	.55 (−1, 1)	1 (−1, 1)	.17 (.04, .30)
Best Fit Model	.48 (.36, .58)	--	.52 (.42, .64)	.29 (.17, .40)	--	.71 (.60, .83)	.54 (.31, .77)	--	.17 (.04, .30)
Cognitive/Inattention Problems- Body Dissatisfaction									
Full Model	.48 (.30, .58)	.01 (0, .14)	.52 (.42, .63)	.36 (.06, .62)	.19 (0, .44)	.45 (.37, .55)	.54 (.13, 1)	−1.0 (−1, 1)	.16 (.02, .30)
Best Fit Model	.48 (.37, .58)	--	.52 (.42, .63)	.55 (.47, .64)	--	.44 (.36, .53)	.41 (.25, .56)	--	.17 (.04, .30)
	Boys								
	ADHD Symptom			**Disordered Eating**			**Correlations**		
	a^2	**c^2**	**e^2**	**a^2**	**c^2**	**e^2**	**r_a (95% CI)**	**r_c (95% CI)**	**r_e (95% CI)**
Conduct Problems-Bulimia									
Full Model	.36 (.01, .51)	.04 (0, .35)	.60 (.50, .72)	.32 (.01, .43)	.001 (0, .25)	.68 (.57, .81)	.57 (−.20, 1)	1 (−.54, 1)	−.06 (−.10, .20)

Best Fit Model	.41 (.30, .51)	--	.60 (.49, .71)	.32 (.20, .43)	--	.68 (.57, .81)	.55 (.30, .80)	--	.06 (−.07, .19)
Cognitive/Inattention Problems-Bulimia									
Full Model	.48 (.28, .58)	0 (0, .14)	.52 (.42, .64)	.32 (.01, .43)	0 (0, .30)	.68 (.57, .81)	.42 (−.10, 1)	.95 (−1, 1)	.10 (−.04, .23)
Best Fit Model	.48 (.36, .58)	--	.52 (.42, .64)	.32 (.19, .43)	--	.68 (.57, .81)	.42 (.18, .66)	--	.10 (−.04, .23)
Hyperactivity-Bulimia									
Full Model	.46 (.24, .56)	0 (0, .18)	.54 (.44, .65)	.31 (.01, .43)	0 (0, .26)	.67 (.57, .82)	.53 (.16, 1)	−.78 (−1, 1)	.02 (−.11, .15)
Best Fit Model	.46 (.35, .56)	--	.54 (.44, .65)	.31 (.19, .43)	--	.69 (.57, .81)	.53 (.30,.80)	--	.02 (−.11, .15)
Cognitive/Inattention Problems-Body Dissatisfaction									
Full Model	.47 (.28, .58)	.01 (0, .15)	.52 (.42, .64)	.38 (.01, .52)	.03 (0, .34)	.58 (.47, .50)	.34 (−21, 1)	1 (−1, 1)	.13 (−.01, .29
Best Fit Model	.48 (.36, .58)	--	.52 (.42, .54)	.42 (.31, .53)	--	.58 (.47, .70)	.35 (.14, .54)	--	.13 (−.01, .25)

a^2 = heritability; c^2 = common environmental estimate; e^2 = unique environmental estimate; r_a = genetic correlation; r_c = common environmental correlation; r_e = unique environmental correlation.

*c^2 and r_c not shown as these were set to 0 in the best fit models.