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Developmental Structure of Genetic Influences on Antisocial Behavior Across Childhood and Adolescence

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Abstract

It is necessary to determine if causal influences on developing antisocial behavior change with age to guide both research and theory on its origins. We estimated the extent to which the same genetic factors influence antisocial behavior across 4–17 years of age using 2,482 sibling pairs of varying genetic relatedness. Assessments of antisocial behavior reflected the changing validity of informants across development: Mothers (4–9 years), mothers and youth (10–13 years), and youth (14–17 years). Genetic influences on antisocial behavior at 14–17 years were entirely shared with those on antisocial behavior at 10–13 years according to both informants. Genetic influences on antisocial behavior at 14–17 years were distinct from those at 4–9 years, however. These age differences in genetic influences cannot be fully distinguished from informant differences across age, but the present findings indicate that youth reported to be persistently antisocial during childhood and adolescence are influenced by one set of genetic factors influencing parent-report conduct problems in childhood and a second set of genetic influences on youth-reported delinquency that come into play around the time of the pubertal transition.

Keywords

Antisocial behavior; genetics; environmental influences; developmental change

Because juvenile crime is a serious public health problem (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998), identifying its genetic and environmental causal influences is a high stakes issue. A meta-analytic review concluded that there are moderate genetic influences, modest shared environmental influences (which make siblings more similar), and substantial nonshared environmental influences (which make siblings less similar plus

measurement error) on antisocial behavior (Rhee & Waldman, 2002). Only small differences in heritability were found between childhood and adolescence, but much remains to be learned about the developmental structure of genetic and environmental influences on antisocial behavior. First and foremost, because different genes can be expressed at different ages, and different environments can be most salient at different ages (Plomin, 1986), it is essential to learn if the same or different causal influences on developing antisocial behavior operate at different ages. This information is needed to design optimal studies of the specific genes and environments that initiate and sustain antisocial behavior.

Moffitt proposed an influential model of antisocial behavior based on hypothesized developmental differences in causal influences (Moffitt, 1993, 2003, 2006). In her model, life-course persistent (LCP) delinquents exhibit persistent antisocial behavior from childhood into adulthood. LCP offending is hypothesized to arise in early childhood when neuropsychological deficits manifested as difficult temperament, cognitive deficits, and attention-deficit/hyperactivity disorder evoke maladaptive parenting. In contrast, adolescent-limited (AL) delinquency emerges in youth without histories of significant neuropsychological deficits or childhood conduct problems as the result of deviant peer influences during adolescence. Moffitt (1993) hypothesized that genetic factors play a greater role in LCP than in AL delinquency, but did not describe the mechanisms involved. One possibility is that genetic influences on LCP delinquency are mediated by the early neuropsychological deficits that give rise to childhood conduct problems.

Lahey and Waldman (2003) explicitly hypothesized that genetic influences on adolescent delinquency are entirely shared with (mediated by) those on earlier childhood conduct problems. The present analyses test this hypothesis for genetic influences on adolescent delinquency. To the extent that Moffitt's (1999) model implies that genetic influences on LCP delinquency are mediated by early deviant behavior, the test of Lahey and Waldman's mediation hypothesis also will shed light on Moffitt's model.

Four previous longitudinal studies of genetically informative samples have reported findings that are relevant to the genetic mediation hypothesis. Three of these studies found that genetic influences on mother-reported conduct problems in late childhood or early adolescence were shared with those on mother-reported conduct problems in earlier childhood. These studies spanned the ages of 4–10 years (Van den Oord & Rowe, 1997), 3–12 years (Bartels et al., 2004), and 8–14 years (Eley, Lichtenstein, & Moffitt, 2003). In contrast, a fourth study found little sharing of causal influences between mother-reported conduct problems at 8–9 years and youth-reported delinquency at age 13–14 years (Tuvblad, Eley, & Lichtenstein, 2005). Unfortunately, none of these studies addressed the sharing of causal influences between antisocial behavior in childhood and in *late* adolescence. This is an important omission because criminal offending reaches a sharp peak in late adolescence then declines (Farrington, 1991).

Within the limits of the age ranges studied, only the Tuvblad et al. (2005) study did not support Lahey and Waldman's (2003) mediation hypothesis. It is tempting to dismiss the findings of Tuvblad et al. study as an artifact of using different informants on antisocial behavior at different ages (mothers in childhood and youth in early adolescence). Although informant differences are an important issue that must be addressed, it is essential not to ignore Tuvblad et al.'s findings on this basis. Because there are marked variations in the validity of informants on antisocial behavior at different ages, there are currently no viable alternatives to using parents and other adults as informants on antisocial behavior during early childhood and youth as informants during late adolescence. This is because children younger than 9 years are not reliable reporters on their behavior in interviews (Schwab-Stone, Fallon, Briggs, & Crowther, 1994) and adolescents successfully conceal most of their antisocial behavior from adults (Loeber et al., 1998). Although it would be possible to use parent reports at all ages from early

childhood through late adolescence, that would seriously underestimate the prevalence of delinquency during adolescence and lead to invalid conclusions.

Therefore, using current methods, data from different informants at different ages must be incorporated if we are to understand the developmental structure of causal influences on antisocial behavior from early childhood through adolescence. For example, the empirical basis for Moffitt's model is a longitudinal study that assessed child conduct problems during early childhood using adult informants and delinquency during adolescence using youth reports (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). This is of central importance to the present analyses because it means that LCP delinquents are defined on the basis of antisocial behavior that persists across *both* informants and age. That is, LCP delinquents, whose public health impact is even greater than AL delinquents (Moffitt et al., 1996; Moffitt, Caspi, Harrington, & Milne, 2002; Odgers et al., 2007), exhibit *both* adult-reported conduct problems in childhood and youth-reported delinquency during adolescence.

Therefore, it is essential to understand the structure of causal influences on antisocial behavior across development and informants. If Tuvblad et al.'s (2005) findings are replicated across a broader age range, that would mean that the genetic influences on mother-reported child conduct problems and youth-reported adolescent delinquency are largely different. This would imply that at least two different sets of genetic influences are involved in LCP delinquency—one operating in childhood and one operating during adolescence. Because of the importance of this possibility to both theory and the design of genetic research, the present study attempts to replicate and extend Tuvblad et al. by examining shared causal influences both within and across informants over 4–17 years of age in a representative U.S. sample.

METHOD

Participants

Mother Generation—The National Longitudinal Survey of Youth (NLSY79) was funded by the Bureau of Labor Statistics to study the U.S. workforce. A nationally-representative household sample of 9,763 14–22 year old male and female youth not in the military was selected for the NLSY79, with an oversample of African American and Hispanic youth. To date, 4,926 NLSY79 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. The response rate for the initial NLSY79 assessment was 90%. Participants were re-interviewed annually from 1979 through 1994 and biennially since then. Retention rates for the NLSY79 during follow-up assessments were $\geq 90\%$ during the first 16 waves and $> 80\%$ in subsequent waves.

Offspring Generation—The present analyses are based on the offspring of the NLSY79 females. Biennial assessments of all biological offspring of each NLSY79 woman began in 1986 (Chase-Lansdale, Mott, Brooks-Gunn, & Phillips, 1991). Participation in the 10 assessments through 2006 averaged $\approx 90\%$. For budgetary reasons, delinquency items were not included in the assessments of youth who were 15 years or older in 2000. A total of 11,428 offspring had participated by 2004. In order to conduct longitudinal analyses on the same youth assessed at different ages, the present analyses were based on 3,259 youth (23% Hispanic, 35% African American, 42% non-African American, non-Hispanic) for whom data on antisocial behavior were available from multiple longitudinal assessments in each range: ≥ 1 assessment during 4–9 years, ≥ 2 assessment during 10–13 years, and ≥ 1 assessment during 14–17 years. Because the assessments were conducted biennially, youth were almost always assessed at either an even or an odd year of age, but not both (e.g., at 10 or 11 years of age).

Measures

Mothers rated their 4–13 year old children using the Behavior Problem Index (Peterson & Zill, 1986). BPI items were selected from the Child Behavior Checklist (Achenbach, 1978) based on correlations with factor scores (Peterson & Zill, 1986). Seven BPI items rated on a 3-point scale defined childhood conduct problems: 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; and does not seem to feel sorry after misbehaving (median Cronbach's α over 4–13 years = .74). These items overlap substantially with items used in previous longitudinal studies to define child conduct problems (Fergusson & Horwood, 2002; Moffitt, Caspi, Rutter, & Silva, 2001). Furthermore, confirmatory factor analysis of the BPI externalizing items in the present sample confirmed this measure of child conduct problems, by indicating that a three-factor model, specifying correlated latent factors for conduct problems, oppositional behaviors (argues too much; is stubborn, sullen, or irritable; and has a very strong temper and loses it easily), and inattentive-hyperactive behaviors, fit better than a one-factor solution or a two-factor solution that constrained the items for conduct and oppositional problems to load on the same factor (D'Onofrio et al., 2008). The validity of this conduct problem score has been confirmed by demonstrating that it significantly predicts future criminal convictions in adolescence in both sexes (Lahey et al., 2006).

In each assessment wave, youth aged 10–14 years (except 14-year olds who turned 15 by the end of the calendar year) completed the Child Self-Administered Supplement (CSAS). In a self-completion format, youth reported on their engagement in 7 delinquent behaviors in the past 12 months from the Self-reported Delinquency (SRD) interview (Elliott & Huizinga, 1983): hurting someone on purpose, lying to parents, shoplifting, vandalism at school, skipping school, staying out all night without permission, and a parent being called to school because of the child's misbehavior (median Cronbach's α was .69). Responses were scored 0 (never), 1 (once) or 2 (more than once). The SRD is the benchmark measure in delinquency research (Loeber et al., 1998; Moffitt et al., 1996).

Youth who were 15 years or older (including 14 year olds who turned 15 during the calendar year) were administered the Young Adult instrument (YA) instead of the CSAS. Using the same private self-completion format, youth reported on the same 7 delinquency items, scored 0 = no; 1 = yes. The only difference was the age-appropriate substitution in the YA of an item on running away from home for the CSAS item on staying out all night without permission. Across ages 14–17, median α in the CNLSY for the 7-item delinquency scale was .65.

In 1994–1998 only, the YA included 10 additional items from the SRD: gotten into a fight at school or work; took things other than from a store; took something worth <\$50; took something worth $\geq$ \$50; used force to get money or things from someone; hit or seriously threatened to hit someone; attacked someone with the idea of seriously hurting or killing them; tried to con someone; took a vehicle without the owner's permission; broke into a building or vehicle; and knowingly held or sold stolen goods. These items are not included in the present analyses, but were used to assess the validity of the 7-item delinquency scale in a previous report. The 7-item SRD delinquency was found to correlate with the additional 10 SRD items in the CNLSY at $r = .81$ (Lahey et al., 2006). Thus, the 7-item delinquency scale, which was used in all analyses, provides a robust estimate of a broad range of serious delinquent behavior. The validity of 7-item delinquency scale was further support by demonstrating that it significantly predicts criminal convictions in adolescence in both sexes (Lahey et al., 2006).

Kinship Links

Youth who were assessed during each age group and who had at least one sibling with complete data were included in the behavior genetic analyses (Table 1). Because only biological children

of NLSY79 mothers participated, all CNLSY siblings are at least half siblings. Algorithms 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 (Van Hulle, Rodgers, D'Onofrio, Waldman, & Lahey, 2007). The kinship links were validated by demonstrating that the heritability of physical height is .90, consistent with twin studies. Because ambiguous siblings are either full or half siblings, they were assigned a genetic coefficient of $r_g = .375$, intermediate between full ($r_g = .5$) and half siblings ($r_g = .25$). 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. Sibling pairs were divided into families with only full siblings (non-blended) and families in which there was at least one half or ambiguous sibling pair (blended) based on analyses of CNLSY data that indicated that there are shared environment influences specific to blended families (Waldman et al., 2007).

Statistical Analyses

For each informant, the antisocial behavior scores at each age were converted to age-standardized z-scores. Approximately 70% of siblings in the present analyses were ≥ 3 years apart in age, and previous analyses on mother- and youth-reported antisocial behavior in the CNLSY indicated small but significant secular trends in mean levels of antisocial behavior (Lahey et al., 2006). Therefore, all z-scores were regressed on year of birth prior to analyses to minimize any attenuation of correlations between siblings created by differential effects of secular trends on siblings of different ages. A sandwich estimator was used to account for the clustering of siblings within families (Rogers, 1993).

Using standard biometric models (Neale & Cardon, 1992), total observed variance in each measure of antisocial behavior was partitioned into unstandardized estimates of variance attributable to latent additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences (E). Magnitudes of A, C, and E were estimated from differences in correlations between sibling pairs of differing genetic relatedness.

To determine if genetic and environmental influences on antisocial behavior during adolescence were shared with those on antisocial behavior at earlier ages, we fit two series of multivariate Cholesky decompositions (Neale & Cardon, 1992). The developmentally ordered phenotypes were antisocial behavior during the age ranges when nearly all children are prepubertal (4–9 years), during the ages when most children experience the onset of puberty (divided into 10–11 and 12–13 years to identify possible age-related differences during this period of rapid developmental change), and during the adolescent age range when most children have completed pubertal development (14–17 years). The specific informants interviewed and the measures used in each age range in the two series of models are summarized in Table 2.

Series 1 examined overlapping genetic influences on mother-reported conduct problems across 4–13 years and youth-reported delinquency at 14–17 years. Series 2 examined overlapping genetic influences on mother-reported conduct problems at age 4–9 years and youth-reported delinquency across ages 10–17 years. This distinguished between differences in age and differences in informants during the age range in which both mothers and youth are valid informants using current assessment methods (10–13 years).

In both series of Cholesky models, covariation between antisocial behavior at 14–17 years and antisocial behavior at earlier ages was partitioned into components reflecting the extent to which A, C, and E were shared by the measures of antisocial behavior at the various different ages. The extent to which the correlation between antisocial behavior at 14–17 years and at an earlier age was greater in full than in half or ambiguous siblings indicated the extent to which

antisocial behavior at 14–17 years and antisocial behavior at the earlier age shared genetic influences.

Cholesky decomposition is appropriate for measuring shared causal influences when one phenotype precedes the other in developmental time (Loehlin, 1996). Its logic is illustrated in Figure 1 for four developmentally ordered phenotypes measured at different ages. The additive genetic pathways (from A_1 , A_2 , etc.) illustrated in Figure 1 represent causal pathways from each latent genetic factor to each phenotype. The first genetic factor (A_1) is casually prior to A_2 , such that A_1 is “partialled out” when estimating genetic influences (A_2) on later phenotypes. Similarly, latent genetic factors A_1 and A_2 are both partialled when estimating the effects of A_3 on phenotypes 3 and 4, and so on. The same logic also applies to the two types of environmental influences, C and E.

In both series of Cholesky models, tests were conducted to determine if there were sex differences in the structure of causal influences on developing antisocial behavior. One model allowed all parameters to vary between sexes, but the underlying factors were the same for both sexes. The fit of this “sex-limitation,” or quantitative sex-differences, model was compared to a “scalar sex-differences” model in which all parameters were constrained to be equal in the two sexes, but the total variation/covariation in males was allowed to be a scalar multiple of the total variation/covariation in females (Neale & Cardon, 1992).

Because the distributions of age-standardized antisocial behavior scores were non-normal, models were fitted in Mplus 5.1 using robust maximum likelihood estimation (Muthén & Muthén, 2007). The statistical significance of parameters was determined by comparing the fit of restricted submodels with deleted parameters to the full model. The fit of each model was expressed as a means-adjusted Satorra-Bentler χ^2 and differences between models were tested using the Satorra-Bentler scaled-difference χ^2 (Satorra & Bentler, 2001). A non-significant change in χ^2 indicated that the submodel with fewer parameters fit the data equally well and was preferred based on parsimony. We also estimated the root mean square error of approximation (RMSEA) for each model, which is an estimate of the discrepancy between each model and the true population variance-covariance matrix of the variables. Values of RMSEA < .05 are indicative of a close fit and values < .08 indicate a good fit to the data (MacCallum, Browne, & Sugawara, 1996). In addition, the Bayesian Information Criterion (BIC) (Raftery, 1995) evaluates the fit of each model, including a penalty for the number of parameters, balancing model parsimony with fidelity in representing the observed covariances among the variables in the model. A larger BIC indicates less close fit.

Tests of Sample Bias—The 3,151 offspring in these analyses are a subset of the 11,428 offspring in the entire CNLSY study. Although some offspring were excluded from the analyses because they were too old in 1986 to have the required number of childhood assessments, most offspring were excluded because they were not yet old enough to have the required assessments during adolescence. Other offspring could not be included in the analyses because they had missing data or did not have a participating sibling. Mothers of offspring included in the analyses had more offspring on average than mothers of excluded offspring (3.5 vs. 2.5), were younger at first birth ($M = 21.0$ vs. $M = 24.1$; $t = 18.4$, $p < .0001$), and had lower family incomes at age 30 years ($M = \$27,514$ vs. $M = \$37,203$, $t = 3.6$, $p = .0001$). Equal proportions of female and male offspring were included in the analyses, but a higher percentage of offspring identified by mothers as Hispanic (20% vs. 18%;) and African American (34% vs. 25%;) were in the present analyses relative to excluded youth. Controlling for the clustering of multiple offspring within families, included youth had higher mother-reported conduct problems across ages 4–13 years ($M = 2.36$ vs. $M = 2.19$, $t = 3.4$, $p = .001$), but did not differ significantly on youth-reported delinquent behavior across 10–17 years. Therefore, the present findings generalize to segments of the population with demographic characteristics that place

them at high risk for childhood conduct problems (Loeber et al., 1998), but perhaps not to the full U.S. population of youth.

RESULTS

As reported in Table 3, both means and variances of mother-reported conduct problems and youth-reported delinquent behavior were significantly lower for females than males in each age group. The magnitudes of phenotypic correlations between youth-reported delinquency at 14–17 years and each measure of antisocial behavior at earlier ages reported in Table 3 did not differ significantly between females and males, however.

Univariate Models¹

Because variances for all measures of antisocial behavior at all ages and informants were greater for children from blended than non-blended families, $F = 1.2$ through 1.6 , all $p < .001$, we tested whether different biometric models were needed for the two family types. At all ages and for both informants, C influences in non-blended families were nonsignificant, whereas C specific to blended families was significant. That is, there are shared environmental factors specific to blended families that tend to make youth from such families more similar to one other than would be expected under a strictly additive genetic model. Therefore, C was specified only for blended families in all biometric models.

In addition, the variances of all measures of antisocial behavior for both informants were greater in males than females ($F = 6.1$ through 11.9 , all $p < .001$). Therefore, we fit (1) sex-limitation and scalar sex-differences models (Neale & Cardon, 1992) for each measure. A previous paper based on earlier releases of data from this study ruled out qualitative sex differences (i.e., entirely different genetic and environmental influences on females and males), but indicated scalar sex differences in causal influences on youth-reported delinquency at 14–17 years (Van Hulle et al., 2007).

The best-fitting univariate models and standardized parameter estimates are summarized in Table 4. All models fit the data acceptably as indicated by RMSEAs $\leq .08$. The scalar sex-differences model was the best-fitting model for mother-reported conduct problems at both 4–9 and 10–11 years and for youth-reported delinquency at 10–11, 12–13, and 14–17 years, but the quantitative sex-differences model was the best-fitting model for mother-reported conduct problems at ages 12–13. For each measure of antisocial behavior, the best-fitting sex-differences model was compared to a *sex-equal* model that also constrained the total variance to be the same in males and females. In all cases, the sex-equal model resulted in a significant loss in fit relative to the sex-differences model.

Multivariate Models

Two series of multivariate Cholesky decompositions were conducted to estimate the extent to which genetic and environmental influences on antisocial behavior in adolescence are unique or shared with those on antisocial behavior at earlier ages.

Tests of Shared Causal Influences Across Development

Series 1: In the first series of Cholesky decomposition models, the developmentally ordered phenotypes were mother-reported child conduct problems at 4–9, 10–11, and 12–13 years, and youth-reported delinquency at 14–17 years (Table 5). Models 1–5 examined four latent factors each for additive genetic (A_1 – A_4), shared environmental (C_1 – C_4), and nonshared environmental (E_1 – E_4) influences. The quantitative sex-differences model (Model 1) fit

¹Detailed results of univariate model fitting available from the first author.

significantly better than the scalar sex-differences model (Model 2). This suggests a quantitative sex difference in the magnitudes of shared causal influences, even though the overall pattern of parameter estimates was very similar across sexes.

Models 3–5 tested whether genetic and environmental influences on youth-reported delinquency at 14–17 years were shared with those on mother-reported conduct problems across 4–13 years. These models were compared to Model 1 to assess changes in fit after dropping selected parameters. In Model 3, all paths from genetic factors A_1 – A_3 to youth-reported delinquency at 14–17 years were dropped, but A_4 (genetic influences on adolescent delinquency not shared with conduct problems at earlier ages) and all paths for both types of environmental influences to youth-reported delinquency at 14–17 years were retained. As reported in Table 5, the genetic paths A_1 – A_3 to delinquency at 14–17 years could not be dropped without a significant loss in fit relative to Model 1. In Model 4, all paths from both genetic factors (A_1 – A_4) and nonshared environmental factors (E_1 – E_4) to youth-reported delinquency at 14–17 years were retained, but all paths from shared environmental factors at younger ages (C_1 – C_3) to youth-reported delinquency were dropped. Again, these paths could not be dropped without a significant loss in fit relative to Model 1. In Model 5, all paths from both genetic factors (A_1 – A_4) and shared environmental factors (C_1 – C_4) to youth-reported delinquency at 14–17 years were retained and all paths from nonshared environmental factors E_1 – E_3 for conduct problems at earlier ages to youth-reported delinquency at 14–17 years were dropped. It was possible to drop these paths for nonshared environmental factors without a significant loss in fit relative to Model 1 (Table 5).

The comparison of Model 3 to Model 1 supported the genetic mediation hypothesis (Lahey & Waldman, 2003) that there are additive genetic influences on delinquency at 14–17 years that are shared in common with antisocial behavior at earlier ages. Three additional models were fitted to examine this finding in detail by determining the number of genetic factors needed account for the sharing of genetic influences across 4–17 years. In these models, the structure of shared and non-shared environmental influences was always the same as in Model 1. In Model 6, genetic factors A_1 – A_3 were retained, but genetic factor A_4 (unique genetic influences of youth-reported delinquency at ages 14–17 years) was dropped. There was not a significant loss in fit relative to Model 1 (Table 5), indicating that there were not significant unique genetic influences on youth-reported delinquency in adolescence that were not shared with antisocial behavior at earlier ages. In Model 7, genetic factors A_1 and A_2 were retained, but both A_3 and A_4 were dropped. There was not a significant loss in fit relative to Model 6, indicating that A_3 and A_4 were not necessary to account for the observed genetic influences on youth-reported delinquency at age 14–17 years (Table 5). Therefore, Model 7 is preferred over Models 5 and 6 based on parsimony. In Model 8, only a single additive genetic factor that influenced antisocial behavior at all ages (A_1) was retained. This resulted in a significant loss in fit relative to Model 7, indicating that both A_1 and A_2 were required to fit the data adequately. Models analogous to Models 7 and 8 were conducted for shared environmental influences (C), but these models both fit significantly less well than Model 1. Because the nonshared environmental factors at each age include measurement error for antisocial behavior at that age, E_1 – E_4 are all required and could not be dropped.

To further evaluate the fit of the preferred model, Model 7 was compared to a saturated model that imposed no constraints on the variances and covariances. As expected with this sample size, based on χ^2 differences, Model 7 fit less well than the saturated model. Nonetheless, both RMSEA and BIC were greater for the saturated model than for Model 7 (Table 5), suggesting that Model 7 captured the data with greater parsimony than the saturated model. In addition, Model 7 was compared to a null model that constrained all covariances to be 0. As expected, all tests and indices suggested that Model 7 provided a better fit to the data than the null model.

Standardized path coefficients for Model 7 are shown in Figure 2 (squared to quantify the variance in each phenotype explained by the path). Parameter estimates do not sum to 1.0 because small parameter estimates with a 95% confidence interval that included 0 were omitted from the figure.² Because quantitative sex differences were detected in Series 1, parameter estimates are reported separately for females and males. As shown in Figure 2, the first genetic factor (A_1) robustly influenced mother-reported conduct problems at 4–9, 10–11, and 12–13 years, but accounted for < 1% of youth-reported delinquency at ages 14–17 years in both sexes. Indeed, dropping the path from A_1 to delinquency at 14–17 years did not result in a significant loss in fit relative to the full Model 7 (Satorra-Bentler scaled-difference $\chi^2 = 4.4$, $df = 2$, $p = .11$). The second genetic factor (A_2) modestly influenced mother-reported conduct problems at 10–11 and 12–13 years and influenced youth-reported delinquency at ages 14–17 years. Because unique genetic influences on delinquency at 14–17 years (A_4) could be dropped without significant loss in fit, these findings indicate that virtually all genetic influences on youth-reported delinquency at ages 14–17 years were shared across informants and age with those on mother-reported conduct problems at 10–11 and 12–13 years.

Series 2: In the second series of Cholesky decomposition models, the developmentally ordered phenotypes were mother-reported child conduct problems at 4–9 years and youth-reported delinquency at 10–11, 12–13 years, and 14–17 years. As in Series 1, Models 1–5 included four latent factors each for additive genetic (A_1 – A_4), shared environmental (C_1 – C_4), and nonshared environmental (E_1 – E_4) influence.

Model 1 allowed quantitative sex differences and Model 2 allowed scalar sex differences. Model 2 did not fit significantly worse than Model 1 at the .05 level (Table 6), and because Model 2 fit slightly better than Model 1 on other fit indices, Model 2 (scalar sex differences) was conservatively used as the baseline model for testing Models 3–6. These models test whether genetic and environmental influences on youth-reported delinquency at 14–17 years were shared with those on mother-reported conduct problems and youth-reported delinquency at earlier ages. In Model 3, all paths from genetic factors A_1 – A_3 to youth-reported delinquency at 14–17 years were dropped, but A_4 and all paths for both types of environmental influences to youth-reported delinquency at 14–17 years were retained. Consistent with the genetic mediation hypothesis, this resulted in a significant loss in fit relative to Model 2 (Table 6).

In Model 4, all paths from both genetic factors and nonshared environmental factors to delinquency at 14–17 years were retained and all paths from shared environmental factors C_1 – C_3 to delinquency at 14–17 years were dropped. This did not result in significant a loss in fit relative to Model 2 (Table 6). In Model 5, all paths from both genetic factors and shared environmental factors to delinquency at 14–17 years were retained and all paths from nonshared environmental factors E_1 – E_3 to delinquency at 14–17 years were dropped. This did not result in significant a loss in fit relative to Model 2 (Table 6).

Three additional models were fitted to test the genetic mediation hypothesis further. In Model 6, genetic influences on delinquency at age 14–17 that were unique to that age were eliminated by retaining A_1 – A_3 , but dropping A_4 . In Model 7, only two genetic factors (A_1 and A_2) with paths to delinquency at all ages were retained. Models 6 and 7 included all environmental factors (C_1 – C_4 and E_1 – E_4) and all of their paths. Model 6 did not fit the data significantly less well than Model 2 and Model 7 did not fit the data less well than Model 6; therefore Model 7 is preferred based on parsimony. This indicates that, after accounting for the shared paths from A_1 and A_2 , it is not necessary to specify additional paths for genetic influences on youth-reported delinquency at ages 12–13 or 14–17 years (i.e., A_3 and A_4 are not needed in the model). In Model 8, only a single additive genetic factor that influenced antisocial behavior at all ages

²Tables of all parameter estimates and their 95% confidence intervals for the full model in each series are available from the first author.

(A₁) was retained. This resulted in a significant loss in fit relative to Model 7, indicating that both genetic factors A₁ and A₂ are required to fit the data adequately.

Model 7 was compared to a saturated model as described in Series 1. Once again, Model 7 fit significantly less well than the saturated model based on the χ^2 difference test (Table 6). In this case, the RMSEA was lower for the saturated model than for Model 7, but the BIC of the saturated model was larger than the BIC values of Model 7, reflecting the greater parsimony of Model 7. As expected, a null model constraining all covariances to 0 fit significantly less well than model 7.

Squared standardized parameter estimates from Model 7 are shown in Figure 3 (small parameter estimates with a 95% confidence interval that included 0 were omitted). Because these estimates were derived from a scalar sex-differences model in which coefficients were constrained to be the same across sexes even though variances differed, results for females and males are not presented separately. The results indicated that there were no unique genetic influences on delinquency at 12–13 or 14–17 years; rather all genetic influences on delinquency at these ages were shared with those on delinquency at 10–11 years. There was no sharing of genetic influences on youth-reported delinquency at 14–17 years with mother-reported conduct problems at 4–9 years and there was only modest sharing of environmental influences on these antisocial phenotypes across this age range, even within the same informant.

Sensitivity Analyses: It should be noted that the measure of child conduct problems refers to a somewhat different antisocial behaviors than the youth self-report of delinquency items. Nonetheless, three of the child conduct problem were analogous to youth self-report items (cheats or lies $\approx$ lies to parents; bullies or is cruel $\approx$ hurt someone on purpose; and breaks things on purpose $\approx$ vandalism at school). As a sensitivity check on these analyses, all model in Series 1 and 2 were fit using only these three items, with the model-fitting results being qualitatively the same as with the full 7-item child conduct problems score. This suggests that the differences in items associated with differences in informants did not substantially account for the results of Series 1 and 2.

DISCUSSION

We fit two series of multivariate Cholesky decomposition models to estimate the extent to which genetic and environmental influences on developing antisocial behavior are the same or different at different ages from childhood through late adolescence. The genetic mediation hypothesis (Lahey & Waldman, 2003) states that all genetic influences on adolescent delinquency are mediated by childhood conduct problems. That is, genetic influences on conduct problems in childhood are hypothesized to set processes in motion that lead some children to develop antisocial behavior that persists through adolescence, but there are no hypothesized genetic influences on adolescent delinquency independent of those of childhood conduct problems (Lahey & Waldman, 2003).

Consistent with the genetic mediation hypothesis (Lahey & Waldman, 2003), no unique genetic influences on adolescent delinquency at 14–17 years were identified in either series of analyses. Rather, the genetic factor that influenced antisocial behavior across 10–13 years (A₂), regardless of the informant at those ages, also influenced youth-reported delinquency at 14–17 years. This suggests that the same genes influence antisocial behavior after about age 10, even when different informants report on antisocial behavior using somewhat different lists of antisocial behaviors at 10–13 and 14–17 years.

Directly contrary to the genetic mediation hypothesis, however, no sharing of genetic influences on youth-reported delinquency at 14–17 years was found with mother-reported

conduct problems at 4–9 years. This disconfirmation will require a major modification of our hypothesis regarding the developmental structure of genetic influences on antisocial behavior across childhood and adolescence (Lahey & Waldman, 2003). Within the limits of current methods of assessing antisocial behavior in young children, it seems clear that child conduct problems at 4–9 years of age do not mediate genetic influences on adolescent delinquency.

It is possible that the change in the sharing of genetic influences with delinquency at 14–17 years that occurs during 10–13 years of age reflects pubertal maturation more than increasing age itself (Burt, McGue, DeMarte, Krueger, & Iacono, 2006; Rowe, Maughan, Worthman, Costello, & Angold, 2004). This is an important topic for future study, but the limited data on pubertal maturation in the CNLSY made it impossible to directly test this hypothesis.

It should be noted that low phenotypic correlations in the present study between mother-rated conduct problems at 4–9 years and youth-reported delinquency at 14–17 years ($r = .14$ in both sexes) limited the extent to which sharing of causal influences across this age span was possible. It is important to note that the present low phenotypic correlations across age and informants are not atypical of other longitudinal studies. Tuvblad et al. (2005) reported similarly low correlations between mother-reported conduct problems at 8–9 years and youth-reported delinquency at age 13–14 years, $r = .07$ –.19. Similarly, in the Dunedin Study (Moffitt et al., 1996), maternal ratings of conduct problems at 5, 7, 9, and 11 years were modestly correlated with youth-reported delinquency at ages 13, 15, and 18 years, $r = .09$ –.17 (T. E. Moffitt, personal communication). Thus, the low correlation of antisocial behavior across age and informants is consistent across studies and is an important *substantive* finding that must be incorporated in developmental models of the development of antisocial behavior. This is because low correlation between any two phenotypes is a clear indication that they do not extensively share causal influences.

Confounding of Age, Informants, and Items Used to Measure Antisocial Behavior

The finding that genetic influences on mother-reported conduct problems at 10–13 years were shared with those on youth-reported delinquency at 14–17 years means that the lack of shared genetic influences between mother-reported conduct problems at 4–9 years and youth-reported delinquency at 14–17 years is not *solely* due to the difference in informants. If that were the case, there would not have been significant sharing of genetic influences on youth-reported delinquency 14–17 years and mother-reported conduct problems at ages 10–13 years.

There is, however, an important “uncertainty” principle inherent in studies of developmental differences in causal influences on antisocial behavior across lengthy age spans. At present, no reliable and valid measures have been devised in which the same informant can report on antisocial behavior with equal reliability and validity at every age from 4 through 17 years. Only youth have full knowledge of their antisocial behavior during adolescence (Loeber et al., 1998; Schwab-Stone et al., 1994) and children < 9 years are not reliable informants on their own behavior in interviews (Schwab-Stone et al., 1994). Thus, given current measures of antisocial behavior, it is unknowable whether genetic influences on antisocial behavior at 4–9 years would have been found to be shared with those on antisocial behavior at 14–17 years if the same informant could have been used at both ages. Nevertheless, it is possible that assessment strategies will be developed in the future to would allow youth to provide data on their conduct problems across 4–17 years. This would be of great value, but attempts to do so have not been successful to date. For example, interviews that provide children with cartoon stimuli to help them understand oral questions about their behavior have been found to be unreliable for children < 7 years of age (Valla, Bergeron, Berube, Gaudot, & St. Georges, 1994).

Regardless, the confounding of informant differences with age differences from early childhood through late adolescence does not justify ignoring questions about the developmental structure of causal influences across broad age spans. Our developmental models of antisocial behavior must incorporate findings about differences in causal influences across the full span of development, even if they are complicated by uncertainty related to the confounding of age and informants. Regardless of how much of the difference in genetic influences on mother-reported conduct problems at 4–9 years and youth-reported delinquency at 14–17 years is due to differences in informants or items used to measure antisocial behavior, any adequate developmental model of antisocial behavior must account for this finding. This is because the present findings suggest that those children who are at greatest risk for persistent and serious antisocial behavior across early childhood and late adolescence (i.e., LCP delinquents) have *both* the genetic factors associated with risk for *mother-reported* childhood conduct problems and the *separate* genetic factors associated with risk for *youth-reported* adolescent delinquency. If correct, this new hypothesis also would mean that LCP delinquency is uncommon in the population partly because two uncorrelated sets of risk genes must both be present to place children at greatest genetic risk for LCP delinquency.

Alternatively, it is possible that it would be more informative to view genetic factors as influencing entire *trajectories* of antisocial behavior over spans of age rather than thinking of genes as influencing antisocial behavior at different ages. For example, there may be separate genes that increase risk for the LCP and AL trajectories (Moffitt, 2006). Given that the definition of LCP delinquency *requires* a history of significant mother-reported conduct problems in early childhood, however, the complete absence of shared genetic influences between early childhood conduct problems and adolescent delinquency in the present study argues against the possibility of genes specific to the LCP trajectory. Nonetheless, future studies should give full consideration to both approaches.

Sex Differences in Genetic Influences on Developing Antisocial Behavior

The present findings also contribute to the growing, but still inconsistent literature on the likely existence of sex differences in genetic and environmental influences on antisocial behavior (Moffitt et al., 2001; Rhee & Waldman, 2002; Van Hulle et al., 2007). Although there is no evidence that the causes of antisocial behavior are qualitatively different, some evidence suggests that there are quantitative differences (Van Hulle et al., 2007). The present findings suggest that there are sex differences in causal influences on antisocial behavior across development (Table 4–Table 6), but the univariate and multivariate models were not consistent regarding the specific nature of those differences. Some analyses suggested that the magnitudes of genetic and environmental influences vary quantitatively by sex, whereas as other analyses suggest that the magnitudes of genetic and environmental influences are equal, but that there are scalar sex differences in variances in antisocial behavior. Because there are large sex differences in the prevalence of antisocial behavior, and findings of quantitative and scalar sex differences could reflect sex differences in thresholds for genetic influences on antisocial behavior (Van Hulle et al., 2007), it is very important to pursue this topic further in the future. If there are different thresholds for the same genetic and environmental influences on antisocial behavior on females and males, the factors that contribute to such inhibitory thresholds should be identifiable. If so, it may be possible to use this knowledge to constructively inhibit antisocial behavior in males.

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Figure 1.

Illustration of a full multivariate Cholesky decomposition model for four developmentally ordered phenotypes. Note: A refers to latent additive genetic factors; C refers to latent shared environmental factors, and E refers to latent nonshared environmental factors and error.

Figure 2. Squared standardized coefficients for paths in Model 7 from Series 1, presented separately for females and males due to quantitative sex differences. Parameters whose 95% confidence intervals included 0 are not shown. Minus sign indicates that the parameter was negative before squaring.

Females and Males

Figure 3. Squared standardized coefficients for paths in Model 7 (specifying scalar sex differences) from Series 2 for both females and males. Parameters whose 95% confidence intervals included 0 are not shown.

Table 1

Number of Sibling Pairs at Each Degree of Genetic Relatedness in the Analyses

	r_g	Male Pairs	Female Pairs	Different-sex Pairs
Half Siblings	.25	119	128	275
Ambiguous Siblings	.375	84	94	154
Full Siblings (blended) ^a	.5	96	82	146
Full Siblings (non-blended) ^b	.5	331	326	647

Note. r_g refers to the genetic correlation between siblings.

^aNumber of full sibling pairs in families in which there also are one or more half sibling pairs.

^bNumber of full sibling pairs in families with only full sibling pairs. (total 2,482)

Table 2

Informants and Measures Available in Each Age Range Used in the Two Series of Cholesky Models Addressing the Sharing of Causal Influences on Delinquency at Ages 14–17 Years with Antisocial Behavior at Earlier Ages

	4 – 9 Years	10 –11 Years	12–13 Years	14–17 Years
Series 1	Mother-reported child conduct problems	Mother-reported child conduct problems	Mother-reported child conduct problems	Youth self-reported delinquency (YA)
Series 2	Mother-reported child conduct problems	Youth self-reported delinquency (CSAS)	Youth self-reported delinquency (CSAS)	Youth self-reported delinquency (YA)

Note: CSAS = Child Self-Administered Supplement; YA = Young Adult Self-Administered Survey.

Table 3

Means and Standard Deviations of Each Measure of Antisocial Behavior and Correlations Between Youth-Reported Delinquent Behavior at 14–17 Years and Each Measure of Antisocial Behavior at Earlier Ages for Females and Males

	Males		Females		Tests of sex differences		Phenotypic correlations with youth-reported delinquency at 14–17 ^a	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	differences in means	differences in variances	Males	Females ^d
Mother-Reported Conduct Problems (4–9 Years)	0.25	0.95	–0.08	0.77	10.3	1.5	.14	.14
Mother-Reported Conduct Problems (10–11 Years)	0.19	1.06	–0.14	0.88	9.7	1.4	.18	.20
Mother-Reported Conduct Problems (12–13 Years)	0.14	1.04	–0.10	0.94	7.0	1.2	.24	.23
Youth-Reported Delinquency (10–11 Years)	0.20	1.10	–0.21	0.85	10.4	1.6	.25	.24
Youth-Reported Delinquency (12–13 Years)	0.14	1.05	–0.17	0.91	7.9	1.3	.35	.41
Youth-Reported Delinquency (14–17 Years)	0.07	0.93	–0.11	0.86	5.6	1.2	—	—

Note: Means and standard deviations are based on standardized z scores for each measure of antisocial behavior.

^a Sex differences in phenotypic correlations between delinquency at 14–17 years and each measure of earlier conduct problems were all non-significant (all $p > .10$) using Fisher's r-to-z transformation.

* All $p < .05$

Table 4
Best Fitting Univariate Models for Mother-Reported Conduct Problems and Youth-Reported Delinquency in Each Age Range (by Informant)

Model	RMSEA	h ²	c ²	c ²
Mother-Reported Conduct Problems (4–9 Years)	.06	.64	.19	.17
Mother-Reported Conduct Problems (10–11 Years)	.02	.34	.22	.44
Mother-Reported Conduct Problems (12–13 Years)	.07	.81 ^a	.09 ^a	.09 ^a
Youth-Reported Delinquency (10–11 Years)	.08	.23 ^b	.30 ^b	.46 ^b
Youth-Reported Delinquency (12–13 Years)	.05	.23	.10	.67
Youth-Reported Delinquency (14–17 Years)	.06	.21	.15	.64

Note. The quantitative sex-differences (sex limitation) model allows parameter estimates to vary across genders; the scalar sex-differences model constrains the parameter estimates for females to be a scalar multiple of parameter estimates for males.

^aParameter estimates for males.

^bParameter estimates for females

Results of Multivariate Cholesky Decompositions Estimating the Sharing of Causal Influences on Youth-Reported Delinquency at 14–17 Years With Mother-Reported Conduct Problems at 4–13 Years (Series 1)

Table 5

	SB χ^2	df	RMSEA	SB $\Delta\chi^2$	df	P	BIC
Model 1: Quantitative sex differences	766.9	438	.060	-	-	-	48527
Model 2: Scalar sex differences	836.9	463	.062	58.9	25	<.001	48478
Model 3: A for adolescent delinquency not shared with earlier ages ^a	793.0	444	.062	26.9	6	<.001	48515
Model 4: C for adolescent delinquency not shared with earlier ages ^d	800.1	444	.062	797.8	6	<.001	48510
Model 5: E for adolescent delinquency not shared with earlier ages ^{d,c}	770.3	446	.059	3.7	8	.88	48469
Model 6: No unique A for adolescent delinquency ^b	770.4	440	.060	3.4	2	.18	48511
Model 7: Retains 2 genetic factors (A ₁ and A ₂)	777.4	444	.060	6.8	4	.15	48480
Model 8: Retains 1 genetic factor	807.9	450	.062	29.4	6	<.001	48475
Saturated Model	600.41	340	.061	178.0	104	<.001	48993
Null Model	4040.1	480	.189	2096.2	36	<.001	52879

Note: Models in this series are defined in the Results section under the heading, *Series 1*. Models 2–6 were compared to Model 1. Model 7 was compared to Model 6 and Model 8 was compared to Model 7. For reference, Model 7 was compared to a saturated model that imposed no constraints on the variances and covariances, and to a null model in which all covariances were constrained to 0.

^aModel drops all genetic (A), shared environment (C), or non-shared environment (E) influences that mother-reported conduct problems at ages 10–13 share with Youth-reported delinquency at ages 14–17.

^bModel drops genetic factor specific to youth-reported delinquency at ages 14–17.

^cAdditive genetic influences unique to delinquency at age 14–17 were fixed at values estimated in Model 2 to obtain valid standard errors.

Results of Multivariate Cholesky Decomposition Estimating the Sharing of Causal Influences on Youth-Reported Delinquency at 14–17 Years With Youth-Reported Conduct Problems at 10–13 Years and With Mother-Reported Conduct Problems at 4–9 Years (Series 2)

Table 6

	SB χ^2	df	RMSEA	SB $\Delta \chi^2$	df	P	BIC
Model 1: Quantitative sex differences	664.7	436	.059				37347
Model 2: Scalar sex differences	700.2	462	.059	37.1	26	.07	37232
Model 3: Drop shared A ^a	741.6	465	.063	139.4	3	<.001	37258
Model 4: Drop shared C ^a	707.1	465	.059	6.4	3	.33	37220
Model 5: Drop shared E ^a	706.9	465	.059	6.2	3	.35	37220
Model 6: No unique A ^b	701.7	463	.059	1.7	1	.19	37224
Model 7: Retains 2 genetic factors (A ₁ and A ₂)	704.8	465	.059	3.0	2	.21	37209
Model 8: Retains 1 genetic factor	752.8	468	.064	167.2	3	<.001	37243
Saturated Model	403.57	340	.035	233.9	96	<.001	37713
Null Model	1773.37	464	.137	102.5	28	<.001	38467

Note: Models in this series are defined in the Results section under the heading, *Series 2*. Model 2 was compared to Model 1, Models 3–6 were compared to Model 2. Model 7 was compared to Model 6 and Model 8 was compared to Model 7. For reference, Model 7 was compared to a saturated model that imposed no constraints on the variances and covariances, and to a null model in which all covariances were constrained to 0.

^aModel drops all genetic (A), shared environment (C), or non-shared environment (E) influences that youth-reported delinquency at ages 10–13 share with youth-reported delinquency at ages 14–17.

^bModel drops genetic factor specific to youth-reported delinquency at ages 14–17.