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Shared Genetic Factors Contributing to the Overlap between Attention-Deficit/Hyperactivity Disorder Symptoms and Overweight/Obesity in Swedish Adolescent Girls and Boys

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Abstract

Attention-deficit/hyperactivity disorder (ADHD) and obesity are positively associated, with increasing evidence that they share genetic risk factors. Our aim was to examine whether these findings apply to both types of ADHD symptoms for female and male adolescents. We used data from 791 girl and 735 boy twins ages 16–17 years to examine sex-specific phenotypic correlations between the presence of ADHD symptoms and overweight/obese status. For correlations

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Ethical Standards:
The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975.

exceeding .20, we then fit bivariate twin models to estimate the genetic and environmental correlations between the presence of ADHD symptoms and overweight/obese status. ADHD symptoms and height/weight were parent- and self-reported, respectively. Phenotypic correlations were .30 (girls) and .08 (boys) for inattention and overweight/obese status and .23 (girls) and .14 (boys) for hyperactivity/impulsivity and overweight/obese status. In girls, both types of ADHD symptoms and overweight/obese status were highly heritable, with unique environmental effects comprising the remaining variance. Furthermore, shared genetic effects explained most of the phenotypic correlations, in girls. Results suggest that the positive association of both types of ADHD symptoms with obesity may be stronger in girls than boys. Further, in girls, these associations may stem primarily from shared genetic factors.

Keywords

Obesity; Attention-deficit/hyperactivity disorder; Inattention; Hyperactivity/Impulsivity; Genetics

Attention-deficit/hyperactivity disorder (ADHD)—characterized by pervasive and impairing inattention, hyperactivity, and impulsivity—affects 3.4% of children and adolescents worldwide (Polanczyk et al., 2015). Individuals with ADHD are also at greater risk for certain conditions, including obesity (Instanes et al., 2018), which has become increasingly prevalent among children and adolescents globally (Ng et al., 2014). Specifically, in meta-analyses, children and adolescents with ADHD (based on an actual or self-reported medical record diagnosis, a research diagnosis, or scores on validated ADHD rating scales) had 1.2 times the odds of obese status (Cortese et al., 2016) and 1.13 times the odds of overweight and/or obese status (Nigg et al., 2016), compared to children and adolescents without ADHD; both odds ratios (i.e., 1.2 and 1.13) were statistically significant. Notably, in the latter meta-analysis, when the association between ADHD and overweight/obese status was calculated separately for adolescents and for younger children, the association in adolescents was qualitatively larger than the association in children (Nigg et al., 2016). This finding converges with results from longitudinal studies showing that body mass index (BMI) trajectories for individuals with ADHD diverge upward from the trajectories of those without ADHD during the adolescent years (Porter et al., 2021; Schwartz et al., 2014). Although the meta-analytic associations (between ADHD and overweight/obese status) did not differ significantly by sex in analyses that combined studies conducted in all age groups (Cortese et al., 2016; Nigg et al., 2016), some individual studies suggest that, among youth, the association between ADHD and high BMI may be stronger in females (Aguirre Castaneda et al., 2016; Byrd et al., 2013; Kase et al., 2021; Kim et al., 2011; Nigg et al., 2016; van Egmond-Fröhlich et al., 2012). Further, meta-analytic results suggest that the positive association between ADHD and obesity pertains only to individuals not taking stimulant medications (Cortese et al., 2016). Finally, although the meta-analyses did not examine whether the association between ADHD and high BMI differed for different ADHD subtypes (i.e., inattention (IN), or hyperactivity/impulsivity (HI)), a recent study conducted across adolescence and young adulthood found a significant association between HI symptoms and BMI, but not IN symptoms and BMI (Kase et al., 2021).

In addition to evidence that ADHD and obesity co-occur within individuals, evidence also points to co-aggregation within families, with ADHD in a proband increasing the likelihood of high BMI in family members (Chen et al., 2017, 2019; Geuijen et al., 2019). The presence of co-aggregation suggests a role for familial influences, specifically genetic and common environmental factors. Regarding genetics, both ADHD and obesity are substantially influenced by genetic factors. *Diagnosed* ADHD is highly heritable (between .70 and .80) (Faraone et al., 2005; Faraone & Larsson, 2019). Further, ADHD *symptoms* are moderately to highly heritable in adolescence (Merwood et al., 2013), with higher estimates of heritability typically observed when the same person (e.g., a parent) rates symptoms for both twins (Brikell et al., 2015; Freitag et al., 2010), potentially because having a single rater reduces discordance in twins' ratings due to differences in rater effects between informants (Brikell et al., 2015). Additionally, heritability is high for both subtypes of ADHD symptoms (i.e., IN and HI) in childhood and adolescence (Larsson et al., 2011), although the genetic influences on the two types of symptoms appear to overlap only partially (Freitag et al., 2010). BMI is also moderately to highly heritable in childhood and adolescence, with twin-based heritability estimates ranging from approximately .50 to .90 (Silventoinen et al., 2010). Estimates of BMI's heritability tend to be higher in studies that use staff-measured (rather than self-reported) BMI, likely due to reduced measurement error for staff-measured BMI (Elks et al., 2012). BMI heritability estimates also tend to be higher in studies conducted in adolescents or in countries with high gross domestic product per capita (Elks et al., 2012; Min et al., 2013), possibly because of developmental and environmental differences, respectively, in the expression of adiposity-related genes (Min et al., 2013). Regarding common environment, childhood ADHD symptoms and obesity share some environmental risk factors (e.g., low parental socioeconomic status, maternal smoking during pregnancy), which may account for their co-occurrence (Donnchadha et al., 2020; Pauli-Pott et al., 2017) and some instances of co-aggregation. Notably, some of these risk factors (e.g., maternal smoking during pregnancy) may share overlapping genetic influences with ADHD and BMI (Obel et al., 2011; Skoglund et al., 2014).

Bivariate behavioral genetic models can be used to elucidate the extent to which genetic and environmental influences on ADHD and obesity overlap. To our knowledge, two studies have conducted this type of analysis (Chen et al., 2019; Do et al., 2019). Do and colleagues (2019) fitted behavioral genetic models to data on retrospectively-reported childhood ADHD symptoms and adolescent through young adult BMI, from twins and siblings in a nationally-representative longitudinal study conducted in the United States. In females, the best-fitting model included shared genetic influences, but not shared unique environmental influences, between overall ADHD symptoms and BMI; in this model, genetic correlations (i.e., correlations between the genetic influences on ADHD symptoms and BMI) ranged from .20 to .24, depending on the BMI timepoint. In contrast, in males, the best-fitting model included shared unique environmental influences, but not shared genetic influences, between ADHD symptoms and BMI; in this model, environmental correlations (i.e., correlations between the environmental influences on ADHD symptoms and BMI) ranged from .16 to .19, depending on the BMI timepoint. Do and colleagues (2019) also explored whether these results held when models were fitted to IN and HI symptoms separately. For IN, results were similar to those for overall ADHD symptoms, with genetic

influences playing a (qualitatively) greater role in the overlap between symptoms and BMI in females: genetic correlations ranged from .10 in adolescence to .25 in adulthood for females, but from 0 in adolescence to .07 in adulthood for males. In contrast, there was no evidence of sex differences in the genetic overlap between HI symptoms and BMI, with genetic correlations ranging from .08 in adolescence to .25 in adulthood for both females and males. In another study, Chen and colleagues (2019) fitted behavioral genetic models to data on ADHD and (clinical) obesity for full and half siblings, using data from the Swedish national registers. Results suggest that overlapping genetic influences contribute to the overlap between ADHD and obesity, with a (significant) genetic correlation of .30 in both sexes combined. The correlations for common and unique environmental influences on ADHD and obesity were .19 and −.03, respectively, and not significant. Taken together, these studies provide evidence that the genetic influences on ADHD and obesity overlap, at least in females. However, the retrospective-reporting of ADHD symptoms in Do et al. (2019) and the clinical ascertainment of ADHD and obesity cases in Chen et al. (2019) are well-acknowledged sources of bias that may impact results, underscoring the importance of independent replications that address these methodological limitations. Further, given evidence that the use of stimulant medications attenuates the association between ADHD and obesity (Cortese et al., 2016; Nigg et al., 2016), it is important to investigate genetic and environmental influences on this association in a sample naïve to prescription stimulants.

Thus, we conducted bivariate behavioral genetic analyses to examine the relative contributions of shared genetic and environmental influences on ADHD symptoms and overweight/obese status, using data from a sample of Swedish adolescent twins. Importantly, ADHD symptoms were reported close to the time of their occurrence, information on BMI was available for the whole sample (i.e., rather than only for those who received a clinical diagnosis of obesity), and the sample was largely naïve to prescription stimulant use given the extremely low prevalence of prescription stimulants in Sweden at the relevant time (Ekman & Gustafsson, 2000). Given evidence that the strength of the association between ADHD symptoms and BMI or obesity may differ by symptom type (Kase et al., 2021) and sex (Aguirre Castaneda et al., 2016; Byrd et al., 2013; Kase et al., 2021; Kim et al., 2011; Nigg et al., 2016; van Egmond-Fröhlich et al., 2012), as well as prior research suggesting that the degree of genetic overlap between ADHD symptoms and BMI may differ for females and males depending on ADHD symptom type (Do et al., 2019), we conducted separate analyses for IN and HI symptoms, and analyses were stratified by sex.

Materials and Methods

Participants

Our sample included participants from the Swedish Twin Study of CHild and Adolescent Development (TCHAD), which includes twins born between May 1985 and December 1986 in Sweden (Lichtenstein et al., 2007). Twins and their parents were identified via the Swedish Medical Birth Register (Cnattingius et al., 1990) and invited to participate by completing parent and self-report questionnaires at four time points: Wave 1 (ages 8–9), Wave 2 (ages 13–14), Wave 3 (ages 16–17), and Wave 4 (ages 18–19).

For this study, we only used Wave 3 data (ages 16–17) because of evidence that the association between ADHD and obesity strengthens with age, emerging only in late adolescence in some samples (Nigg et al., 2016). Although participants were slightly older at Wave 4, there were fewer participants available for analysis, a significant limitation given the sample sizes required for behavioral genetic modelling, especially for categorical phenotypes. At Wave 3, 74% of the twins' parents and 82% of the twins responded to the questionnaire (Lichtenstein et al., 2007).

Our sample included 791 girls and 735 boys with known zygosity and at least some relevant variables (i.e., ADHD symptoms and BMI) observed. These participants comprised 389 complete and 13 incomplete female twins pairs and 357 complete and 21 incomplete male twin pairs (see Table 1a for the number of complete and incomplete pairs by sex and zygosity). Opposite sex dizygotic twin pairs were not included because analyses were stratified by sex (see below).

Consent was obtained from all twins and their parents who participated. Ethical approval for each wave was provided by the Ethics Committee at Karolinska Institutet in Stockholm, Sweden, and the current study was approved by the University of North Carolina Institutional Review Board.

Measures

Zygosity.—Zygosity was determined by an algorithm derived from discriminant analyses of responses to zygosity questionnaires for 106 same-sex twin pairs with known zygosity (i.e., determined based on 16 DNA polymorphisms) (Lichtenstein et al., 2007). In instances where responses yielded discordant classifications (N=86 in the full TCHAD sample), zygosity was set to unknown, and the twin pair was not included in analyses.

ADHD.—Parents completed a checklist assessing the presence or absence of 14 ADHD symptoms in their child over at least the past six months. The checklist was developed based on the ADHD criteria from the Diagnostic and Statistical Manual of Psychiatric Disorders, 4th edition (American Psychiatric Association, 1994), and details regarding its development and validation have been described elsewhere (Larsson et al., 2006, 2011). We focused on the presence of IN and HI symptoms, defined as having at least one symptom (Li et al., 2019) on the IN subscale (6-items) or HI subscale (8-items), respectively.

Overweight/Obese Status.—Height and weight were provided via twin self-report. We used age- and sex-specific means and standard deviations (SD) from a sample of Swedish children born in 1981 to remove values 6 standard deviations above and below the mean for height and log(weight) (Werner & 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). In the present analyses, we used overweight/obese status, which was defined as being at or above +1 standard deviation for the age- and sex-adjusted BMI z-scores.

Analyses

Descriptive statistics and phenotypic correlations were calculated using R Statistical Software (version 4.0.2). Phenotypic tetrachoric correlations were calculated using the corCI() function from the psych package. Bivariate twin analyses were performed only if the correlation between the presence of ADHD symptoms and overweight/obese status exceeded .20, similar to previously used approaches (Baker et al., 2018; Koren et al., 2014). Although bivariate twin models can be fitted to variables with small phenotypic correlations, there is limited value in decomposing correlations that do not statistically differ from zero into genetic and environmental components.

Bivariate twin analyses were conducted in Mx (Neale et al., 2006; Neale & Cardon, 1992). Bivariate twin analyses were performed between each ADHD variable and overweight/obese status (provided their phenotypic correlation exceeded .20), stratified by sex. Of note, we did not formally test for sex differences in bivariate twin model parameters, since prior work with this sample has demonstrated that we have insufficient power to do so (Baker et al., 2017). Cholesky decomposition was used to determine the relative magnitude of additive genetic (a), common environmental (c), and unique environmental (e) effects on the liability to the presence of ADHD symptoms, overweight/obese status, and their covariance. Additive genetic effects represent the accumulation of genetic factors influencing a phenotype, whereas common environmental effects are those factors that make members of a family similar for a given phenotype, and unique environmental effects are those factors that make members of a family dissimilar for a given phenotype. Measurement error is also included in unique environmental effects. With respect to their covariance, we estimated the genetic (r_g), common environmental (r_c), and unique environmental (r_e) correlations between the two phenotypes. The genetic correlation is the correlation between the genetic factors responsible for the presence of ADHD symptoms and the genetic factors responsible for overweight/obese status. If r_g is estimated at unity, it means that the same genetic factors contribute to the development of ADHD symptoms and overweight/obesity.

Full ACE models were fitted first. We then tested whether any parameter estimates (i.e., A, C, or E) could be set to zero and, thus, removed from the model. These submodels were tested against the full model by using minus two log-likelihood (−2LL) and standard chi-square (χ^2) difference tests to determine model fit (Neale et al., 2006). In addition, Akaike's Information Criterion (AIC) (Akaike, 1987) was used for model comparisons. A lower AIC value indicates a better and more parsimonious fitting model.

Results

Summary statistics for the analysis sample are presented in Table 1b.

Phenotypic tetrachoric correlations between ADHD variables and overweight/obese status were larger than .20 in girls (IN: .30 (95% confidence interval [CI] = .12, .46); HI: .23 (.03, .40)), but not in boys (IN: .08 (−.09, .24); HI: .14 (−.07, .31)). Thus, the following results pertain to girls only.

Model-fit indices are presented in Table 2, with parameter estimates for the full and best-fitting models shown in Table 3.

For IN and overweight/obese status, the model that only includes additive genetic and unique environmental effects provided the best fit to the data. This model (Model 2) estimated the heritability of IN and overweight/obesity to be .80 (.66, .89) and .91 (.77, .97), respectively. The remaining variance was attributable to unique environmental effects. The correlation due to additive genetic effects was .22 (−.02, .45), and the correlation due to unique environmental effects was .57 (−.02, .94). Based on these estimates, approximately 5% ($=.22^2$) and 32% ($=.57^2$) of the genetic and unique environmental effects, respectively, between IN and overweight/obesity were overlapping. Model 2's parameter estimates can also be used to calculate the proportion of the phenotypic correlation (between IN and overweight/obesity) that is due to additive genetic effects (71%) and unique environmental effects (29%) (Munn et al., 2010).

Similarly, for HI and overweight/obese status, the best-fitting model (Model 2) did not include common environmental variances or covariance. The heritability of HI was .86 (.73, .94), and unique environmental effects were estimated at .14 (.06, .27). Heritability estimates for overweight/obesity were again .91 (.77, .97), with the remainder due to unique environmental effects. The genetic and unique environmental correlations were .23 (−.02, .46) and .31 (−.48, .90), respectively, indicating that 5% of the genetic effects and 10% of the unique environmental effects between HI and overweight/obesity were overlapping. Using Model 2's parameter estimates to calculate the proportion of the phenotypic correlation (between HI and overweight/obesity) that is due to additive genetic effects and unique environmental effects yielded values of 85% and 15%, respectively.

Discussion

We examined the phenotypic overlap of IN and HI symptoms with overweight/obesity in a sample of adolescents, stratified by sex. Further, in instances where the overlap was significant, we also performed bivariate twin analyses examining the relative importance of genetic and environmental contributions to the overlap.

We found a statistically significant phenotypic association between the presence of both types of ADHD symptoms and overweight/obese status in girls, but not in boys. Although meta-analyses combining age groups have not found significant sex differences in the association between ADHD and overweight and/or obesity (Cortese et al., 2016; Nigg et al., 2016), some studies conducted in youth suggest that the association between ADHD and high BMI may be stronger or more robust in girls than in boys (Aguirre Castaneda et al., 2016; Kase et al., 2021; Kim et al., 2011; Nigg et al., 2016; van Egmond-Fröhlich et al., 2012). Given that reduced self-regulation has been described as central to the deficits observed in ADHD (Shiels & Hawk, 2010), findings of a stronger ADHD – BMI association in female youth converge with research suggesting that a positive association between reduced self-regulation and adiposity may emerge earlier in girls (Javaras et al., 2020). Several explanations have been proposed for why this may be the case, including the lower prevalence of prescription stimulant use in girls, the lower prevalence of the hyperactive/

impulsive subtype (vs. the inattentive subtype) in girls with ADHD, the greater frequency of obesity-related behaviors (e.g., screen time) in girls with ADHD, and the greater prevalence of comorbid pathology (e.g., depression, eating disorders) in girls with ADHD (Aguirre Castaneda et al., 2016; Byrd et al., 2013; Javaras et al., 2020; Kim et al., 2011; van Egmond-Fröhlich et al., 2012). Although our study was not designed to shed light on the validity of these explanations, it does suggest that sex differences in prescription stimulant use are unlikely to explain the qualitatively stronger female ADHD – BMI association in our sample, since prescription stimulant use in our sample was likely to be rare given its extremely low prevalence in Sweden during the relevant time frame (e.g., in 1998, 0.08% of children age 5–18 years in Sweden were prescribed stimulants) (Ekman & Gustafsson, 2000). Further, because we examined the association of high BMI with IN and HI symptoms separately, our findings suggest that sex differences in the different symptom types are unlikely to explain the stronger female ADHD – BMI association.

In girls, both IN and HI symptoms were highly heritable, as expected given that prior research in the current sample has found high heritability for IN and HI symptoms (Larsson et al., 2011). The broader literature suggests that ADHD symptoms are generally moderately to highly heritable (Merwood et al., 2013), with higher estimates typically found when the same person (e.g., a parent) rates symptoms for both twins (Brikell et al., 2015; Freitag et al., 2010), as was the case in the current study. In the current study, overweight/obese status was also highly heritable in girls and at the high end of prior twin-based estimates (Silventoinen et al., 2010). Although our study used self- (i.e., twin-) reported BMI, which tends to decrease estimates of heritability, heritability estimates tend to be higher for adolescents and for countries, such as Sweden, with high gross domestic product per capita (Elks et al., 2012; Min et al., 2013).

For both types of ADHD symptoms and for overweight/obese status, model fitting results suggest that non-genetic influences include unique environmental factors, but not common environmental factors. These findings are in keeping with prior twin analyses of BMI symptoms, which suggest that common environmental influences on BMI are present in early adolescence, but wane by late adolescence (Lajunen et al., 2009). With regard to ADHD symptoms, most twin analyses of ADHD have not found evidence for common environmental influences (Polderman et al., 2007).

In girls, most of the overlap between IN (or HI) and overweight/obese status was due to genetic effects, with the remainder due to unique environmental effects. The finding that genetic effects account for more of the overlap between ADHD symptoms and BMI in females is consistent with results from a prior behavioral genetics study (Do et al., 2019). The estimated genetic correlations in the current study (.22 for IN and overweight/obese status, and .23 for HI and overweight/obese status) are somewhat higher than analogous estimates (.10 and .08, respectively) from the prior study (Do et al., 2019), which may be attributed to differences in study methodology, such as differences in the reporting of ADHD symptoms in the current and prior study. Regardless of these numeric differences, results from the current study and prior behavioral genetic research (Chen et al., 2019; Do et al., 2019) converge in suggesting a modest genetic correlation between ADHD and high BMI, at least in females. Further, genetic correlations based on GWAS data were .22 for

ADHD and childhood obesity (Demontis et al., 2019) and .32 for ADHD and BMI (Mota et al., 2020), the former being especially consistent with estimated genetic correlations in the current study. Collectively, these results suggest that IN/HI symptoms and overweight/obesity may have overlapping genetic risk factors, at least in females. Although the identity of the overlapping genetic variants remains to be fully elucidated, recent research implicates dopaminergic genes – particularly the Dopamine-DARPP32 Feedback in cAMP Signaling pathway – in the genetic association between ADHD and obesity (Mota et al., 2020), consistent with earlier proposals (Liu et al., 2008).

The current study should be interpreted in light of several limitations. First, due to the low levels of ADHD pathology in TCHAD, participants were considered to have ADHD symptoms if they reported at least one ADHD symptom. Therefore, most participants with ADHD symptoms would not receive a diagnosis of ADHD, and these findings may not generalize to clinical populations. Second, as a result of the low prevalence of overweight and obesity in the Swedish population during the study period, we were unable to examine associations of ADHD symptoms with overweight and obese status separately. The relation between ADHD pathology and weight status is potentially complex, and further research is needed to determine whether genetic correlations with ADHD symptoms differ for overweight and obese status. Third, overweight/obese status was determined based on self-reported height and weight, which may have resulted in underestimates, especially in female adolescents (Bowring et al., 2012; Sherry et al., 2007). Although differential measurement error by sex could account for sex differences in the association between ADHD symptoms and overweight/obese status, a recent meta-analysis found that restricting analyses to studies using directly measured height and weight had minimal effect on estimates of the ADHD-obesity association (Cortese et al., 2016). Fourth, confidence intervals for genetic and environmental correlations between ADHD symptoms and overweight/obese status were wide and included zero. However, as noted above, estimates of the genetic correlations were generally very similar to those found in other studies, increasing confidence in these results. Fifth, AIC and the standard chi-square distribution for log-likelihood tests can lead to selection of an overly parsimonious model for some model comparisons (Javaras et al., 2009; Sullivan & Eaves, 2002). However, in the current study, use of a more lenient threshold (Javaras et al., 2009) would not have changed the conclusions regarding the best-fitting model. Sixth, as is standard in twin analyses, our study assumes random mating, equal environments, and additive genetic effects. Other designs are needed to investigate the potential role of epigenetics and gene by environment interactions in the co-occurrence between ADHD and obesity, as well as the validity of the random mating and equal environment assumptions with regards to ADHD symptoms and obesity, although existing evidence suggests that the latter assumption is a reasonable one for ADHD (Cronk et al., 2002).

These limitations aside, the present study provides additional evidence that the overlap between ADHD symptoms and overweight/obese status is stronger in female youth compared to male youth, and that the source of this overlap is primarily genetic in girls. These findings have both clinical and research implications. Clinically, obesity-related prevention efforts may need to begin earlier for girls with ADHD. In terms of research, future molecular genetic research focused on identifying additional genetic variants shared

by both ADHD and obesity may benefit from stratifying analyses by sex. Finally, future research should focus on examining genetic and environmental overlap between ADHD and obesity in samples that are larger and from countries other than the U.S. and Sweden. Further, given that the association between ADHD and obesity may not become substantial in males until later in development, future investigations should also focus on older ages.

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Data Availability:

The data for this study cannot be made publicly available because of ethical limitations on data sharing through the European Union's General Data Protection Regulation (GDPR). Application from researchers to access data from the Swedish Twin Registry is via the register website: https://ki.se/en/research/swedish-twin-registry-for-researchers.

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Table 1a:

Number of Complete and Incomplete Twin Pairs, by Sex and Zygosity

	Girls		Boys	
	Monozygotic	Dizygotic	Monozygotic	Dizygotic
Complete pairs, number	225	164	208	149
Incomplete pairs, number	3	10	13	8

Table 1b:

Analysis Sample Summary Statistics, by Sex[a,b]

	Girls (*n* = 791)	Boys (*n* = 735)
Age, years	16.7 (0.5)	16.7 (0.5)
ADHD symptoms		
At least one IN symptom	33.4%	35.3%
At least one HI symptom	21.1%	15.9%
Overweight/obese status	8.1%	11.7%

[a]Abbreviations: ADHD = attention-deficit/hyperactivity disorder; IN = inattention; HI = hyperactivity/impulsivity.

[b]Statistics presented are mean (standard deviation) for continuous variables, and percentage for categorical variables.

Table 2:

Bivariate Cholesky Decomposition Model-Fitting Results for Girls[a,b]

		Inattention & Overweight/Obesity (Girls)					
Model		Model Fit			Comparative Fit		
Number	Model	−2LL	df	AIC	$\Delta\chi^2$	Δdf	*p*-value
1	ACE-ACE r_g, r_c, r_e	1138.148	1415	−1691.852	---	---	---
2	**AE-AE r_g, r_e**	**1142.201**	**1418**	**−1693.799**	**4.053**	**3**	**.256**
3	CE-CE r_c, r_e	1154.947	1418	−1681.053	16.799	3	.001
4	E-E r_e	1272.188	1421	−1569.812	134.040	6	<.0001
		Hyperactivity/Impulsivity & Overweight/Obesity (Girls)					
Model		Model Fit			Comparative Fit		
Number	Model	−2LL	df	AIC	$\Delta\chi^2$	Δdf	*p*-value
1	ACE-ACE r_g, r_c, r_e	987.380	1418	−1848.620	---	---	---
2	**AE-AE r_g, r_e**	**987.689**	**1421**	**−1854.311**	**.309**	**3**	**.958**
3	CE-CE r_c, r_e	1006.304	1421	−1835.696	18.924	3	<.0001
4	E-E r_e	1115.017	1424	−1732.983	127.637	6	<.0001

[a] A = additive genetic effects; C = common environmental effects; E = unique environmental effects; r_g = correlation between presence of ADHD symptoms and overweight/obesity that is due to additive genetic influences; r_c = correlation between presence of ADHD symptoms and overweight/obesity that is due to common environmental influences; r_e = correlation between presence of ADHD symptoms and overweight/obesity that is due to unique environmental influences; −2LL = −2 x log likelihood; AIC = Akaike's Information Criterion; $\Delta\chi^2$ = chi-square difference test statistic.

[b] Statistics for the best-fitting model appear in bold.

Table 3:

Full and Best-Fitting Model Parameter Estimates (and 95% Confidence Intervals) for Girls[a,b]

		Inattention & Overweight/Obesity (Girls)								
Model		Inattention			Obesity			Correlations		
Number	Model	a^2	c^2	e^2	a^2	c^2	e^2	r_g	r_c	r_e
1	ACE-ACE r_g, r_c, r_e	.48 (.09, .83)	.30 (.19, .64)	.22 (.12, .37)	.45 (.04, .92)	.45 (.00, .82)	.10 (.03, .26)	−.42 (−1.00, .28)	1.00 (.23, 1.00)	.61 (.08, .90)
2	**AE-AE r_g, r_e**	**.80 (.66, .89)**	**---**	**.20 (.11, .34)**	**.91 (.77, .97)**	**---**	**.09 (.03, .23)**	**.22 (−.02, .45)**	**---**	**.57 (−.02, .94)**
		Hyperactivity/Impulsivity & Overweight/Obesity (Girls)								
Model		Hyperactivity/Impulsivity			Obesity			Correlations		
Number	Model	a^2	c^2	e^2	a^2	c^2	e^2	r_g	r_c	r_e
1	ACE-ACE r_g, r_c, r_e	.86 (.46, .94)	.00 (.00, .36)	.14 (.06, .27)	.68 (.06, .98)	.23 (.00, .80)	.10 (.03, .24)	.27 (−.40, 1.00)	−.95 (−1.00, 1.00)	.31 (−.48, .90)
2	**AE-AE r_g, r_e**	**.86 (.73, .94)**	**---**	**.14 (.06, .27)**	**.91 (.77, .97)**	**---**	**.09 (.03, .23)**	**.23 (−.02, .46)**	**---**	**.31 (−.48, .90)**

[a] A = additive genetic effects; C = common environmental effects; E = unique environmental effects; r_g = correlation between presence of ADHD symptoms and overweight/obesity that is due to additive genetic influences; r_c = correlation between presence of ADHD symptoms and overweight/obesity that is due to common environmental influences; r_e = correlation between presence of ADHD symptoms and overweight/obesity that is due to unique environmental influences.

[b] Statistics for the best-fitting model appear in bold. 95% confidence intervals are presented in parentheses.

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