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Sex differences in the genetic and environmental influences on self-reported non-aggressive and aggressive conduct disorder symptoms in early and middle adolescence

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Abstract

Decades of research into the etiology of conduct disorder (CD) has yet to yield a consensus on the existence of sex differences in underlying genetic and environmental influences. This may be partly due to the failure of many previous studies to make a distinction between non-aggressive and aggressive CD symptoms or test for potential developmental changes in sex differences in the etiology of conduct problems. To address these gaps, we fit a series of univariate and bivariate biometric sex-difference models to self-reported non-aggressive and aggressive CD symptoms in a community-based sample of twins ($N=1,548$, ages 9–17 year), grouped into ages 9–13 years and 14–17 years. Relative model fit was evaluated using the Bayesian Information Criterion (BIC), which favors parsimony, and by chi-square difference tests.

The univariate sex-scalar model provided the best fit to the data for both non-aggressive and aggressive CD symptoms at ages 9–13 years and 14–17 years. Thus, the same genetic and environmental factors influenced CD symptoms in both sexes, but the total variability was lower in females than males. At both ages, the heritability of non-aggressive CD symptoms was lower than heritability of aggressive CD symptoms, and shared environmental effects were only observed for non-aggressive CD symptoms. However, estimates for genetic and environmental factors could be not be constrained to be equal across age groups for either CD subtype, suggesting substantive developmental changes in the relative influence of genetic and environmental factors on individual differences in CD symptoms. For both subtypes, the heritability was larger, and shared environmental effect smaller, in the older age group than the younger age group.

A bivariate quantitative sex differences model provided the best fit to the data at ages 9–13 years. Covariation between non-aggressive and aggressive CD symptoms was due to overlapping shared and non-shared environmental factors in males and females but the overall covariation was greater in males than females. In contrast, at ages 14–17 years, the sex-scalar bivariate model provided the

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All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

best fit to the data, and covariation between non-aggressive and aggressive CD symptoms was due to overlapping genetic and non-shared environmental factors.

Thus, the etiology of self-reported conduct disorder varied substantially by symptom type and age. However, quantitative sex differences were only apparent when the covariation between the two subtypes was considered.

Keywords

conduct problems; twins; behavior genetics; adolescents; sex differences

Conduct Disorder (CD) is characterized by a persistent violation of social norms that harm or impede the rights of others. Symptoms can range from relatively mild offenses like lying or minor vandalism, to more serious crimes like theft or violence. Sex differences in youth conduct problems, especially aggression, have long been reported (Rosanoff, Handy, & Rosanoff, 1934), and being male is one of the strongest predictors of antisocial behavior, beginning in early childhood and continuing into adulthood (Moffitt, Caspi, Rutter, & Silva, 2001). Males engage in more direct and physical forms of aggression than females (Archer, 2004; Lahey et al., 2000), whereas females tend to engage in more indirect forms of aggression (Archer, 2004, but see Karriker-Jaffe, Foshee, Ennett, & Suchindran, 2008). Given the reliable phenotypic sex differences in conduct disorder, it is necessary to understand potential genetic and environmental causes of these sex differences to fully understand the causes of CD symptoms themselves. Conversely, any theory of the etiology of conduct disorder that does not explain the sources of sex differences would be inaccurate for one or both sexes (Rutter, Caspi, & Moffitt, 2003). Despite decades of research, however, no consensus has emerged on the existence or nature of sex-specific genetic and environmental influences on CD symptoms in children and adolescents, likely due in part to the substantial heterogeneity in CD symptoms. Researchers have attempted to reduce this heterogeneity by disaggregating subtypes of CD and incorporating child and adolescent development.

Sex differences in the etiology of subtypes of conduct problems

Although conduct disorder is conceptualized as a unitary construct (American Psychiatric Association, 2013), the behaviors that define conduct problems range from physical aggression, to status offenses like truancy, to non-violent property crimes. Several factor analyses support parsing CD into separable non-aggressive and aggressive subtypes (Frick et al., 1993; Lahey et al., 2000; Tackett, Krueger, Iacono, & McGue, 2005). Non-aggressive CD encompasses property crimes and status offenses (Lahey et al., 2000), whereas, aggressive CD refers to overt, physical aggression such as fighting and bullying. In general, genetic influences are stronger on aggressive CD symptoms than non-aggressive CD symptoms. Shared environmental influences are consistently reported for non-aggressive CD symptoms, but do not contribute to variation in aggressive CD symptoms (see review by Burt, 2009).

Are these subtypes of conduct disorder subject to sex-specific etiologies? Unfortunately, as with unitary measures of CD, no definitive answer has yet emerged. There is both substantial heterogeneity in the evidence for sex-specific influences and the strength of those influences, on non-aggressive and aggressive CD symptoms. For example, Gelhorn et al. (2005) tested for sex differences in non-aggressive and aggressive CD subtypes in a sample of 11-18 year old twins, but reported that, after adjusting for mean level sex differences, the best fitting genetic model omitted sex differences entirely. Genetic factors accounted for 55% and of the variance in non-aggressive CD and 49% of the variance in aggressive CD symptoms, with non-shared environment accounting for the remaining variation. Van Hulle, Rodgers, D'Onofrio, Waldman, & Lahey, (2007) likewise did not find evidence for sex differences in the genetic and non-shared environmental influences on non-aggressive and aggressive CD in sample of full and half-siblings aged 14-17 years. The overall phenotypic variance of both CD subtypes was 15% smaller in females compared to males. After adjusting for the sex differences in total variation (i.e. sex scalar effects), genetic factors accounted for only a third of the variation in both non-aggressive and aggressive CD symptoms with non-shared environment accounting for the remaining phenotypic variation. van den Oord, Boomsma, & Verhulst, (1994) also reported finding sex scalar effects in the etiology of non-aggressive CD symptoms in a sample of biological and adopted siblings aged 10-15 years. Unlike studies with older children, van den Oord et al. reported significant shared environmental influences (19%) in addition to genetic (38%) and non-shared environmental influences (41%).

In the same study, van den Oord et al. (1994) reported *quantitative* sex differences (i.e. sex-specific estimates of heritability and environmental variation) for aggressive CD symptoms. Genetic influences were stronger in males than females (89% vs 52%) and shared environment influences were observed for females only (32%). Bartels, van de Aa, van Beijsterveldt, Middeldorp, & Boomsma (2011) also reported significant quantitative sex differences in CD subtypes in twins ages 12-20 years, with higher heritability¹ estimates for males (50%) than females (22%-38%) for both non-aggressive CD as well as aggressive CD; again, shared environmental influences, this time for non-aggressive CD, was observed in females only (21%).

Age as moderator of sex differences in the etiology of subtypes of conduct problems

The studies described above span early to late puberty. Some focus on a relative narrow age range (Van Hulle et al., 2007) and others include participants at very different stages of development (Bartels et al., 2011). The differences in participant ages make comparing results across studies difficult, in part, because non-aggressive and aggressive CD symptoms follow different developmental trajectories (Burt, 2009, 2012). Non-aggressive CD symptoms are characteristic of late onset conduct disorder while aggressive behaviors typify early onset conduct disorder (Moffitt et al., 2001). Tests of age-related changes in the etiology of non-aggressive and aggressive CD typically find that the relative impact of genetic and environmental influences on non-aggressive CD change with age, such that

¹Point estimates reported for average age 16.5 years.

genetic influences increase, and shared environmental influences decrease, from early childhood to adolescence, but genetic and environmental influences on aggressive CD are typically stable from childhood to adolescence (Burt & Neiderhiser, 2009; Eley, Lichtenstein, & Stevenson, 1999).

To date, very few studies have incorporated developmental changes with an examination of sex differences in the etiology of non-aggressive or aggressive CD subtypes. Bartels et al. (2011) reported that heritability of non-aggressive CD was stable and consistently higher for males than females across development in their cross-sectional sample of twins ages 12-20 years. Similarly, although the magnitude of genetic and environmental influences on aggressive CD fluctuated across age, the overall heritability remained higher for males than females. Wang, Niv, Tuvblad, Raine, & Baker (2013) are the only researchers to examine possible age-dependent sex differences in genetic and environmental influences on non-aggressive and aggressive CD symptoms in a longitudinal sample of adolescents. Twin pairs were assessed at three ages: 9-10, 11-13, and 16-18 years. In males, the etiology of non-aggressive CD symptoms followed the expected pattern of increasing genetic influences and decreasing shared environmental influences with age. In females, all familial influences (genetic and shared environment) on non-aggressive CD increased between ages 9-10 years and ages 16-17 years. In contrast, genetic influences on aggressive CD reached their zenith at ages 11-13 in both males and females; aggressive CD was more highly heritable in females than males at the youngest age (9-10), but that pattern reversed in adolescence (11-13, and 16-17 years).

Sex differences in the etiology of covariance between subtypes of conduct problem

Non-aggressive and aggressive CD may be distinct subtypes but they are reliably correlated, both phenotypically and etiologically, even in community-based populations where overall symptom endorsement is low (Burt, 2009). Thus, understanding the etiology of non-aggressive and aggressive CD requires understanding the sources of covariation between them. Van Hulle et al. (2007) reported substantial genetic ($r_A = .59$) and non-shared environmental ($r_E = .55$) correlations between non-aggressive and aggressive CD symptoms. Other adolescent twin studies typically report similar genetic correlations but weaker non-shared environmental correlations between CD subtypes (Button, Scourfield, Martin, & McGuffin, 2004; Gelhorn et al., 2006). In her meta-analysis of, Burt (2013) estimated a genetic correlation of $r_A = .60$ between non-aggressive and aggressive CD symptoms that was stable across age and sex. The non-shared environmental correlation was much lower at $r_E = .32$, but was likewise stable across age and sex. Wang et al. (2013), which was published concurrently with Burt (2013), reported significant quantitative sex differences in the genetic and environmental covariation between non-aggressive and aggressive CD in early, middle, and late adolescence, but no consistent pattern emerged.

The Current Study

In sum, there is no agreed upon finding regarding patterns sex differences in the genetic and shared environmental influences on individual variation in non-aggressive and aggressive

CD symptoms. Our goal was, first, to replicate findings from our previous study of sex differences in the univariate and bivariate etiology of non-aggressive and aggressive CD in full and half-siblings (Van Hulle et al., 2007). Our second goal was to expand on those findings by exploring potential developmental differences in sex differences in the etiology of non-aggressive and aggressive CD symptoms.

We fit four models of increasing complexity, starting with no sex differences and moving to sex scalar effects, quantitative sex differences, and finally qualitative sex differences (i.e. sex-specific underlying genetic and environmental influences) to self-reported non-aggressive and aggressive CD symptoms. To examine possible development changes in the etiology of CD symptoms we fit the models separately to participants aged 9-13 years (early adolescence) and aged 14-17 years (middle adolescent).

Methods

Participants

The Tennessee Twin Study (TTS) sample was representative of 6–17-year-old twins born in Tennessee and still living in one of the state's five MSAs (Nashville, Memphis, Knoxville, Chattanooga, and Bristol) during enrollment. Contact information was provided by Tennessee Department of Health. A random sample of identified families was selected stratified on the age of the twins and 35 geographic subareas. Interviews were completed with 2,063 adult caretakers (90.8% biological mothers, 7.5% biological fathers, 0.5% step-mothers, and 1.2% grandmothers), with a response rate for caretakers of 69.8%. When the adult caretaker was interviewed, 97.9% of the twin pairs were also interviewed. Caretakers gave written informed consent and twins who were old enough to be interviewed (9 years of age) gave oral assent. Zygosity of same-sex twin pairs was determined using nine standard questions about similarities in the twins' physical appearance (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). Zygosity of twins in the ambiguous range of questionnaire scores was determined using 12 highly polymorphic markers genotyped from DNA extracted from swabbed cheek cells. Twins were randomly designated as twin 1 or 2 after data collection; 37% of the twins were monozygotic (MZ), 33% were same-sex dizygotic (DZ), and 30% were opposite-sex DZ. Youth were excluded if the caretaker reported that the youth had been diagnosed as psychotic (twin 1 $N = 17$; twin 2 $N = 11$), or autistic (twin 1 $n = 8$; twin 2 $n = 5$). Only pairs in which both twins were interviewed and for which there were complete CD data were included ($N = 1,981$ pairs, 743 MZ and 1,238 DZ). The full sample ranged in age from 6-18 years old ($M = 11.7$, $SD = 3.3$), 49% was male, and 71.4% of the sample was of Non-Hispanic European-American ethnic background, 24.0% was of African-American background, and the remaining 4.6% was of mixed or other ethnic background. Family income for participants in 2001 US dollars ranged from \$0 - >\$150,000 ($M = \$58,633$, $SD = \$43,086$). Children at least 9 years of age are reliable and valid informants on conduct disorder symptoms (Lahey et al., 2004). Thus, present analyses were limited to 1,548 pairs of 9- to 17-year-old twins ($M = 12.8$, $SD = 2.6$) for whom complete self-report data was available. We divided participants into early adolescent (9-13 years, $n = 926$ pairs) and middle adolescent (14-17 years, $n = 622$ pairs) age groups. There were no

differences in zygosity, sex ratio, ethnicity, family income, or parent education between the two groups.

Measures

Parents and youth were interviewed using the Child and Adolescent Psychopathology Scale (CAPS; Lahey et al., 2004). The CAPS items that cover *DSM-IV* symptoms are based on the “stem questions” of the *Diagnostic Interview Schedule for Children* (DISC). Instead of the typical yes/no format employed by the DISC, each CAPS item was rated on a 4-point scale: 1 indicated not at all; 2, just a little; 3, pretty much; and 4, very much. When choosing a response, participants were instructed to consider how well each question described his or her emotion or behavior, including frequency and severity, during the previous 12 months.

Conduct disorder symptoms were separated into non-aggressive (8 items) and overtly aggressive (7 items) CD subtypes. Non-aggressive CD symptoms included status offenses (e.g. truancy), and property crimes (e.g., vandalism). Aggressive CD symptoms included violence against persons or animals (e.g. fighting). Reliability (Cronbach’s alpha) was .64 for non-aggressive CD, and .63 for aggressive CD. Reliabilities were approximately the same when participants were divided into early (reliability = .62 for both subscales) and middle adolescent age groups (reliability = .64 for both subscales). Mean ratings across symptoms within each subtype were positively skewed, with most participants endorsing few symptoms; thus, scores were log-transformed prior to analyses.

We confined our analyses to self-reported CD symptoms to avoid a) rater biases, and b) potential under-reporting by parents. For each gender and age group, average youth-reported non-aggressive and aggressive CD symptom ratings were significantly higher than parent reported symptoms ratings. Correlations between parent reported behavior and youth reported behavior ranged from .15 (aggressive CD in females ages 9-13) to .37 (non-aggressive CD in females ages 14-17). Adding parent report to self-report (taking the higher of the two ratings) did not change the phenotypic correlations within twin pairs or between CD subtypes. The test–retest intra-class correlations (ICCs) for unit-weighted average youth ratings of CD symptoms was 0.89 and robust correlations between self-reported CD and functional impairment indicated external validity (Lahey et al., 2004). Thus, we considered twins adequate reporters.

Biometric models

Univariate biometrical models partition the observed phenotypic into unmeasured latent additive genetic, shared environmental, and nonshared environmental influences. Greater intra-pair similarity among MZ pairs compared to DZ pairs signifies genetic influences (A). Shared environmental influences (C) are defined as non-genetic factors that make siblings reared together more similar to each other than otherwise expected. Nonshared environmental influences (E) are non-genetic factors that contribute to sibling differences (including measurement error). Estimates of A, C, and E reflect the cumulative effect of genes and environment without implicating any specific set of genes or experiential event.

In standard biometric twin models, the genetic correlation between twins (r_G) is 1.0 for MZ pairs and 0.5 for DZ pairs; the shared environmental correlation (r_C) is 1.0 for MZ and DZ

pairs alike; nonshared environmental influences are uncorrelated. Estimates of A, C and E are typically presented as standardized variance components and sum to 1.0.

The univariate model can be extended to include two or more phenotypes. In addition to decomposing the phenotypic variance of non-aggressive and aggressive CD into underlying genetic, shared environment, and non-shared environmental influences, the *bivariate* model also decomposes the *covariation* between non-aggressive and aggressive CD symptoms into genetic and environmental factors. This allows for the estimation of the extent to which genetic, shared environmental, and nonshared environmental influences may be *common* to non-aggressive and aggressive CD.

To test for sex differences in biometric models, twins were grouped by sex in addition to zygosity. We tested four possible sex-difference outcomes: 1. No sex differences or sex equal – all parameters, including the overall variance were constrained to be the same for males and females. 2. Sex scalar effects – the relative contribution of genes and environment were constrained to be the same for males and females, but the total phenotypic variance in females was expressed as a scalar multiple (k) of the total phenotypic variance in males. Thus, the standardized estimates of genetic and environmental variation are equal for males and females. 3. *Quantitative* sex-differences – r_G and r_C were fixed at 0.5 and 1.0 respectively for both same-sex DZ and opposite-sex DZ pairs, but the influence of A, C and E could vary by gender. Thus, the same set of genes are assumed to influence the outcome in both males and females, but the relative magnitude of genetic and environmental influences on the variance may be higher or lower in females compared to males. 4. *Qualitative* sex-differences – the relative influence of A, C and E could vary by gender and in addition, the correlation between additive genetic factors (r_G), fixed at 0.5 for same sex DZ pairs, was allowed to be freely estimated for opposite-sex DZ pairs. The shared environmental correlation (r_C), fixed at 1.0 for same-sex DZ pairs, was also freely estimated for opposite-sex pairs. An r_G significantly less than 0.5 or r_C less than 1.0 indicates sex-specific genetic or shared environmental influences.

Analytic Strategy

All phenotypic analyses controlled for clustering within families. We tested for demographic predictors of CD symptoms (see Table 2). Participant ethnicity and family income predicted both non-aggressive and aggressive CD symptoms. Compared to non-Hispanic white participants, African American participants endorsed more aggressive CD symptoms and both African American and Hispanic participants endorsed more non-aggressive CD symptoms than non-Hispanic white participants. As family income decreased, non-aggressive and aggressive CD symptom ratings increased. Failing to adjust for these non-focal, “nuisance” characteristics common to members of a twin pair will lead to an overestimation of twin similarity (McGue & Bouchard, 1984). Therefore, we residualized non-aggressive and aggressive CD scores on race/ethnicity and income prior to fitting biometric models.

We tested for potential age-by-sex interactions in the genetic and environmental influences on non-aggressive and aggressive CD disorder by dividing the participants into early adolescent (9-13 years) and middle adolescent age groups (14-17 years). Samples sizes for

the early adolescent group were: MZ females = 180, DZ females = 155, MZ males = 172, DZ males = 150, DZ opposite sex pairs = 269. Sample sizes for the middle adolescent group were: MZ females = 133, DZ females = 98, MZ males = 108, DZ males = 105, DZ opposite sex pairs = 178. We fit the aforementioned series of univariate models to non-aggressive and aggressive CD symptoms separately for each age group. We fit an analogous series of bivariate models to test for sex differences in the relative influence of A, C, and E on the covariation between non-aggressive and aggressive conduct disorder symptoms for each age group.

Income was missing for 21 families. An analysis of patterns of missing data suggested that data were missing completely at random (Little's MCAR: $\chi^2(5) = 5.2$, $p = 0.64$). Models were estimated using *Mplus* version 7.3 (Muthén and Muthén 1998-2012) which uses a Full Information Maximum Likelihood procedure to account for missing data. This procedure produces less biased parameter estimates than a complete-cases-only analyses and is appropriate for use with MCAR data (Enders, 2010). All models were fit using a maximum likelihood estimation with robust standard errors (Muthén & Muthén, 1998-2012).

Overall model fit was considered acceptable if RMSEA was below .08 (MacCallum, Browne and Sugawara). Models were compared using Bayes Information Criteria (BIC) and Satorra-Bentler scaled log-likelihood difference tests (Satorra & Bentler, 2001). A significant change in fit indicated that the more complex model was preferred while a non-significant change indicated that the more parsimonious model was the preferred model. For completeness we also report the Comparative Fit Index (CFI) and the Tucker-Lewis Index (TLI; (Hu & Bentler, 1999). However, these latter indices do not impose a penalty for additional parameters (Rigdon, 1996).

Results

Phenotypic analyses

Results of regressing non-aggressive and aggressive CD on demographic predictors, including age in years and sex, are shown in Table 1. On average, males rated non-aggressive CD symptoms higher than females by 0.33 standard deviations ($p < .001$) and aggressive CD symptoms higher by 0.36 standard deviations ($p < .001$). Age was not significantly related to aggressive CD symptoms (standardized estimate = 0.03, $p = .17$), nor did we find evidence for an age-by-sex interaction ($p = .75$). Consistent with previous studies (e.g. Lahey et al., 2000), older participants overall endorsed more non-aggressive CD symptoms than younger participants (standardized estimate = 0.17, $p < .001$), and age moderated the sex differences in non-aggressive CD symptoms ($p = .003$). The age-related increase in non-aggressive CD symptoms was much steeper for males (standardized simple slope = .07, $p < .0001$) than females (standardized simple slope = .03, $p < .001$). Mean differences in CD subtype by age and sex are summarized in Figure 1. Non-aggressive and aggressive CD symptoms were correlated more strongly for males ($r = .57$) than females ($r = .42$; *Fisher's r-to-z transformation* = 4.3, $p < .001$) among younger adolescents. Among older adolescents the correlation between non-aggressive and aggressive CD was nearly identical for males and females ($r = .53$ and $.51$, respectively' *Fisher's r-to-z transformation* = 0.37, $p = .35$).

Univariate Analyses

Means and standard deviations for raw non-aggressive and aggressive CD symptom ratings are shown in Table 2 by sex, zygosity, and age group. We fit the series of sex differences models separately to non-aggressive and aggressive CD symptoms and to early and middle adolescent age groups. Model fitting results are summarized in Table 3. We compared each model to the previous one to determine if the more complex model improved on model fit. All the sex-differences models provided a good fit to the data with the exception of the no sex differences model. Starting with the early adolescence group, the sex-scalar model provided the best fit to non-aggressive CD symptoms (difference chi-square = 10.9, 1 *df*, $p < .001$ compared to no sex differences model) and aggressive CD symptoms (difference chi-square = 53.2, 1 *df*, $p < .001$ compared to no sex differences model). The qualitative and quantitative sex differences models failed to provide a significantly better fit to the data. Standardized variance components from the sex-scalar models are presented in Table 4 (left panel). The estimated total variance of non-aggressive and aggressive CD was approximately 25% to 30% lower in females than males. At this age, non-shared environment accounted for the majority of the variance for both non-aggressive (58%) and aggressive (69%) CD symptoms. We found significant shared environmental influences on non-aggressive CD symptoms (31%) and marginal genetic influences (11%). In contrast, we found marginal shared environmental influences (11%) and modest genetic influences (20%) on aggressive CD symptoms.

The sex-scalar model also provided the best fit to non-aggressive and aggressive CD symptoms in middle adolescence (Table 3, right panel). Standardized variance components from the sex-scalar models are presented in Table 4 (right panel). We found a similar reduction of 20% to 30% in total variation in CD symptoms in females relative to males. However, for this age group, genetic influences on non-aggressive CD were robust (39%) with modest shared environmental influences (13%). Genetic (47%) and non-shared environmental (53%) influences accounted for all of the variance in aggressive CD, with no evidence for shared environmental influences.

To formally test for these striking age differences in the genetic and environmental point estimates, we fit a 12 group (6 sex/zygosity groups per age band) univariate sex-scalar model (see supplemental Table 1). Allowing the magnitude of genetic and environmental influences on non-aggressive CD to vary by age group produced a significantly better model fit than one that constrained estimates to be the same across age groups (chi-square difference = 13.9, 4 *df*, $p = .007$). The genetic variance of non-aggressive CD was smaller in the early adolescent group (11%) compared to middle adolescent group (39%). The reverse pattern was found for shared environment which accounted for 31% and 13% of the variation in non-aggressive CD respectively. Non-shared environmental influences were slightly larger in early adolescents than middle adolescents (58% and 48% respectively). The total variation was approximately 33% lower in females than males for both younger and older adolescents.

Likewise, the more complex model allowing for age-related variations in parameter estimates provided a better fit to aggressive CD symptoms than a model that held parameter estimates constant across ages (chi-square difference = 10.8, 4 *df*, $p = .03$). Genetic influences accounted for little variation for early adolescents compared to middle adolescents (18% vs

47%). Consequently, non-shared environmental influences were significantly greater for early adolescents than middle adolescents (70% vs 53%) while. In addition, the reduction in total variation in aggressive CD in females relative to males went from 46% in the younger age group to only 28% smaller in the older age group.

Bivariate Analyses

We tested for sex differences in the bivariate models for younger and older adolescents separately. Model fitting results are summarized in Table 5. Like the univariate models, we found marked differences between the two age groups. For the early adolescent group, the quantitative sex differences model was the best fitting model² (chi-square difference = 32.9, $p < .00$ compared to the sex scalar model). Parameter estimates from the best fitting model (shown in Table 6) suggest that the sex differences were driven by differences in the etiology of the covariation between CD subtypes. We detected significant shared and non-shared influences common to both non-aggressive and aggressive CD symptoms. Together these factors accounted for twice as much of the variation in aggressive CD symptoms in males (37%) as females (19%). Genetic variation unique to aggressive CD symptoms, though modest overall, was twice as large in females as males. The genetic (r_A) and environmental (r_C and r_E) correlations provide a measure of the degree of overlap between sources of variation on each subtype. The degree of overlap can be high even though the amount of variation accounted for in either trait is low. For example, the modest shared environmental influences on aggressive CD symptoms was entirely due to shared environmental factors that also influenced non-aggressive CD symptoms (i.e. $r_C = 1.0$) for both males and females. The non-shared environmental correlation (r_E) between non-aggressive and aggressive CD symptoms was .20 for males and .10 for females. For both males and females, shared and non-shared environment fully accounted for the variation in non-aggressive CD symptoms, thus $r_A = 0$.

For the middle adolescent group, the sex scalar model was the best fitting model (chi-square difference = 11.2, 1 df, $p < .001$ compared to the sex equal model). More complex sex differences models failed to improve model fit. Parameter estimates from the best fitting model are shown in Table 6. Genetic and non-shared environmental influences common to both subtypes accounted for 27% of the variation in aggressive CD symptoms; r_A was .38 and r_E .16. In the bivariate model, we found evidence for substantial genetic influences (55%) on non-aggressive CD symptoms and no evidence for shared environmental influences. Likewise, we did not find any evidence for shared environmental influences on aggressive CD symptoms, but the total genetic variation for aggressive CD was 44%.

Discussion

Females consistently engage in fewer delinquent behaviors than males despite the fact that early risk correlates for CD and developmental trajectories for CD are largely the same for males and females (Lahey et al., 2006; Karikker-Jaffe et al., 2008). Thus, it is unclear if the

²For this age group, the qualitative model failed to converge under numerous start values. When constraints were added to ensure that the genetic correlations between opposite sex pairs were .5 or less the model estimated genetic correlation was at the boundary value. Therefore, we did not consider this model further.

underlying genetic factors that contribute to individual differences in CD can be expected to be the same or different for males and females.

To test for sex differences in the context of age, we divided our sample into participants aged 9-13 years and 14-17 years. We chose these age bands for theoretical and practical reasons. An increase in delinquency across adolescence is normative to some extent. Thus, in theory, the older age group is more likely to include individuals following a typical or at least adolescent-limited delinquency pathway, while individuals high on CD symptoms in the younger age group are more likely to follow an atypical or life-course persistent delinquency pathway (Moffitt & Caspi, 2001). Practically, dividing the sample into the chosen age bands allowed us sufficient sample size in both groups to test complex sex-differences and allowed us to compare the current study to our previous work (Van Hulle et al., 2007). Thus, our findings must be interpreted with caution given that, in a cross-sectional cohort, any age grouping is inherently arbitrary.

Univariate results

Univariate analyses revealed four main findings. First, we replicated our previous finding that the etiology of non-aggressive and aggressive CD was the same for middle adolescent males and females, after adjusting for sex differences in the total phenotypic variation. We found this pattern to be true for the early adolescent age group as well. Heritability and shared environmental estimates were larger in the present study likely owing to the use of twins vs. siblings. These results are in keeping with Gelhorn et al. (2005) but stand in contrast with Bartels et al. (2011) and Wang et al. (2013).

If we accept that there are no sex differences in the underlying etiology of CD symptoms, what accounts for the persistent phenotypic sex differences? There are numerous factors that may contribute to sex differences in the expression and development of conduct problems. One theory is that sex differences reflect a failure to capture all relevant forms of aggression. For instance, relational aggression is not considered part of the diagnostic classification for CD (American Psychiatric Association, 2013) despite studies that indicate that the consequences for social and emotional health are similar to other forms of aggression (Crick & Grotpeter, 1995). This explanation only holds to the extent that the same set of genes contributed to some underlying trait (i.e. reactive anger) that manifests as relational aggression in females and physical aggression in males. However, the correlation between relational and physical aggression is quite high for both females and males (Crick, Ostrov, & Werner, 2006; Tiet, Wasserman, Loeber, McReynolds, & Miller, 2001), which suggests that females are not necessarily substituting relational aggression for other forms aggression. Another theory posits that, although the antecedents of conduct disorder symptoms are the same for both sexes, males are more likely to experience or be exposed to those antecedents (Lahey et al., 2006). For example, socialization factors appear to act as a stronger deterrent for females than males, and interpersonal relationships provide more protection against developing CD for females than males (Ehrensaft, 2005). Measures of CD symptoms have a strong floor effect so any reduction in aggressive behavior will shrink the variance in females relative to males.

Second, genetic influences accounted for more variability in aggressive CD symptoms than non-aggressive CD symptoms, whereas shared environment influenced non-aggressive CD symptoms. However, we found significant developmental changes in shared environment influences on non-aggressive CD symptoms which decreased with age, while genetic influences on non-aggressive CD symptoms increased with age. Both findings are consistent with previous research (Burt & Klump, 2009, Burt & Neiderhiser, 2009; Eley et al., 1999 but see Bartels et al., 2011) and are perhaps unsurprising. Changes in brain development during the adolescent period lead to an increase in risk-taking in general (Dahl, 2004). Changes in genetic influences on non-aggressive CD across ages can be accounted for by pubertal development, whereas, shared environment is moderated by age itself (Harden et al., 2015). Thus, at younger ages, when non-aggressive CD symptoms are less normative, external factors like parenting behaviors may be more influential (Hoeve et al., 2009) than at later ages.

Fourth, we found that the etiology of aggressive CD symptoms was not stable from early to middle adolescence. Rather, genetic factors accounted for a greater proportion of the individual variation in middle adolescence than early adolescence. These findings are somewhat similar to Wang et al. (2013) who reported an increase in genetic influences on aggressive CD with age among males, but not females. This discrepancy between previous studies that show genetic influences on aggressive CD are stable across time (Burt & Klump, 2009; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003) and our findings may be due to different informants. Burt & Klump (2009) rely on mother reports of adolescent behavior whereas the present study and Wang et al. (2013) rely on self-reports.

Bivariate sex differences

We found that age moderated the sex differences in genetic and environmental influences on the covariation between non-aggressive and aggressive CD. Again, we replicated our previous finding of no sex differences beyond scalar effects in the relationship between non-aggressive and aggressive CD in middle adolescence. In contrast, we found evidence for quantitative sex differences in early adolescence, with greater covariation overall in males than females and more shared environmental covariation, in particular, in males than females. Wang et al. (2013) also found that a larger proportion of covariation was due to shared environment in males than females at ages 9-10 and 11-13. While these results indicate that the dimensions of CD share underlying genetic and environmental influences, we were unable to determine if non-aggressive CD has a *causal* influence on aggressive behavior or vice versa. However, early disruptive behavior has been shown to directly influence later delinquency in males (Tremblay et al., 1992). This may help explain the higher covariation between aggressive and non-aggressive CD reported by males than females.

Limitations

Several limitations deserve highlighting. First, while parent reported data were available, we focused on self-reported symptom ratings. As noted, adding parent report to self-report (i.e. taking the higher of the two ratings) did not change the phenotypic correlations. Nevertheless, there is some evidence that self-reported CD symptoms are less reliable than

parent-reported CD symptoms (Bartels et al., 2003). Second, different fit indices make different assumptions, and while RMSEA and BIC consistently pointed to the same best-fitting model, the CFI and TLI did not. CFI and TLI do not penalize complexity as do the BIC and RMSEA, and favored the qualitative sex differences bivariate model at age 9-13. However, given a near absence of qualitative sex differences in the literature on CD, we chose to reject the qualitative sex difference model in favor of the more parsimonious quantitative sex differences model. Third, we chose to group participants into two age bands, rather than examine continuous moderation of variance components by age. This was done to avoid detecting spurious gene x environmental effects when the outcome is highly skewed (Rathouz, Van Hulle, Rodgers, Waldman, & Lahey, 2008; Zheng, Van Hulle, & Rathouz, 2015). Fourth, researchers have begun to break non-aggressive CD and aggressive CD into even more fine-grained subtypes such as status/property offenses (Lahey et al., 2000) or premeditated/impulsive aggression (Mathias et al., 2007). These more refined constructs may be subject to different genetic and environmental factors. We omitted measures of relational aggression because it is not part of the CD construct (American Psychiatric Association, 2013). In addition, phenotypic sex differences in relational aggression are not as consistent as sex differences in direct aggression and may depend even more heavily on age and informant (Archer, 2004; Tackett, Waldman, & Lahey, 2009).

Conclusions

These results confirm several previous findings. Namely, that the underlying genetic and environmental influences on CD symptoms appear to be the same for males and females, regardless of CD subtype. Shared environmental influences are greater for non-aggressive CD than aggressive CD symptoms, whereas genetic influences account for more variation in aggressive CD than non-aggressive CD symptoms. Genetic influences account for more variability in both subtypes in middle adolescence compared to early adolescence. Overlapping non-shared environmental factors influence both non-aggressive and aggressive CD symptoms. At the same time, unlike other studies we found that genetic influences on aggressive CD increased with age. We also found quantitative sex differences in the shared environmental influences on non-aggressive and aggressive CD at younger ages. And finally, the contribution of genes or shared environment to the covariation in CD symptoms appears to be age-dependent.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Mean log-transformed aggressive (Agg) and Nonaggressive (NonAgg) CD symptoms for early (9-13) and middle (14-17) adolescent males and females.

Table 1

Demographic predictors of nonaggressive and aggressive conduct disorder symptoms.

	Non-aggressive CD			Aggressive CD		
	β	<i>Z</i>	<i>p</i>	β	<i>Z</i>	<i>p</i>
Youth's sex ^a	-0.167	9.1	<.001	-0.178	9.9	<.001
African-American	0.130	5.6	<.001	0.055	2.3	.02
Other minority	0.057	2.2	.03	0.032	1.4	.16
Income	-0.091	3.6	<.001	-0.098	3.6	<.001
Age in years	0.170	8.5	<.001	0.026	1.4	.17

Note: 24% of participants are African American, 4% other; average log-transformed income category is 2.9 (range 0-3.2).

^a reference group = males

Table 2

Means (SD) for raw non-aggressive and aggressive conduct disorder symptoms by zygosity, sex, and age group.

	Non-Aggressive CD		Aggressive CD	
	Male	Female	Male	Female
<i>9-13 Years</i>				
MZ	0.113 (0.19)	0.061 (0.13)	0.081 (0.18)	0.032 (0.10)
SS-DZ	0.119 (0.23)	0.089 (0.19)	0.110 (0.24)	0.040 (0.12)
OS-DZ	0.103 (0.20)	0.064 (0.12)	0.073 (0.16)	0.022 (0.08)
<i>14-17 years</i>				
MZ	0.183 (0.25)	0.085 (0.18)	0.083 (0.17)	0.038 (0.15)
SS-DZ	0.214 (0.26)	0.122 (0.19)	0.100 (0.21)	0.044 (0.14)
OS-DZ	0.201 (0.24)	0.104 (0.17)	0.101 (0.19)	0.049 (0.16)

Note: *N* pairs at age 9-13 years: 184 MZ females, 143 DZ females, 159 MZ males, 151 DZ males, 239 DZ opposite sex pairs; *N* pairs at ages 14-17 years: 163 MZ females, 119 DZ females, 142 MZ males, 130 DZ males, 227 DZ opposite sex pairs.

Table 3
Results from fitting univariate sex differences models separately to non-aggressive and aggressive CD symptoms.

Model Fit Indices										
Model	Ages 9-13 years					Ages 14-17 years				
	RMSEA	TLI	CFI	BIC	χ^2, df	RMSEA	TLI	CFI	BIC	χ^2, df
Non-aggressive CD										
No sex differences	.098 (.067-.129)	.881	.504	-2297		.073 (.019-.116)	.954	.808		-1116
Sex scalar	.064 (.022-.099)	.949	.795	-2353	10.9, 1	.000 (.000-.022)	1.0	1.0	-1152	13.4, 1
Quantitative differences	.068 (.027-.104)	.943	.790	-2341	3.7, 2	.000 (.000-.000)	1.0	1.0	-1145	3.4, 2
Qualitative differences	.067 (.020-.107)	.943	.829	-2317	3.1, 2	.000 (.000-.000)	1.0	1.0	-1133	1.6, 2
Aggressive CD										
No sex differences	.098 (.067-.129)	.881	.504	-2297		.044 (.000-.094)	.972	.882		-1623
Sex scalar	.044 (.000-.083)	.898	.592	-2967	53.2, 1	.000 (.000-.032)	1.0	1.0	-1680	5.5, 1
Quantitative differences	.045 (.000-.085)	.894	.611	-2956	1.1, 2	.000 (.000-.028)	1.0	1.0	-1674	2.0, 2
Qualitative differences ^a	.043 (.000-.085)	.901	.652	-2948	.18, 1	.000 (.000-.016)	1.0	1.0	-1672	2.9, 1

Note: Subsequent models are compared to the prior model; standard errors for RMSEA are shown in parentheses; sex equal model constrains all path estimates to be the same for males and females; sex scalar model constrains path estimates for females to be a scalar multiple (*k*) of path estimates for males; quantitative difference model constrains $rG = 5$ and $rC = 1.0$ for opposite sex (OS) DZ pairs; qualitative differences model allows the genetic correlation (rG) and shared environmental correlation (rC) to be freely estimated OS DZ pairs. The best fitting model is shown in bold.

^a rC omitted because shared environmental paths were estimated at 0.

Table 4

Parameter estimates (95% CI) from univariate scalar sex difference model for non-aggressive and aggressive CD symptoms by age group.

	9-13 years				14-17 years			
	a^2	c^2	e^2	k	a^2	c^2	e^2	k
Non-aggressive CD	.11 (-.14 .35)	.31 (.10 .52)	.58 (.49 .67)	.77	.39 (.11 .67)	.13 (-.07 .33)	.48 (.36 .59)	.77
Aggressive CD	.20 (-.18 .58)	.11 (-.15 .36)	.69 (.53 .86)	.56	.47 (.33 .61)	.00	.53 (.38 .67)	.73

Note: a^2 = genetic variance, c^2 = shared environmental variance, and e^2 = non-shared environmental variance are standardized estimates and indicate the proportion of total variability accounted for by each factor; k = sex-scalar parameter.

Table 5

Results from fitting sex differences bivariate models to non-aggressive and aggressive CD symptoms.

Model	Model Fit Indices				Model Comparison		
	RMSEA	TLI	CFI	BIC	df	χ^2	p
<i>Ages 9-13 years^a</i>							
Sex equal	.117 (.098-.137)	.546	.773	-4378	72		
Sex scalar	.073 (.049-.095)	.828	.913	-4599	71	95.8	1 <.001
Quantitative sex differences	.051 (.013-.078)	.923	.956	-4643	64	32.9	8 <.001
Quantitative sex difference, drop genetic influence^b	.052 (.016-.078)	.916	.955	-4661	68	6.2	4 .18
<i>Ages 14-17 years^c</i>							
Sex equal	.057 (.018-.085)	.941	.970	-3102	72		
Sex scalar	.000 (.000-.029)	1.0	1.0	-3191	71	11.2	1 <.001
Quantitative sex differences	.000 (.000-.012)	1.0	1.0	-3169	64	10.5	7 .16
Qualitative sex differences	.000 (.000-.026)	1.0	1.0	-3150	61	0.12	3 .99
Sex Scalar, drop all shared environment^d	.000 (.000-.029)	1.0	1.0	-3202	73	1.0	2 .39

Note: NonAgg = nonaggressive CD; standard errors for RMSEA are shown in parentheses; sex equal model constrains all path estimates to be the same for males and females; sex scalar model constrains path estimates for females to be a scalar multiple (*k*) of path estimates for males; quantitative difference model constrains $r_G = .5$ and $r_C = 1.0$ for OS DZ pairs; qualitative differences model allows r_G and r_C to be freely estimated in OS DZ pairs. The best fitting model is shown in bold.

^aQualitative sex differences model failed to converge.

^b*Nested*/model omitting genetic influences associated with nonaggressive CD is compared to the full quantitative sex differences model.

^cShared environment influences unique to aggressive CD symptoms were omitted from all models.

^d*Nested*/model omitting all shared environmental influences is compared to the full sex scalar model.

Table 6

Parameter estimates (95% CI) from best fitting bivariate sex difference models for each age group.

Ages 9-13 years (Quantitative sex differences model)							
	Shared ^a						
Females	<i>a</i> ²	<i>c</i> ²	<i>e</i> ²	<i>a</i> ²	<i>c</i> ²	<i>e</i> ²	
Non-aggressive	0.0	.40 (.34-.55)	.60 (.46-.74)				
Aggressive	.13 (.03-.33)	—	.67 (.49-.86)	—	.12 (.03-.27)	.07 (.03-.14)	
	Shared ^a						
Males	<i>a</i> ²	<i>c</i> ²	<i>e</i> ²	<i>a</i> ²	<i>c</i> ²	<i>e</i> ²	
Non-aggressive	0.0	.38 (.26-.52)	.62 (.50-.75)				
Aggressive	.06 (.003-.18)	—	.56 (.46-.68)	—	.24 (.15-.37)	.13 (.06-.22)	
Ages 14-17 years (Sex scalar model)							
	Shared ^a						
	<i>a</i> ²	<i>c</i> ²	<i>e</i> ²	<i>a</i> ²	<i>c</i> ²	<i>e</i> ²	<i>k</i>
Non-aggressive	.55 (.45-.65)	—	.45 (.36-.56)				
Aggressive	.25 (.16-.37)	—	.47 (.34-.62)	.19 (.11-.29)	—	.08 (.04-.14)	.76

Note: Parameter estimates based on the best fitting bivariate from Table 5; a^2 = genetic variance; c^2 = shared environmental variance and e^2 = non-shared environmental variance are standardized estimates, k = sex-scalar parameter.

^aProportion of variance in Aggressive CD symptoms due to genetic, shared, or non-shared environmental factors that influence nonaggressive CD symptoms.