

# Are Oppositional-Defiant and Hyperactive–Inattentive Symptoms Developmental Precursors to Conduct Problems in Late Childhood?: Genetic and Environmental Links

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**Abstract** Inattentive–hyperactive and oppositional behavior have been hypothesized to be developmental precursors to conduct problems. We tested these hypotheses using a longitudinal sample of 6,466 offspring of women selected from nationally representative US households. Conduct problems across 8–13 years were robustly predicted by conduct problems at 4–7 years, but also were independently predicted to a small extent by both inattentive–hyperactive and oppositional behaviors at 4–7 years. Longitudinal multivariate behavior genetic analyses revealed that the genetic and environmental factors that influence conduct problems at both 4–7 and 8–13 years

also influence the putative precursors at 4–7 years. After genetic and environmental influences on conduct problems at 4–7 years were taken into account, however, inattentive–hyperactive and oppositional behavior at 4–7 years shared causal influences with conduct problems 8–13 years to a negligible extent. These findings suggest that after early conduct problems are controlled, little is gained in terms of prediction or understanding genetic and environmental influences on later child conduct problems by treating early inattentive–hyperactive and oppositional behavior as developmental precursors to later conduct problems.

**Keywords** Attention deficit/Hyperactivity disorder · Oppositional behavior · Conduct problems · Developmental precursors

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An important goal of developmental psychopathology is the description of regular developmental progressions from early precursors to more serious problems later in development. This is because it would be both theoretically revealing and practically useful if milder problems (a) typically emerge earlier than more serious problems, and (b) reliably predict later serious problems. In such cases, it should be possible to identify factors that govern the developmental progression from milder to more serious problems and find ways to prevent serious problems. Therefore, considerable effort has been made to identify the developmental precursors to serious conduct problems in older children and adolescents.

The behaviors that define attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) have long been hypothesized to be developmental precursors to serious conduct problems (Lahey et al. 2000).

ADHD refers to developmentally inappropriate levels of attention problems, motor hyperactivity, and impulsive behavior. ODD is defined by developmentally inappropriate levels of irritability, defiance, and the intentional annoyance of others.

### Do ADHD and ODD Precede Conduct Problems?

By definition, developmental precursors must precede the more serious problem in development. Unfortunately, there appears to be only one study in which the ages of onset of symptoms of ADHD, ODD, and conduct problems were directly compared in the same sample. A clinic study of 7- to 12-year-old males, which retrospectively assessed age of onset of all symptoms that were reported during the clinic assessment, found that most symptoms of ADHD and ODD emerged early in life, with half of their symptoms having median ages of onset by 5 years of age (Loeber et al. 1992). The median ages of emergence for the remaining symptoms of ADHD and ODD were later, however, with the last symptoms of each disorder emerging around 11 years (Loeber et al. 1992). In the same study, most symptoms of conduct disorder (CD) were reported to emerge only slightly later than those of ADHD and ODD, with the median age of onset for half of CD symptoms reported by 6 years. On the other hand, a number of serious conduct problems, such as forced sex, theft by confrontation, running away from home, and truancy, did not emerge until late childhood or adolescence (Loeber et al. 1992). Thus, this study suggested that many symptoms of all three disorders are present before 6 years of age, even though some CD symptoms emerge later than those of ADHD and ODD. This study is of limited value, however, because it is based on retrospective reports from a clinic sample that was not representative. Nonetheless, the ages of onset found in this study for ADHD, ODD, and CD symptoms are similar to those found in other clinic-based (Applegate et al. 1997) and population-based studies (Lahey et al. 1998; Willoughby et al. 2000) that did not compare ages of onset of across disorders.

Furthermore, recent longitudinal studies that began during toddler and preschool ages clearly indicate that many young children already exhibit serious conduct problems during the early childhood years when most symptoms of ADHD and ODD are first evident (Keenan et al. 2007; Tremblay 2004). Thus, although there may be a longer developmental window for the emergence of some serious conduct problems than most ADHD and ODD symptoms, it seems likely that many conduct problems emerge in early childhood concurrently with symptoms of ADHD and ODD. This raises a question about the extent to which early symptoms of ADHD or ODD function as

independent developmental precursors to later child conduct problems.

### Does Childhood ADHD Predict Later Conduct Problems?

Seminal prospective longitudinal studies (Hechtman et al. 1984; Loney et al. 1981) showed that children with ADHD symptoms are at increased risk for exhibiting serious conduct problems in adolescence. These studies did not control for conduct problems in childhood, however. Some more recent longitudinal studies did not support the hypothesis that childhood ADHD symptoms predict later conduct problems when early childhood conduct problems were controlled (Lahey et al. 2000; Lilenfeld and Waldman 1990; Loeber 1988; Loeber et al. 1995; Magnusson 1984; Satterfield and Schell 1997). Firm conclusions cannot be reached, however, as other longitudinal studies suggest that ADHD predicts later conduct problems even in the apparent absence of high levels of childhood conduct problems (Lambert 1988; Mannuzza et al. 2004; Mannuzza et al. 1993; Mannuzza et al. 1991).

### Does Childhood ODD Predict Later Conduct Problems?

Several studies suggest that childhood ODD predicts later serious conduct problems. A longitudinal study of 7- to 12-year-old clinic-referred boys found that ODD in the first assessment was a significant predictor of meeting criteria for CD for the first time during the next 7 years (Lahey et al. 2000; Loeber et al. 1995). Another longitudinal study of clinic-referred 6- to 17-year-old males who met criteria for ADHD found that ODD in the first assessment did not predict meeting criteria for CD over the next 3 years (Biederman et al. 1996). When the prevalence of new cases of CD among males who met criteria for ODD in the baseline assessment was compared to all youth who did not meet criteria for ODD at baseline in this sample (those with only ADHD and the normal controls), however, CD was significantly more likely to emerge at follow-up among youths with ODD (Biederman et al. 1996). Similarly, a longitudinal study of a representative sample reported that meeting criteria for ODD in the first assessment predicted meeting criteria for CD for the first time in later assessments in males but not females (Rowe et al. 2002). These studies suggest that meeting criteria for ODD at an early age means that a child is at increased risk for later meeting criteria for CD for the first time. This does not necessarily mean that the emergence of all symptoms of ODD preceded the emergence of all symptoms of CD in development, however. It only refers to the sequence of meeting criteria for the two disorders.

## Are There Genetic and Environmental Links Between Conduct Problems and Earlier ADHD and ODD?

If children with high levels of ADHD or ODD symptoms prove to be at high risk for future serious conduct problems, it would be very important to know why this occurs. For example, if early ADHD and/or ODD symptoms are early manifestations of the same genetic or environmental influences that will influence conduct problems later in childhood, knowing that is the case should facilitate the design of early interventions to reduce antisocial outcomes. This is clearly a possibility, as many cross-sectional studies suggest that concurrently assessed ADHD, ODD, and conduct problems are largely influenced by the same genetic factors (Dick et al. 2005; Nadder et al. 2002; Waldman et al. 2001) and perhaps the same environmental factors (Burt et al. 2005).

The critical question, however, is whether early childhood ADHD and ODD share genetic or environmental influences with later conduct problems, over and above the genetic and environmental influences that later conduct problems share with earlier conduct problems. If so, obtaining measures of ADHD and ODD in early childhood would allow prevention researchers to identify children who are at risk for later conduct problems for reasons not revealed by their early conduct problems. If early ADHD and ODD do not share genetic and environmental influences with later conduct problems beyond those shared with early conduct problems, the focus of prevention research probably should be on early childhood conduct problems rather than on ADHD and ODD.

Therefore, we conducted two kinds of analyses of longitudinal data from a large and diverse sample of the offspring of a representative sample of mothers. First, we used regression analyses to test the predictive hypotheses that inattentive-hyperactive behaviors resembling symptoms of ADHD and oppositional behaviors resembling symptoms of ODD at 4–7 years of age predict conduct problems at 8–13 years of age, controlling for demographic factors and conduct problems at 4–7 years of age. Second, we used longitudinal multivariate behavior genetic analyses to test the hypotheses that inattentive-hyperactive and oppositional behavior at 4–7 years share genetic or environmental influences with those on conduct problems at 8–13 years, over and above the genetic and environmental influences shared by the putative precursors with conduct problems at 4–7 years.

## Method

### Sample

The National Longitudinal Survey of Youth (NLSY) was funded by the Bureau of Labor Statistics to study the US

workforce. A nationally representative household sample of 9,763 14- to 22-year-old male and female youth who were not in the military was selected in 1979 using a complex survey design, with an oversample of African American and Hispanic youth. To date, 4,926 NLSY females (1,472 African American, 977 Hispanic, and 2,477 non-Hispanic European American and other groups) have given birth to children who have participated in the assessments of offspring. Biennial assessments of all biological offspring of each NLSY woman have been conducted during 1986–2004 on the Children of the NLSY (CNLSY; Chase-Lansdale et al. 1991), with participation in these assessments averaging  $\approx 90\%$ . In order to conduct longitudinal analyses on the same children at different ages, the present analyses were conducted on 6,994 offspring of 2,256 mothers who were assessed at least once during 4–7 years and at least once during 8–13 years of age. Because total family income is an important covariate in the regression analyses, and the variable with the greatest amount of missing data in survey research, the present regression analyses were limited to the 6,466 offspring of women who reported income data. Descriptive statistics on the sample are presented in Table 1.

Because the longitudinal behavior genetic analyses could only be conducted on children with at least one sibling, both of whom were assessed at both 4–7 and 8–13 years, 5,281 sibling pairs were included in these analyses (Table 1). Because only biological children of NLSY mothers participated, all CNLSY siblings are at least half siblings. Algorithms were developed to classify offspring pairs as either twins, full siblings, or half siblings, with some pairs classified as ambiguous siblings because they share a biological mother, but their sharing of the same biological father is uncertain (Rodgers et al. 2005, 1994). The CNLSY kinship links (including for ambiguous siblings) used in the present and previous analyses of this data set (Van Hulle et al. 2007) were validated by demonstrating that the heritability of physical height is 0.90, consistent with twin studies (Rodgers et al. 1994). Because ambiguous siblings are either full or half siblings, they were assigned a genetic coefficient of  $r_g=0.375$ , intermediate between full ( $r_g=0.5$ ) and half siblings ( $r_g=0.25$ ). There were too few identical twin pairs ( $n=11$ ) to include them in these analyses, but the 23 fraternal twin pairs were included with other full siblings.

### Measures of Child Problem Behavior

In each assessment across 4–13 years of age, mothers rated their children's behavior using the behavior problem index (BPI; Peterson and Zill 1986). The BPI was created for the CNLSY by selecting items from the Child Behavior Checklist (Achenbach 1978) that were strongly correlated

**Table 1** Descriptive Data

| Demographic characteristics of the full sample   |                  |                    |   |                     |
|--|------------------|--------------------|---|---------------------|
| Sex  | Females          | Males              |   |                     |
|  | 3,194            | 3,272              |   |                     |
| Race-Ethnicity   | African American | Hispanic           | Non-Hispanic white and other <sup>a</sup> |                     |
|  | 1,972            | 1,340              | 3,154                                     |                     |
| Total family income <sup>b</sup>   | Mean             | Standard deviation |   |                     |
|  | 31,789           | 72,469             |   |                     |
| Number of sibling pairs of each degree of genetic relatedness in the behavior genetic analyses               |                  |                    |   |                     |
|  | $r_g$            | Male pairs         | Female pairs                              | Different-sex pairs |
| Half siblings  | 0.25             | 259                | 255                                       | 572                 |
| Ambiguous siblings   | 0.375            | 177                | 193                                       | 361                 |
| Full siblings (blended)  | 0.5              | 190                | 184                                       | 313                 |
| Full siblings (non-blended)  | 0.5              | 711                | 644                                       | 1,422               |
| Intra-class correlations between siblings on each measure of child problems behavior among each sibling type |                  |                    |   |                     |
|  | Half             | Ambiguous          | Full blended                              | Full non-blended    |
| Hyperactive-inattentive 4–7  | 0.21             | 0.24               | 0.33                                      | 0.30                |
| Oppositional-defiant 4–7   | 0.32             | 0.31               | 0.38                                      | 0.36                |
| Conduct problems 4–7   | 0.34             | 0.30               | 0.41                                      | 0.43                |
| Conduct problems 8–13  | 0.36             | 0.37               | 0.42                                      | 0.46                |

<sup>a</sup> Almost exclusively non-Hispanic European American, but includes some Asian and Native American children

<sup>b</sup> In 1986 dollars

with factor scores. The mean of 3 BPI items rated on a 3-point scale were used to define *inattentive-hyperactive behavior*: has difficulty concentrating, cannot pay attention for long; impulsive, or acts without thinking; and restless or overly active, cannot sit still. The mean of three BPI items define *oppositional behavior*: argues too much; stubborn, sullen, or irritable; and has a strong temper and loses it easily. The mean of seven BPI items rated on a three-point scale define child *conduct problems* at each age: cheats or tells lies; has trouble getting along with teachers; disobedient at home; disobedient at school; bullies or is cruel or mean to others; breaks things on purpose or deliberately destroys things; and does not seem to feel sorry after misbehaving. Confirmatory factor analysis of these 15 items supported the distinction of these three dimensions of problem behaviors (D'Onofrio et al. 2008). Each mean problem behavior score was multiplied by the number of items in the scale. The median alpha coefficients over ages 4–13 years for inattentive-hyperactive behavior, oppositional behavior, and conduct problems was 0.70, 0.73, and 0.74, respectively. These definitions of problem behaviors overlap substantially with those used in previous longitudinal studies of population-based samples (Fergusson and Horwood 2002; Lahey et al. 2000; Moffitt et al. 1996). The CNLSY measure of child conduct problems is valid in the sense of robustly predicting convictions for nontrivial offenses during adolescence in the CNLSY (Lahey et al. 2006). Similarly, the inattentive-hyperactive score predicts tested academic achievement

controlling for oppositional behavior and conduct problems (D'Onofrio et al. 2007).

#### Demographic Covariates

Mothers classified their offspring using mutually exclusive categories as African American, Hispanic, or all other race-ethnic groups. Total family income was assessed in the wave when the mothers were closest to 30 years old to index earning capacity, expressed as the logarithm of 1986 inflation-adjusted dollars. This includes all income received by the woman and her spouse (if married), including food stamps and other forms of governmental support. Income from any cohabiting partner was not included, but there are few substantive differences when this source of income is included in economic analyses (Avellar and Smock 2005).

#### Statistical Analysis

*Predictive Regression Analyses* We first tested the roles of inattentive-hyperactive and oppositional behavior at 4–7 years as predictors of conduct problems at 8–13 years in the same children using predictive regression analyses, controlling for conduct problems at 4–7 years. Because the response variable of conduct problems at 8–13 years was highly skewed and kurtotic with a mode of zero, log-linear regression was conducted specifying Poisson working distributions in SAS PROC GENMOD. In order to take

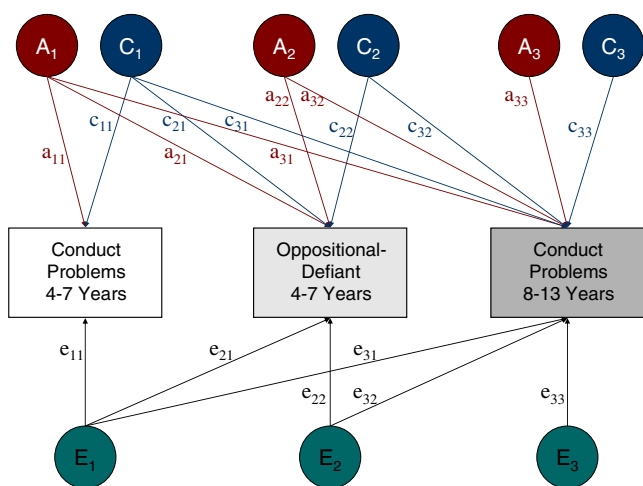
the clustering of children within families into account, these regressions were conducted in generalized estimating equations using robust standard errors (Zeger and Liang 1986).

*Longitudinal Multivariate Behavior Genetic Analyses* To determine if genetic and environmental influences on conduct problems at 8–13 years were shared with those on inattentive–hyperactive and oppositional behavior at 4–7 years, after genetic and environmental influences on conduct problems at 8–13 years shared with conduct problems at 4–7 years were taken into account, two separate series of multivariate behavior genetic analyses were conducted. Total observed variance in each of the four measures of child problem behavior based on the correlations among siblings of difference genetic relatedness (Table 1) was partitioned into estimates of variance attributable to latent additive genetic influences, shared environmental influences (i.e., shared experiences such as neighborhood factors that make siblings more similar), and nonshared environmental influences (i.e., either only one sibling experiencing an event such as bullying, or events experienced by both siblings that influence them in different ways, plus measurement error) based on differences in correlations between sibling pairs of differing genetic relatedness. Separate multivariate Cholesky decompositions (Neale and Cardon 1992) were fit for the two putative precursors, one for inattentive–hyperactive behavior and one for oppositional behavior. Cholesky decomposition is a saturated model with as many potential underlying additive genetic and environmental influences as measured variables (Loehlin 1996).

In the first series of Cholesky decompositions, the order of the three phenotypes was conduct problems at 4–7 years, oppositional behavior at 4–7 years, and conduct problems at 8–13 years. As illustrated in Fig. 1, the first genetic factor specified ( $A_1$ ) influenced conduct problems at 4–7 years (path coefficient  $a_{11}$ ), oppositional behavior at 4–7 years ( $a_{21}$ ), and conduct problems at 8–13 years ( $a_{31}$ ). The second genetic factor ( $A_2$ ) influenced oppositional behavior at 4–7 years ( $a_{22}$ ) and potentially conduct problems at 8–13 years ( $a_{32}$ ). If path  $a_{32}$  from  $A_2$  to conduct problems at 8–13 years can not be dropped from the model without a significant loss of fit, that would indicate that there are genetic influences on oppositional behavior at 4–7 years that influence conduct problems at 8–13 years that are distinct from the genetic influences on conduct problems at 4–7 years. The shared environmental factors ( $C_1$  and  $C_2$ ) and nonshared environmental factors ( $E_1$  and  $E_2$ ) have same interpretations as  $A_1$  and  $A_2$ , but for these environmental influences. The same tests for pathways from these shared and nonshared environmental factors (i.e., paths  $c_{32}$  and  $e_{32}$  in Fig. 1) were conducted separately for each factor to determine if early oppositional behavior shares environmental influences with conduct problems at 8–13 years that are distinct from the environmental influences on conduct problems at 4–7 years. A second series of models conducted the same tests for the relevant genetic and environmental parameters for inattentive–hyperactive behavior at 4–7 years.

In previous analyses of this sample, we found that variances for conduct problems at all ages were greater for children from blended families (those with at least one half sibling) than non-blended families (all full siblings) and the term for shared environmental influences in non-blended families was nonsignificant, even though a shared environmental influence term specific to blended families was significant (Waldman et al., under review). Therefore, a term for shared environmental influences was specified only for blended families in the present biometric models.

In each of the two series of Cholesky decompositions, omnibus tests were conducted to determine if there were sex differences in the structure of genetic and environmental influences on developing antisocial behavior. Model 1 (sex equal) constrained all parameters to be equal for females and males. Model 2 (scalar sex differences) constrained all parameters to be equal between sexes, but allowed the total variation/covariation in males to be a scalar multiple of the total variation/covariation in females (Neale and Cardon 1992). If model 2 were the best fitting model, that would imply that the total influence of genetic and environmental influences on conduct problems is proportionally less in one sex than the other. Model 3 (quantitative sex differences) allowed all parameters to vary between sexes, but the underlying factors were the same for both sexes.



**Fig. 1** Illustration of the full Cholesky decomposition model for oppositional behavior at 4–7 years and conduct problems at both 4–7 and 8–13 years. The same model was used for inattentive–hyperactive behavior at 4–7 years, substituting that phenotype for oppositional behavior. Please see text under “Longitudinal Multivariate Behavior Genetic Analyses” for definitions of  $A_1$ – $A_3$ ,  $C_1$ – $C_3$ , and  $E_1$ – $E_3$

Because the distributions of the three phenotypes were non-normal at all ages, models were fitted in Mplus 4.2 using robust maximum likelihood estimation, taking the clustering of children within families into account (Muthén and Muthén 2007). The significance of model parameters was determined by comparing the fit of alternative models. The overall fit of each model was expressed as a means-adjusted Satorra–Bentler  $\chi^2$  and differences between models were tested using the Satorra–Bentler scaled-difference  $\chi^2$  (Satorra and Bentler 2001) and  $\chi^2$  tests. A non-significant change in  $\chi^2$  would indicate that the model with fewer parameters fits the data equally well and would be preferred based on parsimony. Because concerns have been raised about fitting Cholesky models (Carey 2005; Dominicus et al. 2006), we present fit statistics in addition to the Satorra–Bentler  $\chi^2$ . Lower values of the Bayesian information criterion (BIC) reflect better fit and/or greater parsimony. The root mean square error of approximation (RMSEA) reflects the discrepancy between observed and predicted covariances per degree of freedom. Values of RMSEA <0.05 are indicative of a close fit and values <0.08 indicate a good fit to the data (MacCallum et al. 1996).

#### Tests of Sample Bias

The 6,994 offspring used in the present analyses are a subset of the total 11,428 identified CNLSY offspring. Some offspring were excluded from the present analyses because they were too old when assessments of offspring began in 1986 to be assessed at 4–7 years of age. Most excluded youth were too young in 2004 to have been assessed at 8–13 years of age, and some had missing data in one age range due to non-participation. Compared to the 4,437 CNLSY offspring whose data were not used, the offspring who participated in the present analyses were not significantly more likely at  $p < 0.05$  to be boys and did not differ in log family income when the mother was 30 years old, but were more likely to be Hispanic, odds ratio=1.46 (95% confidence interval, 1.32–1.61) and more likely to be African American, odds ratio=1.52 (95% confidence interval, 1.40–1.66). These demographic variables were used as covariates in the predictive regression analyses to estimate population parameters more accurately.

## Results

The means and standard deviations of each measure of child problem behavior at 4–7 years and conduct problems at 8–13 years are presented in Table 2. As is typical for maternal ratings of problem behaviors across these ages (Leve et al. 2005), there was a slight decrease in conduct

problems at the older ages. The Spearman rank correlation between inattentive–hyperactive behavior and oppositional behavior at 4–7 years was  $\rho = 0.55$  ( $n = 3,559$ ,  $p < 0.0001$ ) for boys and  $\rho = 0.52$  ( $n = 3,443$ ,  $p < 0.0001$ ) for girls. The correlation between inattentive–hyperactive behavior and conduct problems at 4–7 years was  $\rho = 0.58$  ( $n = 3,559$ ,  $p < 0.0001$ ) for boys and  $\rho = 0.52$  ( $n = 3,433$ ,  $p < 0.0001$ ) for girls. The correlation between oppositional behavior and conduct problems at 4–7 years was  $\rho = 0.62$  ( $n = 3,560$ ,  $p < 0.0001$ ) for boys and  $\rho = 0.58$  ( $n = 3,433$ ,  $p < 0.0001$ ) for girls. The only significant sex difference in correlations among the measures of disruptive behavior at 4–7 years was between oppositional behavior and conduct problems,  $p < 0.01$ .

#### Prospective Prediction Analyses

##### Formal Predictive Tests

The first set of analyses formally tested the extent to which child conduct problems, inattentive–hyperactive behavior, and oppositional behavior at 4–7 years predict conduct problems at 8–13 years using log-linear regression. As shown in Table 3, when each early putative precursor and early conduct problems were tested in separate regression models, controlling the child's sex, ethnicity (two contrasts for African American versus other and Hispanic versus other), and log family income, each early putative predictor accounted for significant variance in conduct problems at 8–13 years. When early inattentive–hyperactive behavior, oppositional behavior, and conduct problems were all entered simultaneously with the demographic covariates, each behavioral predictor still accounted for significant variance in conduct problems at 8–13 years, although the magnitudes of the beta coefficients for inattentive–hyperactive and oppositional behavior dropped by more than 67%. When interactions between child sex and each of the three early behavioral predictors were tested in this joint model, only the sex-by-early conduct problems interaction was significant,  $\beta = -0.05$ ,  $z = -2.07$ ,  $p < 0.04$ , reflecting a slightly stronger prediction in girls.

##### Explained Variance

In order to provide easily interpreted estimates of effect sizes, correlations between each predictor at 4–7 years and conduct problems at 8–13 years are presented in Table 4. Because the distributions of the behavior problem scores were not normal, Spearman rank correlations were calculated. Sex differences in the magnitudes of all of the predictive zero-order correlations between earlier and later problem behaviors in Table 4 were small, but statistically significant in each case in this large sample.

**Table 2** Means and Standard Deviations of Each Measure of Child Problem Behavior at 4–7 Years and Conduct Problems at 8–13 Years Among Females and Males (*N*=6,466)

|  | Males    |           | Females  |           | Tests of sex differences in means <sup>a</sup> | Test of sex differences in variances <sup>b</sup> |
|--|----------|-----------|----------|-----------|--|---|
|  | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> | $\chi^2$                                       | <i>F</i>  |
| Conduct problems (4–7 years)                 | 2.53     | 2.07      | 2.01     | 1.72      | 130.51   | 1.45  |
| Oppositional behavior (4–7 years)            | 1.73     | 1.39      | 1.61     | 1.34      | 12.87  | 1.07  |
| Inattentive hyperactive behavior (4–7 years) | 1.94     | 1.41      | 1.50     | 1.29      | 184.82   | 1.20  |
| Conduct problems (8–13 years)                | 2.47     | 2.20      | 1.80     | 1.74      | 201.59   | 1.60  |

<sup>a</sup> Tested using Poisson regression; all *p*<0.001

<sup>b</sup> All sex differences in variances *p*<0.001

To reveal the extent to which inattentive–hyperactive and oppositional behaviors independently predicted conduct problems at 8–13 years after the correlation with conduct problems at 4–7 years was taken into account, two sets of partial correlations also were calculated to estimate predictive effect sizes for each predictor at 4–7 years (1) controlling the demographic covariates only, and (2) controlling the demographic covariates and both of the other two behavioral predictors at 4–7 years.

The zero-order Spearman correlations show that conduct problems at 4–7 years explained 28–37% of the variance in conduct problems at 8–13 years. Similarly, inattentive–hyperactive and oppositional behaviors at 4–7 years each explain 17–23% of the variance in conduct problems at 8–13 years. When the demographic covariates were partialled, there was little reduction in the variance in conduct problems at 8–13 years explained by the three predictors. In contrast, when inattentive–hyperactive and oppositional behaviors were tested partialling both the demographic covariates and conduct problems at 4–7 years, oppositional behavior explained only 2% of the variance in conduct problems at 8–13 years in both sexes and inattentive–hyperactive behavior explained 2–4% of the variance in conduct problems at 8–13 years. Thus, inattentive–hyperactive and oppositional behavior each explained little

variance in future conduct problems after early conduct problems were taken into account.

### Genetic and Environmental Influences Shared With Early Oppositional Behavior

#### Tests of Omnibus Sex Differences

The first series of Cholesky decompositions tested for omnibus sex differences in the sharing of genetic and environmental influences among oppositional behavior and conduct problems. As reported in Table 5, the scalar sex-differences model fit significantly better than the model allowing no sex differences, but the quantitative sex-differences model did not fit significantly better than the scalar sex-differences model. Therefore, the scalar sex-differences model was used as the basis for the tests of specific genetic and environmental parameters to provide a standard of reference.

#### Tests of Hypotheses Regarding Specific Genetic and Environmental Parameters

As reported in Table 6, a second series of Cholesky decompositions was conducted to test hypotheses regarding

**Table 3** Formal Tests of Mean Inattentive–Hyperactive Behavior, Oppositional Behavior, and Conduct Problems at 4–7 Years as Predictors of Mean Conduct Problems at 8–13 Years Using Log-Linear Regression in Generalized Estimating Equations

| Predictor at 4–7 Years   | $\beta$ | <i>z</i> | <i>p</i> |
|--|---------|----------|----------|
| Separate models: in which each single predictor <sup>a</sup> and the demographic covariates were entered simultaneously in three separate models |         |          |          |
| Inattentive–hyperactive behavior   | 0.34    | 33.82    | <0.0001  |
| Oppositional behavior  | 0.36    | 35.74    | <0.0001  |
| Conduct problems   | 0.39    | 36.74    | <0.0001  |
| Single model: in which all three early predictors and demographic covariates were entered simultaneously in one model                            |         |          |          |
| Inattentive–hyperactive behavior   | 0.11    | 8.84     | <0.0001  |
| Oppositional behavior  | 0.10    | 7.75     | <0.0001  |
| Conduct problems   | 0.27    | 20.36    | <0.0001  |

<sup>a</sup> Each predictor was converted to a standardized *z* score to facilitate comparisons.

**Table 4** Spearman Correlations and Partial Spearman Correlations Between Each Problem Behavior at 4–7 Years and Conduct Problems at 8–13 Years Among Females and Males in the Full Sample

|  | Spearman correlations (and $r^2$ ) | Partial Spearman correlations (and $r^2$ ): Demographic covariates | Partial Spearman correlations (and $r^2$ ): demographic covariates and conduct problems at 4–7) |
|--|------------------------------------|--|---|
| Conduct problems (4–7 years)                 |                                    |  |   |
| Males <sup>a</sup>                           | 0.61 <sup>b</sup> (0.37)           | 0.58 (0.34)  | –   |
| Females                                      | 0.53 (0.28)                        | 0.52 (0.27)  |   |
| Oppositional behavior (4–7 years)            |                                    |  |   |
| Males  | 0.45 (0.20)                        | 0.45 (0.20)  | 0.14 (0.02)   |
| Females                                      | 0.41 (0.17)                        | 0.41 (0.17)  | 0.16 (0.02)   |
| Inattentive–hyperactive behavior (4–7 years) |                                    |  |   |
| Males  | 0.48 (0.23)                        | 0.46 (0.21)  | 0.19 (0.04)   |
| Females                                      | 0.41 (0.17)                        | 0.38 (0.14)  | 0.16 (0.02)   |

<sup>a</sup> Sex differences in correlations between each measure of early problem behavior were tested by testing the sex-by-early predictor in Poisson regression

<sup>b</sup> All correlations and partial correlations significantly different from zero,  $p < 0.0001$

specific genetic and environmental pathways from oppositional behavior at 4–7 years to conduct problems at 8–13 years. The base model for this series was Model 2 (scalar sex differences) in Table 5. Because pathway  $a_{33}$  from genetic factor  $A_3$  (see Fig. 1) was estimated to be 0 for both sexes in model 2, we first determined if  $a_{33}$  (unique genetic influences on conduct problems at 8–13 years) could be dropped by eliminating pathway  $a_{33}$  from model 2. The lack of a significant Wald  $\chi^2$  between model 3 and model 2 indicated that  $a_{33}$  could be dropped without significant loss in fit, indicating that model 2 was preferred on the basis of parsimony. Therefore, the key hypotheses regarding the specific pathways relevant to the sharing of genetic and environmental influences between the putative developmental precursor of oppositional behavior at 4–7 years and conduct problems at 8–13 years ( $a_{32}$ ,  $c_{32}$ , and  $e_{32}$ ) were tested by comparing the fits of models 5, 6, and 7 (in which specific pathways were dropped) to model 2.

In model 5, parameter  $a_{32}$  (genetic influences shared by oppositional behavior and later conduct problems, separate from genetic factor  $A_1$ ) was dropped from model 2 (along

with  $a_{33}$ ). Comparison of model 5 to model 2 revealed that dropping genetic parameter  $a_{32}$  did not result in a statistically significant decrease in fit, indicating that model 5 was preferred. BIC for model 5 was slightly lower than for model 2, reflecting its greater parsimony.

In model 6, parameter  $c_{32}$  (shared environmental influences shared by oppositional behavior and later conduct problems, separate from shared environmental factor  $C_1$ ) was dropped from model 2 (along with  $a_{33}$ ). Comparison of model 6 with model 2 revealed that it was possible to drop  $c_{32}$  without significant loss in fit, indicating that model 6 was preferred. BIC for model 6 was slightly lower than for model 2, reflecting its greater parsimony.

In model 7, parameter  $e_{32}$  (nonshared environmental influences shared by oppositional behavior and later conduct problems, separate from nonshared environmental factor  $E_1$ ) was dropped from model 2 (along with  $a_{33}$ ). Comparison with model 4 revealed that was possible to drop the nonshared environmental parameter  $e_{32}$  without significant loss in fit, indicating that model 7 was preferred.

**Table 5** Results of Multivariate Cholesky Decompositions Testing for Sex Differences in the Sharing of Genetic and Environmental Influences Among Oppositional Behavior and Conduct Problems at 4–7 Years and Conduct Problems 8–13 Years

|  | SB $\chi^2$ | $df$ | BIC     | RMSEA | SB $\Delta\chi^2$ | $df$ | $P$     |
|--|-------------|------|---------|-------|-------------------|------|---------|
| Model 1 (no sex differences)           | 858.66      | 282  | 113,544 | 0.068 |                   |      |         |
| Model 2 (scalar sex differences)       | 574.71      | 279  | 113,115 | 0.049 | 100.73            | 3    | <0.0001 |
| Model 3 (quantitative sex differences) | 567.84      | 264  | 113,189 | 0.051 | 18.9              | 15   | n.s.    |

Model 2 (scalar sex differences) constrained all parameters to be equal between sexes, but allowed the total variation/covariation in males to be a scalar multiple of the total variation/covariation in females. Model 3 (quantitative sex differences) allows quantitative sex differences in the structure of genetic and environmental influences. The Satorra–Bentler scaled-difference  $\chi^2$  is based on a comparison of each model with the model above it.

SB  $\chi^2$  Satorra–Bentler chi-square, SB  $\Delta\chi^2$  Satorra–Bentler scaled-difference  $\chi^2$ ,  $df$  degrees of freedom, BIC Bayesian information criterion, RMSEA root mean square error of approximation. Model 1 (No Sex Differences) constrained all parameters to be equal between sexes.

**Table 6** Results of Multivariate Cholesky Decompositions Testing Specific Parameters in the Sharing of Genetic and Environmental Influences Among Oppositional Behavior and Conduct Problems at 4–7 Years and Conduct Problems 8–13 Years

|                                       | SB $\chi^2$ | df  | BIC     | RMSEA | Wald test | df | P    |
|---------------------------------------|-------------|-----|---------|-------|-----------|----|------|
| Model 2 (full model)                  | 574.71      | 279 | 113,115 | 0.049 |           |    |      |
| Model 4 (drop $a_{33}$ )              | 576.77      | 280 | 113,106 | 0.049 | 2.63      | 2  | 0.27 |
| Model 5 (drop $a_{33}$ and $a_{32}$ ) | 577.33      | 281 | 113,098 | 0.049 | 0.18      | 2  | 0.92 |
| Model 6 (drop $a_{33}$ and $c_{32}$ ) | 576.39      | 281 | 113,100 | 0.049 | 0.65      | 2  | 0.72 |
| Model 7 (drop $a_{33}$ and $e_{32}$ ) | 576.71      | 281 | 113,101 | 0.049 | 0.76      | 2  | 0.68 |

All models allow for scalar sex differences. Pathway labels are shown in Fig. 1. Model 2 is the full model illustrated in Fig. 1. Model 4 is the same as the full model but dropping  $a_{33}$ , which was estimated to be 0. The fits of models 5–7 are compared to model 4. In model 5, genetic pathways  $a_{32}$  and  $a_{33}$  were dropped. In model 6, genetic pathway  $a_{33}$  and environmental pathway  $c_{32}$  were dropped. In model 7, genetic pathway  $a_{33}$  and environmental pathway  $e_{32}$  were dropped. The Satorra–Bentler scaled-difference  $\chi^2$  is based on a comparison of each model with model 4. *SB  $\chi^2$*  Satorra–Bentler chi-square, *SB  $\Delta\chi^2$*  Satorra–Bentler scaled-difference  $\chi^2$  df degrees of freedom, *BIC* Bayesian information criterion, *RMSEA* root mean square error of approximation

Again, BIC for model 7 was lower than for model 2, reflecting its greater parsimony.

Squared standardized coefficients for each path in model 4 are presented in Fig. 2. Based on the comparative tests of the alternative models reported in Table 6, paths  $a_{32}$ ,  $c_{32}$ , and  $e_{32}$  were omitted from Fig. 2. Path  $e_{33}$  was not omitted even though its 95% confidence interval included 0 because it includes the estimate of error in conduct problems at 8–13 years. Overall, Fig. 2 shows that genetic factor  $A_1$  robustly influences conduct problems at both ages ( $a_{11}$  and  $a_{31}$ ) and early oppositional behavior ( $a_{21}$ ). Indeed, there were no genetic influences on conduct problems at 8–13 years that were not shared with earlier conduct

problems. In contrast, genetic factor  $A_2$ , which also influences early oppositional behavior, did influence later conduct problems. There also appeared to be very limited common environmental influence on early and later problem behaviors.

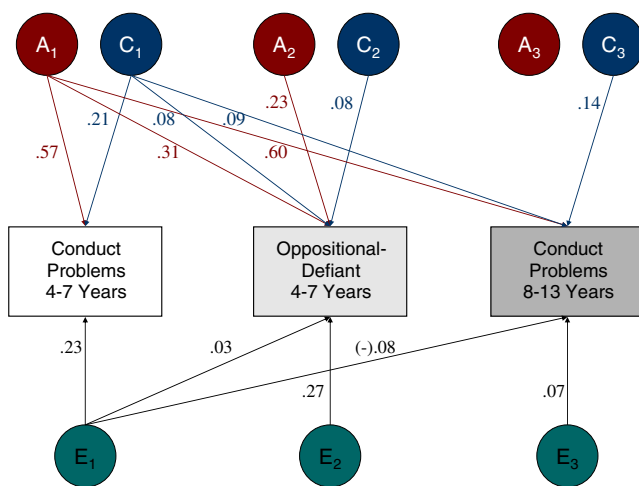
Genetic and Environmental Influences Shared With Early Inattentive–Hyperactive Behavior

Tests of Omnibus Sex Differences

As shown in Table 7, the scalar sex-differences model fit significantly better than the model allowing no sex differences, but the quantitative sex-differences model fit significantly better than the scalar sex-differences model. Therefore, the quantitative sex-differences model was used as the basis for the tests of specific genetic and environmental parameters. As a result, separate path coefficients for the two sexes are presented in Fig. 3.

Tests of Hypotheses Regarding Specific Genetic and Environmental Parameters

The base model for the series of Cholesky decompositions to test hypotheses regarding specific genetic and environmental pathways from inattentive–hyperactive behavior at 4–7 years to conduct problems at 8–13 years was model 3 in Table 7. Because pathway  $a_{33}$  from genetic factor  $A_3$  (see Fig. 1) was estimated to be essentially 0 for both sexes, we first determined if  $a_{33}$  (unique genetic influences on conduct problems at 8–13 years) could be dropped from Model 3 for inattentive–hyperactive behavior. The non-significant Wald  $\chi^2$  (Satorra and Bentler 2001) between model 3 and model 4 indicated that  $a_{33}$  could be dropped without significant loss in fit. Therefore, model 4 was preferred on the basis of parsimony, which is reflected in the slightly lower BIC for model 4 than for model 3. Thus,



**Fig. 2** Squared standardized coefficients for each path in Model 4 for females and males together in a sex-scalar model. Path  $a_{33}$  was set to 0 in this model; paths were omitted for each sex if their 95% confidence interval included 0 for that sex. The coefficient for path  $e_{33}$  is shown even though its 95% confidence interval included 0 because it includes the estimate of error in conduct problems at 8–13 years. Please see text under “Longitudinal Multivariate Behavior Genetic Analyses” for definitions of  $A_1$ – $A_3$ ,  $C_1$ – $C_3$ , and  $E_1$ – $E_3$

**Table 7** Results of Multivariate Cholesky Decompositions Testing for Sex Differences in the Sharing of Genetic and Environmental Influences Among Inattentive–Hyperactive Behavior and Conduct Problems at 4–7 Years and Conduct Problems 8–13 Years

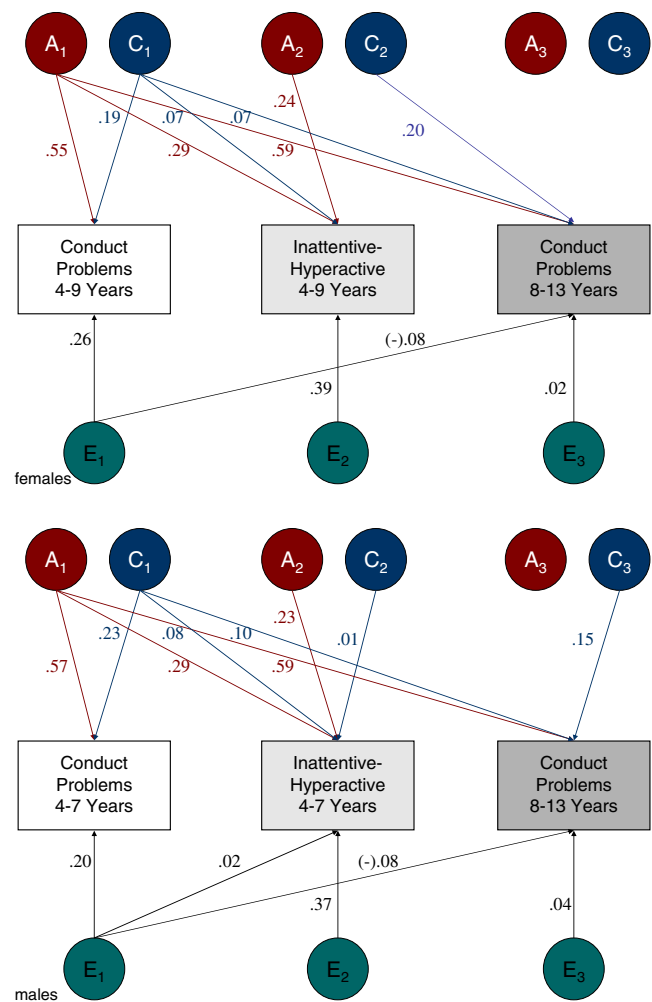
|  | SB $\chi^2$ | df  | BIC     | RMSEA | SB $\Delta\chi^2$ | df | p      |
|--|-------------|-----|---------|-------|-------------------|----|--------|
| Model 1 (no sex differences)           | 910.40      | 282 | 113,938 | 0.083 |                   |    |        |
| Model 2 (scalar sex differences)       | 631.91      | 279 | 113,517 | 0.064 | 101.11            | 3  | <0.001 |
| Model 3 (quantitative sex differences) | 585.50      | 264 | 113,550 | 0.061 | 42.00             | 15 | <0.001 |

Model 1 (no sex differences) constrained all parameters to be equal between sexes. Model 2 (scalar sex differences) constrained all parameters to be equal between sexes, but allowed the total variation/covariation in males to be a scalar multiple of the total variation/covariation in females. Model 3 (quantitative sex differences) allows quantitative sex differences in the structure of genetic and environmental influences. The Satorra–Bentler scaled-difference  $\chi^2$  is based on a comparison of each model with the model above it. SB  $\chi^2$  Satorra–Bentler chi-square, SB  $\Delta\chi^2$  Satorra–Bentler scaled-difference  $\chi^2$  df degrees of freedom, BIC Bayesian information criterion, RMSEA root mean square error of approximation

the hypotheses regarding specific pathways relevant to the sharing of genetic and environmental influences between the putative developmental precursor of inattentive–hyperactive behavior at 4–7 years and conduct problems at 8–13 years ( $a_{32}$ ,  $c_{32}$ , and  $e_{32}$ ) were tested by comparing models with each dropped pathway to model 4 (from which  $a_{33}$  was dropped).

In model 5, parameter  $a_{32}$  (genetic influences shared by inattentive–hyperactive behavior and later conduct problems, separate from genetic factor  $A_1$ ) was dropped (along with  $a_{33}$ ). As shown in Table 8, the comparison of model 5 to model 4 showed that it was possible to drop genetic parameter  $a_{32}$  without a significant decrease in fit. Correspondingly, the BIC for the more parsimonious model 5 was slightly smaller than for model 4. In model 6, parameter  $c_{32}$  (shared environmental influences shared by inattentive–hyperactive behavior and later conduct problems, separate from shared environmental factor  $C_1$ ) was dropped (along with  $a_{33}$ ). Comparison of model 6 with model 4 revealed that it was not possible to drop parameter  $c_{32}$  (shared environmental influences in blended families that are common to both inattentive–hyperactive behavior at 4–7 years and to conduct problems at 8–13 years) without a significant loss in fit. This reflects the sex difference in paths from  $C_2$ . As shown in the two panels of Fig. 3, either  $c_{22}$  or  $c_{32}$  is negligible for one of the sexes. Either way, the implication is that there are no appreciable shared environmental influences common to both inattentive–hyperactive behavior at 4–7 years and conduct problems at 8–13 years independent of conduct problems at 4–7 years.

In model 7, parameter  $e_{32}$  (nonshared environmental influences shared by inattentive–hyperactive behavior and later conduct problems, separate from nonshared environmental factor  $E_1$ ) was dropped (along with  $a_{33}$ ). Comparison of model 7 with model 4 revealed that it was possible to drop  $e_{32}$  without significant loss in fit. BIC for model 7 was slightly lower than for model 4, reflecting its greater parsimony.



**Fig. 3** Squared standardized coefficients for each path in model 4 for females (top) and males (bottom). Path  $a_{33}$  was set to 0 in this model; paths were omitted for each sex if their 95% confidence interval included 0 for that sex. The coefficient for path  $e_{33}$  is shown even though its 95% confidence interval included 0 because it includes the estimate of error in conduct problems at 8–13 years. Please see text under “Longitudinal Multivariate Behavior Genetic Analyses” for definitions of  $A_1$ – $A_3$ ,  $C_1$ – $C_3$ , and  $E_1$ – $E_3$

Squared standardized coefficients for each path in model 4 for each sex are presented in Fig. 3, except that parameters were omitted for each sex when their 95% confidence intervals included 0 for that sex. Coefficient  $a_{32}$  was not presented in Fig. 3 for either sex because it could be dropped without significant loss in fit and its 95% confidence intervals included 0 for both sexes. Path  $e_{33}$  was not omitted from Fig. 3 even though its 95% confidence interval included 0 because it includes the estimate of error in conduct problems at 8–13 years.

**Discussion**

As in many previous studies (Lahey et al. 2000; Lilienfeld and Waldman 1990), conduct problems at 8–13 years were significantly predicted by both inattentive–hyperactive and oppositional behaviors at 4–7 years in the present sample. This suggests that the measures of these putative precursors used in the CNLSY are prospectively related to conduct problems at 8–13 years in the much same way as the various measures of these constructs used in other studies. When conduct problems at 4–7 years were added as a simultaneous predictor to the prediction models, however, inattentive–hyperactive and oppositional behaviors at 4–7 years each accounted for very little independent variance in later conduct problems. This suggests that knowing that young children exhibit inattentive–hyperactive or oppositional behaviors adds very little to our ability to predict conduct problems in middle and late childhood after early conduct problems are considered.

In addition, the longitudinal multivariate behavior genetic analyses revealed that the same genetic and environmental factors that influence conduct problems at both 4–7 and 8–13 years also influence the putative early precursors at 4–7 years. After the genetic and environmental influences that the putative precursors share with early conduct problems

(genetic factor  $A_1$ ) were taken into account, however, inattentive–hyperactive and oppositional behavior each shared little or no additional genetic influences with later conduct problems (genetic factor  $A_2$ ). This suggests that, after early conduct problems are considered, early inattentive–hyperactive and oppositional behavior do not function as useful early manifestations of additional genetic and environmental influences that later give rise to conduct problems in middle and late childhood.

These findings argue for a reconceptualization of the early childhood manifestations of later conduct problems. Prospective longitudinal studies beginning in early childhood have revealed that young children engage in serious conduct problems to a much greater extent than previously suspected (Keenan et al. 2007; Tremblay 2004). This is important, as the present findings reveal that the best indicator that a child will exhibit conduct problems at 8–13 years is his or her conduct problems at 4–7 years. Indeed, early inattentive–hyperactive and oppositional behaviors are almost entirely redundant predictors of later conduct problems after early conduct problems are controlled. Thus, inconsistent with their hypothesized role as developmental precursors, early inattentive–hyperactive and oppositional behaviors do not function as informative early behavioral manifestations of future conduct problems after early conduct problems are considered. Similarly, the longitudinal Cholesky models revealed that early inattentive–hyperactive and oppositional behaviors only minimally reflect the same genetic and environmental influences that will give rise to later conduct problems after the genetic and environmental influences on early conduct problems are considered. Thus, little is gained by viewing inattentive–hyperactive and oppositional behaviors as developmental precursors to later conduct problems. Rather than focusing on early inattentive–hyperactive or oppositional behavior, early interventions designed to prevent conduct problems in late childhood should focus on children who already exhibit conduct problems in early childhood.

**Table 8** Results of Multivariate Cholesky Decompositions Testing Specific Parameters in the Sharing of Genetic and Environmental Influences Among Inattentive–Hyperactive Behavior and Conduct Problems at 4–7 Years and Conduct Problems 8–13 Years

|  | SB $\chi^2$ | df  | BIC     | RMSEA | Wald test | df | p      |
|--|-------------|-----|---------|-------|-----------|----|--------|
| Model 3 (full model)                               | 585.50      | 264 | 113,550 | 0.053 |           |    |        |
| Model 4 (drop $a_{33}$ )                           | 589.75      | 266 | 113,533 | 0.053 | 0.83      | 2  | 0.66   |
| Model 5 (drop $a_{32}$ and $a_{33}$ )              | 593.31      | 268 | 113,521 | 0.053 | 3.38      | 2  | 0.18   |
| Model 6 (drop $a_{33}$ and $c_{32}$ ) <sup>a</sup> | 644.43      | 268 | 113,563 | 0.057 | 64.0      | 2  | <0.001 |
| Model 7 (drop $a_{33}$ and $e_{32}$ )              | 596.91      | 268 | 113,530 | 0.053 | 2.9       | 2  | 0.22   |

All models allow for quantitative sex differences. Pathway labels are shown in Fig. 1. Model 3 is the full model illustrated in Fig. 1. Model 4 is the same full model but dropping  $a_{33}$ , which was estimated to be 0. The fits of models 5–7 are compared to model 4. In model 5, genetic pathways  $a_{32}$  and  $a_{33}$  were dropped. In model 6, genetic pathway  $a_{33}$  and environmental pathway  $c_{32}$  were dropped. In model 7, genetic pathway  $a_{33}$  and environmental pathway  $e_{32}$  were dropped.

SB  $\chi^2$  Satorra–Bentler chi-square, SB  $\Delta\chi^2$  Satorra–Bentler scaled-difference chi-square, df degrees of freedom, BIC Bayesian information criterion, RMSEA root mean square error of approximation

<sup>a</sup> A Wald test indicates that dropping  $c_{32}$  from the model results in a significant loss in fit.

This is not to say that inattentive–hyperactive and oppositional behaviors are unimportant, however. Each is a significant dimension of psychopathology in its own right that is associated with different forms of functional impairment (August and Garfinkel 1993; Lahey et al. 2004a, b). The present findings are consistent with previous cross-sectional studies of shared genetic and environmental influences on ADHD, ODD, and conduct problems (Burt et al. 2005; Dick et al. 2005; Nadder et al. 2002; Waldman et al. 2001) in revealing that inattentive–hyperactive behaviors, oppositional behaviors, and conduct problems at 4–7 years are all influenced to a substantial extent by the same genetic and environmental influences. Nonetheless, Figs. 2 and 3 indicate that inattentive–hyperactive and oppositional behaviors each have some unique genetic ( $a_{22}$ ) and environmental influences ( $c_{22}$  for males and  $a_{22}$  for both sexes).

#### Limitations and Future Research

Like most previous longitudinal studies of the predictive associations of inattentive–hyperactive and oppositional behaviors with later conduct problems (Hechtman et al. 1984; Lambert 1988; Loney et al. 1981; Magnusson 1984; Mannuzza et al. 1993; Mannuzza et al. 1991), the present analyses were not based on DSM measures of ADHD or ODD. The present findings were quite consistent with the smaller set of previous studies that did use DSM-based measures in terms of the predictive associations found between inattentive–hyperactive and oppositional behaviors and later conduct problems (Biederman et al. 1996; Lahey et al. 2000; Loeber et al. 1995; Rowe et al. 2002), however. This suggests that the present findings are relevant to DSM conceptualizations of these disorders. Nonetheless, it is important to note that different results might have been found had DSM measures been used. For this reason, longitudinal studies of large representative samples using DSM-based measures would be very useful.

It also will be very important for future studies to examine links between early inattentive–hyperactive and oppositional behaviors and delinquency during adolescence, as relations between adolescent delinquency and early problem behaviors could be quite different. In particular, the present findings do not rule out the possibility that inattentive–hyperactive and oppositional behaviors could play roles as developmental precursors to childhood-onset delinquency (Moffitt 1993, 2006). Because childhood-onset delinquency is characterized by high levels of early child conduct problems, however, it is possible that early inattentive–hyperactive and oppositional behaviors do not independently predict childhood-onset delinquency trajectories after early conduct problems are controlled. Fortunately, it will be possible to test this hypothesis when enough

children in the present sample have completed assessments from early childhood through adolescence in the future.

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