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Aggressive Versus Nonaggressive Antisocial Behavior: Distinctive Etiological Moderation by Age

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Abstract

Research has supported the existence of distinct behavioral patterns, demographic correlates, and etiologic mechanisms for aggressive (AGG) versus nonaggressive but delinquent (DEL) antisocial behavior. Though behavioral genetic studies have the potential to further crystallize these dimensions, inconsistent results have limited their contribution. These inconsistencies may stem in part from the limited attention paid to the impact of age. In the current study, the authors thus examined age-related etiological moderation of AGG and DEL antisocial behavior in a sample of 720 sibling pairs (ranging in age from 10 to 18 years) with varying degrees of genetic relatedness. Results reveal that the magnitude of genetic and environmental influences on AGG remained stable across adolescence. By contrast, genetic influences on DEL increased dramatically with age, whereas shared environmental influences decreased. Subsequent longitudinal analyses fully replicated these results. Such findings highlight etiological distinctions between aggression and delinquency, and offer insights into the expression of genetic influences during development.

Keywords

genetic influences; age; aggression; delinquency

Antisocial behavior (ASB) describes actions and attitudes that violate societal norms and the personal or property rights of others. Though typical examples include vandalism, theft, and bullying/assault, the specific manifestation varies markedly from individual to individual (Lahey & Waldman, 2003; Loeber & Stouthamer-Loeber, 1998; Offord & Bennett, 1994; White, Bates, & Buyske, 2001). Moreover, this phenotypic heterogeneity has important long-term consequences, with outcomes ranging from conventional, crime-free lifestyles to multiple stints in the prison system (Lahey & Waldman, 2003; Lynam, 1996).

To better understand this variability, researchers have long advocated the parsing of ASB into conceptually meaningful dimensions. The factor analytic literature has consistently indicated that there are at least two moderately correlated factors, an *overt* or aggressive/

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oppositional factor and a *covert* or nonaggressive/delinquent factor (Frick et al., 1993; Loeber & Schmaling, 1985). This distinction is evident in both empirically derived behavioral rating scales, such as the Child Behavior Checklist (Achenbach & Edelbrock, 1983), and in factor analyses of conduct disorder and oppositional defiant disorder symptoms (Tackett, Krueger, Iacono, & McGue, 2005; Tackett, Krueger, Sawyer, & Graetz, 2003).

Importantly, this aggressive/nonaggressive distinction appears to neatly map onto the more typical approach to subtyping the heterogeneity of ASB, that regarding age-of-onset (Moffitt, 1993, 2003). Research has indicated that, as compared with those whose ASB began in adolescence, those whose onset was prior to 10 years of age exhibited higher rates of aggressive behaviors but roughly the same prevalence of nonaggressive, rule-breaking behaviors (Lahey et al., 1998). These results extend previous findings indicating that the median age-of-onset of aggressive behaviors is earlier than that of nonaggressive but delinquent behaviors (Lahey, Loeber, Quay, Frick, & Grimm, 1992), collectively indicating that the age-of-onset of ASB may be intimately tied to the presence or absence of aggression.

Consistent with the above point, aggressive and delinquent ASB also demonstrate markedly different demographic and developmental trajectories. Physical aggression typically first manifests itself in early childhood. Indeed, it is most prevalent during the toddler years (Tremblay, 2003), after which mean levels of aggression steadily decrease (though they increase again briefly during mid-adolescence; Stanger, Achenbach, & Verhulst, 1997; Tremblay, 2003). Even so, aggression exhibits high levels of rank-order stability across development, such that those young children with the highest levels of aggression continue to be particularly aggressive as adults (Tremblay, 2003). Delinquency, by contrast, is far less stable during development, is quite infrequent during childhood, and increases over the course of adolescence, only to fall off again during the transition into adulthood (Stanger et al., 1997). Also of note, recent research has supported conceptual distinctions between aggressive and delinquent ASB, such that deficits in affective regulation appear to be largely exclusive to aggression and do not extend to nonaggressive delinquency (Burt & Donnellan, 2008; Burt & Larson, 2007; Cohen & Strayer, 1996; Pardini, Lochman, & Frick, 2003), whereas impulsivity seems to be specific to delinquency (Burt & Donnellan, 2008).

Though the above literature has begun to resolve the heterogeneity within ASB, genetically informative studies have yet to substantively contribute to this resolution. Indeed, as noted in a meta-analysis (Rhee & Waldman, 2002), most twin and adoption research has conceptualized ASB as a unitary construct, either conduct disorder or broadly defined ASB, without regard to the presence or absence of aggression. Although such studies are useful for understanding causal influences on a general tendency to act out, they do not illuminate distinctions within the overarching construct of ASB, a particularly important goal given the distinct behavioral correlates and developmental patterns of aggression and delinquency highlighted above. Should there be evidence of different etiologic mechanisms across the two subtypes, it would offer strong support for prior indications that aggression and delinquency represent distinct forms of pathology.

That said, there are a handful of existing studies that have examined the Child Behavior Checklist Aggression and Delinquency scales or have focused on aggressive and nonaggressive factors of conduct disorder as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994). Unfortunately, results have been inconsistent. Some studies have found that aggression is more heritable than delinquency (Eley, Lichtenstein, & Stevenson, 1999; Hudziak et al., 2003), whereas delinquency is more strongly influenced by the shared environment (Eley, Lichtenstein, & Moffitt, 2003; Eley et al., 1999; Tackett et al., 2005). Other studies, however, have reported strikingly similar heritabilities across aggression and delinquency (Deater-Deckard & Plomin, 1999; Gelhorn et al., 2005).

One factor likely contributing to these inconsistencies is the minimal consideration given to age (Rhee & Waldman, 2002). Studies contrasting aggression and delinquency to date have regressed out age prior to analysis (i.e., conducted regressions with age as the predictor, and computing residuals for the heritability analyses; Deater-Deckard & Plomin, 1999), have studied only a circumscribed age range (Bartels et al., 2003; Eley et al., 1999; Tackett et al., 2005), or have focused more on understanding etiological continuity across age rather than etiological change with age (Eley et al., 2003; Hudziak et al., 2003). This lack of consideration of the impact of age on genetic and environmental parameter estimates represents a key interpretative limitation of prior studies. Specifically, although the vast majority of behavioral genetic studies continue to report static heritability estimates (i.e., from a single point in time), there is evidence that the magnitude of genetic influences on ASB/aggression varies markedly across development (Bergen, Gardner, & Kendler, 2007; Lyons et al., 1995; Miles & Carey, 1997; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003). For example, Lyons et al. (1995) examined retrospectively reported juvenile and adult symptoms of antisocial personality disorder within a sample of 3,226 pairs of male twins from the Vietnam Era Twin Registry. They found evidence that additive genetic influences were much stronger in adult versus juvenile ASB (with standardized genetic estimates of 43% vs. 7%, respectively), whereas shared environmental factors demonstrated the opposite pattern. Given this, prior studies' minimal consideration of age may have obscured differences in the heritability of aggression and delinquency at particular ages.

Moreover, these increases in heritability from childhood to adulthood are thought to have key theoretical implications. They have typically been interpreted as reflective of active genotype–environment correlational (rGE) processes (Plomin, DeFries, & Loehlin, 1977), such that as individuals transition from childhood through adolescence and into adulthood, they exert a progressively greater impact on the environments they experience, allowing their genetic predispositions to more fully manifest themselves (Scarr & McCartney, 1983). Put differently, if individuals are choosing environments consistent with their genotypes, we would expect environmental feedback to iteratively validate their genetic proclivities, a reinforcing process that should eventuate in increased expression of their genetic predispositions (Bergen et al., 2007). Understanding the age-related timing and sequencing of genetic influences is thus of notable theoretical significance to developmental psychologists, refining not only our understanding of the mechanisms through which genes

influence human behavior but also informing our understanding of the developmental course of the phenotypes under study.

Given all of the above, evidence of differences (or lack thereof) in the timing of genetic expression between aggression and delinquency would certainly help resolve the heterogeneity within ASB. Namely, we hypothesize that, consistent with the subtype-specific changes in aggressive and nonaggressive ASB with age (both within and across persons), genetic influences on aggression and delinquency are expressed during different developmental periods. In particular, we propose that the magnitude of genetic influences on delinquency will increase significantly during adolescence, at least partially driving the notable increases in delinquency (and increasing variation) both across and within individuals during this developmental period. By contrast, given the early childhood onset that is typical of physical aggression and its high levels of rank-order stability thereafter, we suspect that the genes of risk for aggression are expressed quite early in life. Indeed, prior work by van Beijsterveldt et al. (2003) has suggested just this. They reported that genetic influences on aggression increased dramatically (tripling in magnitude) from 3 to 7 years of age, after which they remained largely stable (van Beijsterveldt et al., 2003). Given this, we further hypothesize that the magnitude of genetic and environmental influences on aggression will remain constant during adolescence. Should results support these contentions, such findings would offer circumstantial evidence that active rGE (which is thought to be a less salient developmental influence before adolescence; see Scarr & McCartney, 1983) may be particularly important for delinquency but may have a more limited role in the development of aggression. Moreover, positive results would buttress prior work indicating that aggression and delinquency are distinct developmental phenomena, thereby shaping future research and intervention efforts (e.g., future developmental work could specifically examine factors that distinguish and unite the subtypes).

In the present article, we thus sought to explicitly evaluate whether genetic contributions to aggressive and delinquent ASB shifted differentially over the course of adolescence. To do so, we examined the etiological moderation of aggression and delinquency by age in a sample of adolescent sibling pairs with varying degrees of genetic relatedness (Neiderhiser, Reiss, & Hetherington, 2007). Analyses were conducted two ways. First, we used a series of nested interaction models (separately for each phenotype; Purcell, 2002) with age as the moderating variable, thereby allowing us to explicitly evaluate how age moderates the etiology of aggressive and nonaggressive ASB within a cross-sectional design. However, to ensure that any positive results were not a function of our cross-sectional design, we then attempted to replicate and extend our age moderation results using simple longitudinal comparisons of genetic and environmental variance components. As noted, we specifically conjectured that genetic influences on delinquency would increase over the course of adolescence, whereas aggression would remain etiologically stable. Positive results would further illuminate the heterogeneity of ASB and offer insights into the expression of genetic influences during development.

Method

Participants

The data for this study were from the Nonshared Environment in Adolescent Development (NEAD) Project (Neiderhiser et al., 2007). Families were sampled by means of random-digit dialing and commercial market panels with family information to identify certain family types (e.g., twin, step-, and nondivorced families). At intake, the resulting sample consisted of a total of 720 families with same-sex adolescent sibling pairs (51.6% boys) from 47 states in the United States. Eligibility for the study required sibling pairs to be no more than 4 years apart in age, to be between 10 and 18 years of age (one 9-year-old is included in the 10-year-old group), and to reside in the household at least half time. The latter was a necessary requirement of the classical twin/sibling study: Siblings must live together to adequately estimate shared environmental influences (e.g., dizygotic [DZ] twins and full-siblings are assumed to share 50% of their segregating genes and 100% of their "shared" or familial/neighborhood environment, whereas step-siblings are assumed to only share their familial/neighborhood environment). Step-families were additionally required to have been in existence for at least 5 years. The final sample was composed of monozygotic (MZ) and DZ twin pairs (n = 93 and 99, respectively; 12 twin pairs were unable to be classified and so were omitted from our analyses), full-sibling pairs from nondivorced families (n = 95), full-sibling pairs in step-families (n = 182), half-sibling pairs in step-families (n = 109), and genetically unrelated sibling pairs in step-families (n = 130). Of note, these proportions were not meant to be representative of the general population. For example, twins are overrepresented relative to their prevalence in the general U.S. population. Instead, family types were sampled with the express goal of facilitating examinations of genetic and environmental influences (e.g., sufficient MZ twin pairs were needed to power the estimation of genetic influences, whereas step-siblings served to enhance the estimation of shared environmental influences).

Families were invited to participate in a follow-up assessment roughly 3 years after their intake assessment (Neiderhiser et al., 2007). To be included in the follow-up assessment, both adolescents were again required to reside in the home at least half time. Failure to meet these criteria resulted in a reduced Time 2 sample of only 405 families. Importantly, however, only 9% of eligible families refused to participate. There were no mean demographic differences (in parents' education, family income, sex of siblings, or age difference of siblings) between families that participated at Times 1 and 2 and those that participated only at Time 1.

At intake, the mean age of the older child was 14.5 years (±2.2) and that of the younger child was 12.9 years (±2.2). Age of entry into the study varied significantly (p = .022), though minimally, across family type, with a range of 13.31–14.18 years (average age was 13.71 years, with a standard deviation of 2.34 years). The largest Cohen's d effect size for any one family type was 0.25 (whereas the smallest was 0.00), suggesting at most only small differences in age of entry by family type. At follow-up, the mean age of the older child was 16.2 years (±2.1) and that of the younger child was 14.7 years (±1.9). The families reported a wide range of family incomes (median family income range was $25,000–35,000, and

families ranged from lower working class to upper middle class on the Hollingshead Four Factor Indicator of socioeconomic status; Hollingshead, 1975) and education (mean years of education = 13.6 for mothers, 14.0 for fathers). Most participants (i.e., 94% of mothers and 93% of fathers) were European American. Adolescents gave informed assent, whereas parents gave informed consent for themselves and their children. Additional details on the sample, measures, and zygosity procedures are available in prior publications (Neiderhiser et al., 2007; Reiss, Neiderhiser, Hetherington, & Plomin, 2000).

Measures

At both the Time 1 and Time 2 assessments, data were collected in two 3-hr home visits separated by no more than 2 weeks. ASB was measured by adolescent self-, mother-, and father-report on the Behavior Problems Index (Zill, 1985), adapted from the Child Behavior Checklist (Achenbach & Edelbrock, 1983). This measure includes items assessing behaviors that corresponded to *DSM–IV* criteria for conduct disorder (e.g., argued, disobedient at home or school, destructive toward objects). Participants were asked to rate on a 3-point scale the extent to which a series of statements generally described their behavior (or that of their child) over the last 3 months. Prior work has indicated that the use of single informants may bias estimates of genetic and environmental contributions (Eaves et al., 1997, 2000; Sherman, McGue, & Iacono, 1997; Simonoff et al., 1995), in part because the use of only parent or only child informants provides a less valid indication of child and adolescent behavior problems (Bird, Gould, & Staghezza, 1992; Hart, Lahey, Loeber, & Hanson, 1994; Jensen et al., 1999). To address these concerns, we averaged each item across informant prior to scale creation.

We utilized previously defined aggressive (AGG; α = .82; i.e., “bullied,” “cruel,” “mean,” “strong temper”) and delinquent (DEL; α = .84; i.e., “cheat or lie,” “break things,” “bad friends,” “no guilt,” “disobedient at home,” “disobedient at school”) item subsets from the 10-item ASB scale on the Behavior Problems Index (Feinberg, Button, Neiderhiser, Hetherington, & Reiss, 2007). Consistent with the literature (Hudziak et al., 2003), our AGG and DEL scales were moderately to highly correlated (*r*s ranged from .56 for adolescent-reports to .71 for father-reports; all *p*s < .01). To ensure that these item subsets captured separate dimensions of ASB, we submitted all 10 items at Time 1 to a confirmatory factor analysis using the generalized least squares estimation method. We compared an oblique two-factor model ($\chi^2_{n=1,414}=323.836$ on 34 *df*, *p* = .000; Akaike information criterion [AIC] = 365.836; root-mean-square error of approximation [RMSEA] = .078) with a single-factor model ($\chi^2_{n=1,414}=378.448$ on 35 *df*, *p* = .000; AIC = 418.448; RMSEA = .083). We then repeated these analyses on the Time 2 data (two-factor model: $\chi^2_{n=808}=191.772$ on 34 *df*, *p* = .000; AIC = 233.772; RMSEA = .076; one-factor model: $\chi^2_{n=808}=246.077$ on 35 *df*, *p* = .000; AIC = 286.077; RMSEA =.086). The improved fit of the two-factor model, as indicated by a smaller AIC value, suggests that AGG and DEL are separate though correlated dimensions of ASB at both time points. Moreover, the two-factor model provided an adequate fit to the data, with RMSEA values of .078 and .076 at Times 1 and 2, respectively.[1]

Statistical Analyses

Behavioral genetic analyses make use of the difference in the proportion of segregating genes shared between reared-together siblings (all of whom share 100% of their shared or common environment): MZ twins (who share 100% of their genetic material), DZ twins and full-siblings (who share an average of 50% of their segregating genetic material), half-siblings (who share an average of 25% of their segregating genetic material), and step-siblings (who do not share any segregating genetic material). Utilizing these differences, the variance within observed behaviors or characteristics (i.e., phenotypes) is partitioned into three components: additive genetic (a^2), shared environment (c^2), and nonshared environment plus measurement error (e^2). The additive genetic component (a^2) is the effect of individual genes summed over loci, and it acts to increase sibling correlations relative to the proportion of genes shared. The shared environment (c^2) is that part of the environment common to siblings that acts to make them similar to each other regardless of their genetic similarity. As they share no genes, correlations between genetically unrelated step-siblings function as largely "direct" estimates of shared environmental effects. The nonshared environment (e^2) encompasses environmental factors (and measurement error) differentiating siblings within a pair. More information on genetically informative studies is provided elsewhere (Plomin, DeFries, McClearn, & McGruffin, 2001).

Etiological moderation by age—We first evaluated the impact of age on the etiology of DEL and AGG via a series of nested moderation models (see Figure 1; Purcell, 2002) using the cross-sectional Time 1 data. Of note, models were run separately for each measure, as models that would allow age to simultaneously and differentially moderate AGG and DEL are not currently available to our knowledge. The first and least restrictive model allows for both linear and nonlinear moderation of the genetic, shared, and nonshared environmental contributions (i.e., a, c, e) to AGG and DEL. Namely, at each age, we added linear (i.e., A_1, C_1, E_1) and nonlinear (i.e., A_2, C_2, E_2) moderators to genetic and environmental paths using the following equation: Unstandardized $\text{Variance}_{\text{Total}} = [a + A_1(\text{age}) + A_2(\text{age}^2)]^2 + [c + C_1(\text{age}) + C_2(\text{age}^2)]^2 + [e + E_1(\text{age}) + E_2(\text{age}^2)]^2$. We then fit a series of progressively restrictive moderator models for each subtype, in which the various linear and nonlinear moderators were constrained to be zero.

Several steps of data preparation were necessary for these age moderation analyses. First, because skewness in the distribution of the phenotype can artifactually suggest the presence of moderator effects (Purcell, 2002), we log-transformed each scale to better approximate normality (skew following transformation was 0.74 for DEL and 0.23 for AGG). We also statistically controlled gender effects via regression techniques (McGue & Bouchard, 1984). We then standardized the log-transformed scale scores to facilitate interpretation of the

[1]The placement of the "no guilt" item in DEL is somewhat peculiar given that affective dysregulation has been found to be specific to aggression. However, placing it on the AGG scale instead resulted in a significant decrease in fit ($\chi^2_{n=1,414}=342.3$ on 34 *df*). "Lacks guilt" is also contained within the Rule-Breaking (RB) scales in the Achenbach family of instruments (rather than the AGG scales; Achenbach & Edelbrock, 1983). There is thus a precedent for this pattern of association. This similarity with the Achenbach scales is also important for other reasons: First, AGG (and not RB) was found to be uniquely associated with affective dysregulation using the Achenbach Adult Self-Report scales (among others), indicating that although "lacks guilt" loads on RB, AGG is still uniquely tied to affective dysregulation. Second, Moffitt (2003) noted that studies using the Achenbach AGG and RB scales are largely tapping life-course persistent and adolescent-limited ASBs, respectively, which mirror the subtypes of AGG and DEL.

unstandardized estimates derived from the model. This step was necessary because Purcell (2002) has strongly recommended that unstandardized (or absolute) estimates be reported for moderator models, as the more typical standardized (or proportional) estimates can obscure absolute changes in genetic and environmental influences across different levels of the moderator. We also subtracted 10 from each age prior to data analysis, thereby bringing the floor moderator value to 0. Finally, Purcell has reported that quadratic moderator models are prone to local minima and should be run at least five times using multiple start values to ensure that the obtained estimates do in fact minimize minus twice the log-likelihood (−2lnL). This procedure was implemented.

Because these interaction models effectively involve fitting a separate biometric model for each individual as a function of their age, they require the use of full-information maximum-likelihood raw data techniques. Mx, a structural equation modeling program (Neale, Boker, Xie, & Maes, 2003), was used to fit models to the transformed raw data. The more restrictive models were compared statistically with the least restrictive full nonlinear model by taking the difference in −2lnL between the nonlinear and reduced models, which is chi-square distributed under the null hypothesis implied by the reduced model. Nonsignificant changes in chi-square indicate that the more restrictive model (i.e., that model with fewer parameters and thus more degrees of freedom) provides a better fit to the data.

Longitudinal changes in etiology—We next sought to extend our moderator results to a longitudinal design, evaluating whether and how genetic and environmental influences on AGG and DEL changed over time. We fit a series of two univariate models for each subtype. In the first model, genetic and environmental influences were allowed to vary over time. We next constrained parameter estimates to be equal across time and evaluated the change in model fit. Because of the missing data at Time 2, we again made use of full-information maximum-likelihood raw data techniques in Mx, which produce less biased and more efficient and consistent estimates than pairwise or listwise deletion in the face of missing data. When fitting models to raw data, their variances, covariances, and means are first freely estimated to get a baseline index of fit (−2lnL). The −2lnL under this unrestricted baseline model is then compared with −2lnL under more restrictive biometric models (separately for AGG and DEL). This comparison provides a likelihood-ratio chi-square test of goodness of fit for the model, which is then converted to the AIC (Akaike, 1987; AIC = $\chi^2 - 2\,df$), the traditional fit index of behavioral genetics research. The AIC measures model fit relative to parsimony to determine the best fitting model amongst nested models. Better fitting models have lower or more negative values.

Results

Moderation of Genetic and Environmental Influences by Age

Descriptives at Time 1—Raw means of AGG and DEL are presented in Figure 2. Results indicate that mean levels of both DEL and AGG change significantly with age, $F(8, 1405) = 2.5$ and 3.6, respectively; both ps ≤ .01. The variance in DEL also changes somewhat with age: For homogeneity of variance, $F(8, 1405) = 1.70$, $p < .10$. By contrast,

the variance in AGG appears to remain constant with age: For homogeneity of variance, $F(8, 1405) = 0.68$, $p = .71$.

Intraclass correlations at Time 1—Prior to moderator model-fitting analyses, we calculated cross-sectional intraclass correlations. These correlations offer a preliminary indication of genetic and environmental influences on DEL and AGG. To interpret intraclass correlation comparisons across development, we divided the sample into two equally sized groups for these analyses: late-childhood/early-adolescence (10–13 years of age) and mid- to late-adolescence (14–18 years of age). This division allowed us to accommodate the broad age range in these data, but it also made sense conceptually, as members of the older cohort were typically in high school, whereas members of the younger cohort were most typically in middle school. To avoid interpretative difficulties, we computed correlations only for those siblings in the same age range (i.e., 10–13 or 14–18 years of age). Correlations that decrease with decreasing genetic similarity among siblings implicate genetic effects. (Of note, the subsequent moderation analyses did *not* make use of the older/younger distinction used here. Instead, those analyses used the participants' actual age as the moderator of genetic and environmental effects and, thus, made use of the full sample, irrespective of sibling age or age difference).

As can be seen in Table 1, MZ correlations are uniformly larger than their corresponding DZ correlations, suggesting that genetic effects are important for AGG and DEL during both earlier and later adolescence. However, close inspection across age and subtype reveals some key differences. For DEL, the MZ–DZ difference is approximately twice as large in later adolescence as compared with earlier adolescence, tentatively implying that genetic influences on DEL may increase over the course of adolescence.[2] Conversely, the correlation between step-siblings, which functions as a largely direct estimate of shared environmental influences because they do not share genes, was highly significant for DEL when measured in earlier adolescence and effectively zero for those in later adolescence, suggesting that shared environmental influences on DEL decrease across adolescence. By contrast, genetic influences on AGG do not appear to shift across adolescence, as the magnitude of the MZ–DZ difference is largely constant across earlier and later adolescence. Similarly, the correlation between step-siblings is not significantly different from zero in either age range, suggesting that shared environmental contributions to AGG are minimal in both earlier and later adolescence. Such findings offer a preliminary indication of differential etiological moderation of AGG and DEL by age.

Time 1 moderator models—Test statistics for a series of nested moderator models are reported separately for each subtype in Table 2. We compared the −2lnL obtained in the least restrictive linear and nonlinear moderation model with the −2lnL found for each of the more restrictive models to yield a likelihood-ratio chi-square test of the constraints implied

[2]As expected, intraclass correlations for DZ twins were generally equivalent to those for full-siblings (both of whom share 50% of their segregating genetic material on average, as well as 100% of their common environment). The only exception was the DZ–full-siblings comparison for DEL in early adolescence, which was significantly different at $p < .01$. Though it remains unclear what may account for this pattern of DZ–full-siblings differences, it could reflect the fact that same-age DZ twins are more likely to commit delinquent acts (but not aggressive acts) together during early adolescence in particular and, thus, are more similar to each other than are full-siblings. Future research should examine this possibility.

by the more restrictive model. For AGG, there was little evidence of etiological moderation by age. None of the individual moderators were statistically significant. Indeed, all linear and quadratic moderators could be fixed to zero (i.e., the most restrictive moderator model; Model 9) without a significant decrement in fit, indicating that the magnitude of genetic and environmental contributions to individual differences in AGG does not appear to vary across adolescence.

By contrast, there was evidence of significant etiological moderation by age for DEL. As can be seen in Table 2, neither the linear nor the quadratic A (genetic) moderators could be fixed to zero without a significant decrement in fit, suggesting that the magnitude of genetic influences on DEL varies both linearly and nonlinearly across age. Similarly, fixing the linear C (shared environmental) moderator to zero also resulted in a decrement in fit (note that although Model 7 demonstrated only a marginal decrease in fit relative to Model 1, the comparison with Model 5 is significant; $\chi^2 = 6.02$ on 1 *df*, $p = .014$). In sum, such findings imply that the magnitudes of genetic and shared environmental influences on DEL vary significantly across age, whereas non-shared environmental influences appear to remain constant across age.

For the best fitting models, we made use of the estimated paths and moderators (presented in Table 3) to calculate and plot (see Figure 3) the unstandardized genetic and environmental variance components at each age using the following equation: $\text{Variance}_{\text{Genetic}} = [a + A_1(\text{age}) + A_2(\text{age}^2)]^2$. For AGG, the plots are flat because neither the linear nor the quadratic moderators were statistically significant.[3] Put differently, because moderator parameters are not estimated in this model, the genetic and environmental estimates apply to all ages. Moreover, as indicated by the confidence intervals presented in Table 3 (confidence intervals that do not overlap with zero indicate statistical significance at $p < .05$), only genetic and nonshared environmental influences made statistically detectable etiological contributions to AGG (60% and 40% of the variance at each age, respectively). Shared environmental contributions were estimated to be zero and were not statistically significant.

For DEL, all parameters were significant at 10 years of age (i.e., a, c, and e), with shared environmental influences contributing the largest fraction of variance. There was also evidence of significant linear and nonlinear moderation of genetic influences by age, such that genetic variation increased in magnitude from early- to mid-adolescence, peaking in strength at 15 years of age, after which it slowly decreased. Shared environmental effects decreased significantly from early- to mid-adolescence, though they appear to increase slightly after 15 years of age. Nonshared environmental effects, by contrast, remained stable across adolescence.

[3]Because the AGG DZ correlation was less than half the MZ correlation, we also tested an ADE main effects model (in which A, D, and E represent genetic, dominant, and nonshared environmental parameters, respectively) for AGG, which estimates nonadditive or dominant (D) genetic effects in place of shared environmental (C) influences. Importantly, the D effect was estimated to be zero, indicating that all genetic influences on AGG are additive in nature. Indeed, the AE model (which does not include C or D effects) provided the best fit to the data, as indicated by the lowest AIC value (AE: $\chi^2 = 32.6$ on 16 *df*, AIC = 0.58; ACE: $\chi^2 = 32.6$ on 15 *df*, AIC = 2.58; ADE: $\chi^2 = 32.6$ on 15 *df*, AIC = 2.58). The lack of genetic dominance likely stems from the full-sibling and half-sibling correlations, which suggest additive genetic effects. Such findings highlight the added value of twin-sibling designs, such as this one, when examining genetic and environmental influences.

Longitudinal Changes in Genetic and Environmental Influences

We next sought to extend the results of our moderator analyses using a longitudinal design. Of note, because our moderator results indicate that the most pronounced etiological shifts occurred during early- to mid-adolescence, we restricted our longitudinal analyses to those 240 pairs in which both siblings were between the ages of 10 and 13 years at Time 1 (average age = 11.4 years, *SD* = 1.16). There were no differences in age of entry by family type for these participants ($p = .63$). At Time 2, these same participants ranged in age from 12 to 17 years (average age = 14.0 years, *SD* = 1.30). As a consequence, we were better able to capture etiological change during the key developmental period of early- to mid-adolescence. Importantly, comparisons of sibling pairs with data at Time 2 ($n = 195$ pairs; 81% of the Time 1 sample) with those without data at Time 2 ($n = 45$ pairs; 19% of Time 1 sample) revealed little evidence of differences in mean levels of DEL or AGG (both $ps \geq .14$), suggesting that the two groups evidence similar levels of aggressive and nonaggressive ASBs. Also of note, the 81% retention rate in this subsample is far higher than that for the NEAD Project sample as a whole (i.e., 56%), likely because the Time 2 assessment took place roughly 3 years after the Time 1 assessment, and the vast majority of 12- to 17-year-olds continued to live at home and thus remained eligible for participation. In short, the current subsample appears to be well suited for a longitudinal examination of AGG and DEL.

Descriptives—Paired *t*-test analyses indicated that mean levels of DEL increased significantly from Time 1 to Time 2 (Time 1: $M = 1.85$, $SD = 1.60$; Time 2: $M = 2.03$, $SD = 1.76$; means are significantly different at $p = .017$), whereas mean levels of AGG did not (Time 1: $M = 2.16$, $SD = 1.38$; Time 2: $M = 2.25$, $SD = 1.48$; means are not different, $p = .127$). Such results are consistent with prior literature in suggesting that DEL increases during adolescence.

Intraclass correlations at Times 1 and 2—Prior to longitudinal model-fitting analyses, intraclass correlations at both intake and follow-up assessments were calculated. Note that because we restricted these analyses to those younger than 14 years of age at Time 1, the Time 1 correlations are identical to those presented Table 1. These correlations offer a preliminary indication of genetic and environmental influences on DEL and AGG during each assessment period. As can be seen in Table 4, there are key differences across assessment and subtype, differences that mirror those in the cross-sectional data presented in Table 1. For DEL, the MZ–DZ difference is again approximately twice as large at Time 2 as compared with Time 1, implying that genetic influences on DEL may increase over the course of adolescence. Moreover, the correlation between step-siblings, which was moderate in magnitude and significant at Time 1, was small and not statistically significant at Time 2, again suggesting that shared environmental influences on DEL decrease across adolescence. By contrast, genetic and environmental influences in AGG did not appear to substantively change across adolescence, as there was little difference in the correlations across assessment. Such findings are thus consistent with our prior results, and offer a preliminary indication of differential change in genetic and environmental influences on AGG and DEL during adolescence.

Longitudinal models—We initially estimated variances, covariances, and means for the raw data to get a baseline index of fit for each subtype. The univariate biometric models were then fit both allowing for differences in parameter estimates across assessments and constraining parameter estimates to be equal across assessments.[4] As can be seen in Table 5, the better fitting model for AGG was the no-assessment-differences model, as indicated by the lower AIC value. As with the moderator analyses, such results suggest that the magnitude of genetic and environmental influences on AGG remain invariant across adolescence. By contrast, for DEL, the better fitting model was the assessment-differences model, indicating that the magnitude of genetic and environmental influences on DEL changes significantly as adolescents develop.

Parameter estimates from the better fitting models are presented in Table 6. Note that because the no-assessment-differences model fit better for AGG, estimates are identical across the two assessments. As before, AGG was found to be largely genetic in origin, although nonshared environmental influences also significantly contributed. Genetic and environmental influences on DEL, by contrast, changed significantly across assessments. In particular, although DEL appeared to be highly genetically influenced at both assessments, genetic influences were stronger at Time 2 as compared with Time 1. Indeed, constraining the A (genetic) parameter to be equal across assessments resulted in a significant decrement in fit (χ^2 = 88.44 on 41 *df*, AIC = 6.44). Shared environmental influences were moderate and statistically significant at Time 1 but were estimated to be zero at Time 2. Moreover, constraining the C (shared environmental) parameter to be equal across assessments resulted in a significant decrement in fit (χ^2 = 88.05 on 41 *df*, AIC = 6.05), further suggesting that shared environmental influences decrease from early- to mid-adolescence. By contrast, non-shared environmental influences did not change significantly across assessments (constraining the E [nonshared environmental] parameter across assessments improved the fit of the model; χ^2 = 85.25 on 41 *df*, AIC = 3.25). Such findings are notably consistent with our moderator results and further imply that genetic influences on DEL increase, whereas shared environmental influences decrease, over the course of adolescence. Moreover, this pattern does not extend to AGG.

Discussion

The aim of the present study was to evaluate the impact of age on the etiology of AGG versus DEL antisocial behaviors. Analyses revealed that AGG remained etiologically stable across adolescence, both within and across individuals, with no significant shifts in the magnitude of genetic or environmental variances with age. By contrast, DEL demonstrated pronounced shifts in the magnitude of genetic and shared environmental influences with age. Genetic influences on DEL essentially tripled in magnitude from 10 to 15 years of age, after which they slowly began decreasing. Shared environmental influences on DEL were strongest at 10 years of age and decreased to near-zero levels by 13 years of age. Critically, these results persisted when analyzing the longitudinal data, implying that results may

[4]Note that although the univariate model imposes little structure on the data, it did not provide an especially good fit for either subtype, as evidenced by the positive AIC values. Visual inspection of the variance–covariance matrices suggests that the misfit stems from unequal variances across family type.

represent true delinquency-specific, developmental shifts in genetic and environmental influences. Accordingly, such findings are thought to highlight marked and consistent differences between aggression and delinquency in the developmental timing of their genetic influences.

Though we know of no other study to explicitly evaluate the moderation of AGG and DEL by age, our findings are generally consistent with those of other twin and adoption studies of these constructs. We found evidence of strong genetic influences on aggression (roughly 60% of the variance), results that are notably consistent with the high heritabilities reported in most studies of aggression (Eley et al., 1999, 2003; van Beijsterveldt et al., 2003). Shared environmental influences were significant only for DEL, and they were estimated to be zero for AGG, findings that again generally replicate those of other studies (Deater-Deckard & Plomin, 1999; Eley et al., 1999, 2003; Tackett et al., 2005). However, there are some differences. Some studies have reported shared environmental influences on AGG (van Beijsterveldt et al., 2003), results that were not replicated here. However, several other studies have reported no shared environmental influences on AGG (Deater-Deckard & Plomin, 1999; Eley et al., 1999, 2003; Tackett et al., 2005). Further, the very high heritability of DEL in late adolescence is somewhat unique in the literature. Though the exact cause of this difference remains unclear, it may in part reflect our use of a multi-informant measure of delinquency, as recent studies have indicated that multi-informant indicators are more heritable than are single-informant indicators (Arsenault et al., 2003; Reiss et al., 2000). Alternately, as suggested in previous work (Rhee & Waldman, 2002), such differences may result from the prior lack of consideration given to age. Namely, collapsing across a broad range of ages may obfuscate the “true” heritability of nonaggressive delinquency at any given age.

There are several limitations to consider when interpreting the results of this study. In particular, though we examined the impact of age on the etiology of AGG and DEL, we did not examine how age-of-onset impacted the results. However, because of the close link between aggression and early-onset ASB, and between delinquency and adolescent-onset ASB (Moffitt, 2003), our findings are certainly related to Moffitt’s (1993) developmental taxonomy. Even so, the high heritability of delinquency is clearly not in keeping with Moffitt’s original hypothesis that adolescent-onset ASB is largely a function of peer mimicry. Of note, however, more recent research has suggested that adolescent-limited ASB may have been “underpathologized” in Moffitt’s original theory. Indeed, follow-up studies in early adulthood have revealed that adolescent-onset delinquents had not, as was proposed, desisted from ASB and become psychologically healthy by early adulthood. Instead, they (a) continued to commit low-level crimes such as property offenses (Moffitt, Caspi, Harrington, & Milne, 2002), (b) self-reported problems with mental health and substance abuse/dependence (Moffitt et al., 2002; Nagin, Farrington, & Moffitt, 1995), and (c) evidenced an impulsive personality style (i.e., unconventional, spontaneous, and sensation seeking; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). Consistent with this, other research has indicated that adolescent-onset ASB is at least partially genetically influenced (Burt & Mikolajewski, 2008; Moffitt, 2003). In this light, our finding of strong genetic influences on adolescent delinquency, particularly during mid-adolescence, may not be wholly

unexpected. In any case, future genetically informed research should evaluate the impact of age on early- versus late-onset ASB.

Second, given the large number of family types included in the NEAD Project, analyses incorporating gender in a meaningful way are unwieldy and underpowered (e.g., we have 93 MZ pairs at intake, of which only 46 are female–female pairs). Thus, gender was regressed out of AGG and DEL prior to analysis. Fortunately, prior studies (Burt, Krueger, McGue, & Iacono, 2003; Eley et al., 2003; Hudziak et al., 2003; Rhee & Waldman, 2002) have strongly suggested that heritability estimates for ASB do not vary significantly across gender, suggesting that our decision to exclude gender is unlikely to have impacted our results. That said, our measures did not include relational aggression, a form of aggression thought to have a higher prevalence in girls than boys, and one for which genetic and environmental contributions have only rarely been examined (Brengden et al., 2008). Accordingly, it may be that age and gender interact with one other when examining a broader (and perhaps more inclusive) index of aggression. Future analyses should incorporate relational aggression into analyses of gender and age. Similarly, there is a significant body of literature highlighting subtypes within AGG that were not considered here: proactive (i.e., premeditated, instrumental aggression) and reactive (i.e., impulsive, affective aggression; Barratt, Stanford, Kent, & Felthous, 1997; Davidson, Putnam, & Larson, 2000). It would thus be important for future research to consider whether these subtypes of AGG also evidence differential patterns of genetic expression.

Next, though they do appear to comprise separate but related indices of ASB (as indicated by our factor analyses), our measures of AGG and DEL are rather short and limited in content. Future studies should seek to replicate these findings using more comprehensive measures of AGG and DEL (e.g., the Child Behavior Checklist scales). Also of note, our inclusion of multiple family types with different degrees of genetic relatedness is considered an important strength of our study, as results therefore generalize to non-twin-siblings and adolescents in general. That said, although our requirement that siblings live together at least half time was necessary for us to accurately estimate genetic and environmental influences via the classical twin/sibling design, our results may not generalize to siblings residing in different homes. Next, we made use of a standard behavioral genetic (and structural equation modeling) approach to model fitting: dropping terms that are not statistically significant. Although this approach does not appear to bias the actual parameter estimates, it does serve to artifactually tighten their confidence intervals. Given this, the parameter confidence intervals are likely to be too narrow. That said, *all* parameters significantly greater than zero in the reported models (i.e., with confidence intervals that do not overlap with zero) were also significantly greater than zero when all parameters were estimated (i.e., in the full model).

Finally, though the results presented here highlight distinctions within the overarching construct of ASB, it should be noted that AGG and DEL demonstrate considerable overlap as well. Previous studies have found that at least 50% of individuals with childhood-onset aggressive behavior also exhibit clinically significant delinquent behaviors (Hudziak et al., 2003), results that were replicated here (50% overlap at Time 1, and 46% overlap at Time 2). How do we make sense of these seemingly incompatible results? Psycho-pathology

research provides some clues. Specifically, the comorbidity of mental disorders, once thought to be the exception, now seems to be the rule (Clark, Watson, & Reynolds, 1995) and has recently been conceptualized as evidence that core psychopathological processes link separate mental disorders (Kendler, Prescott, Myers, & Neale, 2003; Kendler et al., 1995; Krueger et al., 2002). In addition to these common processes, however, there is also evidence of causal processes that are disorder specific. For example, Krueger et al. (2002) found evidence of genetic and environmental influences *common* to conduct disorder, alcohol dependence, drug dependence, and the personality trait of constraint, as well as genetic and environmental influences *unique* to each phenotype. Accordingly, though our results highlight distinctions between aggression and delinquency in the timing of genetic expression, they do not rule out the possibility of common etiological influences contributing to their covariation and to the comorbidity between ASB and other externalizing spectrum disorders (including, and perhaps especially, comorbid attention problems). Future studies should explore these processes.

In spite of these limitations, the results of the current study yield several interrelated conclusions that inform both developmental psychology in general and developmental behavioral genetics more specifically. First, our results indicate that the increasing levels of delinquency within individuals and the increasing variation in delinquency across individuals during mid-adolescence is in large part a function of the increasing expression of genetic influences, findings that are generally consistent with those of Lyons et al. (1995). Such findings may reflect simple genetic main effects within the adolescent milieu. Namely, adolescence is a developmental period in which delinquency and rebellion against authority are tolerated and even encouraged (Moffitt, 2003), enabling those with these predilections to express them relatively openly. Alternately, as discussed in the introduction, the increasing genetic expression of delinquency during adolescence is suggestive of an active rGE process. One likely candidate for active rGE is that of affiliation with deviant peers, a well-documented risk factor for adolescent delinquency (Deater-Deckard, 2001). In particular, it may be that affiliation with deviant peers acts as a self-selected “trigger” for genetic predispositions toward nonaggressive delinquency (Bergen et al., 2007). Future research should seek to further examine this hypothesis.

Furthermore, there is some (albeit cross-sectional only) evidence that genetic influences on DEL begin to decrease in magnitude during late adolescence. Though these findings were not anticipated prior to analysis, they are provocative, as they suggest that the aforementioned increase in genetic influences on delinquency during mid-adolescence may be somewhat transient. This pattern can perhaps be best understood via Moffitt’s (1993) theory of adolescent-limited ASB, such that nonaggressive delinquency is largely specific to the developmental period of adolescence and dissipates during the transition into adulthood. The current findings are consistent with the notion that these developmental changes are mirrored at the genetic level, though such interpretations are speculative and should be examined more closely in future research.

By contrast, the magnitude of genetic influences on AGG remained constant across adolescence. Such findings imply that although genetic influences on nonaggressive delinquency may not fully express themselves until adolescence, this timeline does not apply

to all phenotypes. This finding is consistent with the notion that active rGE processes either do not impact aggression (instead, aggression may be better explained by genetic main effects or gene–environment interactions) or that they do so prior to adolescence.

Regardless, knowledge of the timing of genetic expression could inform both the search for relevant candidate genes as well as prevention work. For example, the current results suggest that prevention interventions designed to reduce physical aggression per se may be most effective in early- to mid-childhood, prior to the stabilization of genetic influences on this phenotype. Similarly, prevention interventions specifically targeting delinquency might focus on early adolescence given the simultaneous (and perhaps related) increased genetic expression and exposure to adolescent norms valuing delinquency during this developmental period. Accordingly, the timing of genetic expression should be pursued more rigorously in future research.

Finally, our results serve to further crystallize distinctions between aggressive and delinquent ASB. Genetic and environmental influences on aggression remained constant throughout adolescence, whereas genetic and environmental influences on delinquency changed dramatically during the same period. Such findings are consistent with the notion that the genetic and environmental influences on aggression are expressed early in life (i.e., prior to 10 years of age). Genetic influences on delinquency, by contrast, appear to be somewhat “weaker,” in that they express themselves later in life, may be somewhat transient, and may require a delinquent peer network as a trigger. Future etiological research should seek to further explore this process, perhaps via a developmental, molecular genetic design.

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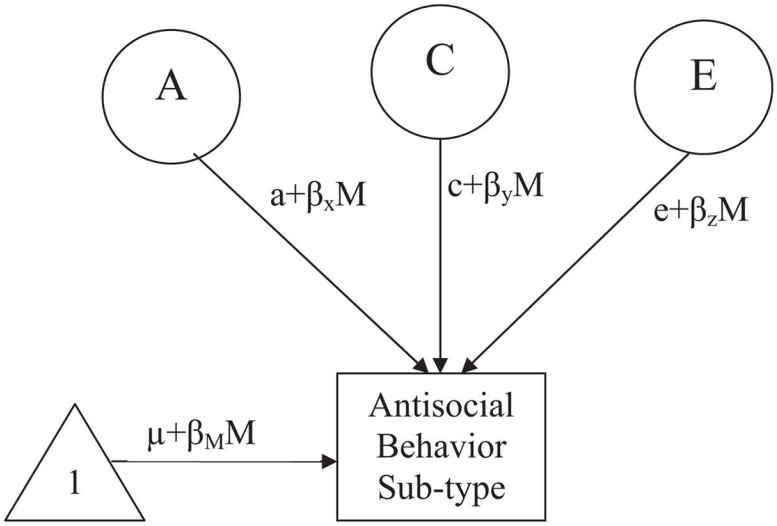

Figure 1.

Path diagram of the linear moderator model. Note: A, C, and E represent genetic, shared environmental, and nonshared environmental influences, respectively. Interactions with the moderator (i.e., $\beta_M M$; M is the moderator) are added to these genetic and environmental influences, and are estimated separately for each component of variance (i.e., $\beta_X M$, $\beta_Y M$, and $\beta_Z M$ for a, c, and e paths, respectively). For ease of presentation, the co-twin variables and paths are omitted here, though they are estimated in the model.

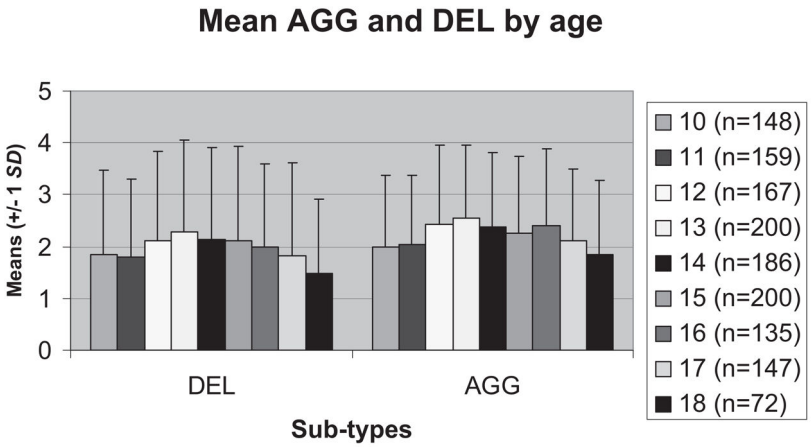

Figure 2.

Raw means of delinquency measures (±1 *SD*) across adolescence. Note: AGG and DEL represent aggressive and nonaggressive but delinquent antisocial behaviors, respectively. Means and standard deviations are presented at each age. The ranges of possible values are 0–12 for DEL and 0 – 8 for AGG.

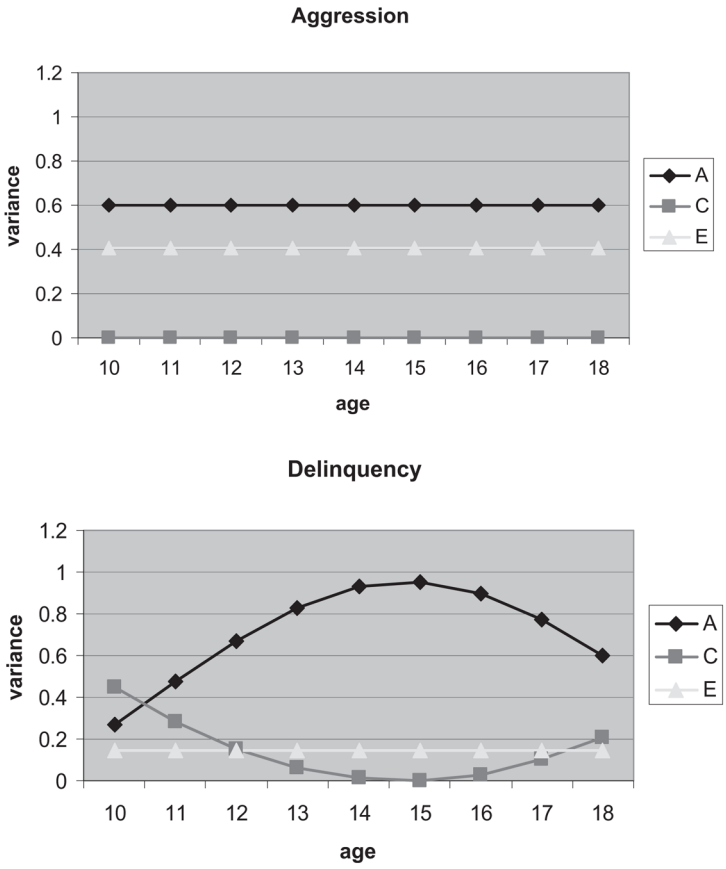

Figure 3.

Unstandardized variance components in aggressive (AGG) and delinquent (DEL) antisocial behavior across adolescence. Note: A, C, and E represent genetic, shared, and nonshared environmental variance components, respectively. These estimates index the absolute changes in genetic and environmental variance in AGG and DEL from 10 to 18 years of age.

Table 1

Sibling Intraclass Correlations for Delinquent (DEL) and Aggressive (AGG) Antisocial Behavior at Time 1 by Age Range

Sibling type	DEL	AGG	*N*
Younger siblings[a]			
MZ	.84**	.62**	46
DZ	.65**	.08	49
Full-siblings	.30**	.22**	77
Half-siblings	.40**	.26*	37
Step-siblings	.35**	.08	31
Older siblings[b]			
MZ	.82**	.48**	47
DZ	.42**	−.02	50
Full-siblings	.39**	.12	91
Half-siblings	.19	.18	24
Step-siblings	.07	.00	61

Note. Monozygotic (MZ) twins share 100% of their genetic material, dizygotic (DZ) twins and full-siblings share an average of 50% of their genetic material, half-siblings share an average of 25% of their genetic material, and step-siblings are genetically unrelated. *N* corresponds to the number of sibling pairs.

[a] Both siblings are between 10 and 13 years of age.

[b] Both siblings are between 14 and 18 years of age.

* Correlation is significantly larger than zero at $p < .05$.

** Correlation is significantly larger than zero at $p < .01$.

Table 2

Indices of Fit for a Series of Nested ACE Models Examining the Etiology of Aggressive (AGG) and Delinquent (DEL) Antisocial Behavior

Model	−2lnL	*df*	$\Delta\chi^2$	Δdf	*p*
AGG					
1. Full model (linear and quadratic moderation)	3,939.53	1403			
2. No quadratic A	3,939.53	1404	0.00	1	*ns*
3. No quadratic C	3,942.33	1404	2.80	1	*ns*
4. No quadratic E	3,939.53	1404	0.00	1	*ns*
5. No quadratic moderation	3,942.53	1406	3.00	3	*ns*
6. Model 5 and no linear A	3,943.08	1407	3.55	4	*ns*
7. Model 5 and no linear C	3,942.53	1407	3.00	4	*ns*
8. Model 5 and no linear E	3,945.70	1407	6.17	4	*ns*
9. No linear or quadratic moderation	**3,946.63**	**1409**	**7.10**	**6**	***ns***
DEL					
1. Full model (linear and quadratic moderation)	3,801.37	1403			
2. No quadratic A	3,806.94	1404	5.57	1	.02
3. No quadratic C	3,802.31	1404	0.94	1	*ns*
4. No quadratic E	3,801.55	1404	0.18	1	*ns*
5. No quadratic C or E	3,802.40	1405	1.03	2	*ns*
6. Model 5 and no linear A	3,812.10	1406	10.73	3	.01
7. Model 5 and no linear C	3,808.42	1406	7.05	3	.07
8. Model 5 and no linear E	**3,802.41**	**1406**	**1.04**	**3**	***ns***

Note. A, C, and E represent genetic, shared environmental, and nonshared environmental parameters, respectively. The fit indices for a series of nested ACE moderator models are presented for each subtype. The fit of each model is compared to that of the least restrictive model (i.e., Model 1; allows for linear and quadratic ACE moderation). Nonsignificant changes in chi-square indicate that the more restrictive model (i.e., the model with fewer estimated parameters and therefore more degrees of freedom) provides a better fit to the data. The best fitting model for each subtype is highlighted in bold font. Thus, for AGG, the best fitting model was the main effects model (i.e., there was no etiological moderation by age), whereas for DEL, the best fitting model allowed for linear and quadratic moderation of genetic influences and linear moderation of shared environmental influences.

Table 3

Unstandardized Path and Moderator Estimates in the Best Fitting Models for Aggressive (AGG) and Delinquent (DEL) Antisocial Behavior

	Paths			Linear			Quadratic		
Measure	**a**	**c**	**e**	**A_1**	**C_1**	**E_1**	**A_2**	**C_2**	**E_2**
AGG	−**.775** (−.856, −.665)	.000 (−.268, .268)	**.638** (.560, .726)						
DEL	**.517** (.317, .745)	**.672** (.412, .841)	−**.382** (−.442, −.334)	**.191** (.077, .283)	−**.141** (−.188, −.042)		−**.020** (−.030, −.006)		

Note. Paths and moderators are presented; their 95% confidence intervals are presented below them in parentheses. A, C, and E (both upper and lower case) represent genetic, shared, and nonshared environmental parameters, respectively. In the left portion of the table, the path estimates (i.e., a, c, and e) are presented. Because we subtracted 10 from each age prior to data analysis (setting the floor to 0), these path estimates function as intercepts. Accordingly, the genetic and environmental variance components at 10 years of age can be obtained simply by squaring these path estimates. At each subsequent age, significant linear (i.e., A_1, C_1, E_1) and nonlinear (i.e., A_2, C_2, E_2) moderators are added to these genetic and environmental paths using the following equation: Unstandardized $\text{Variance}_{\text{Total}} = [a + A_1(\text{age}) + A_2(\text{age}^2)]^2 + [c + C_1(\text{age}) + C_2(\text{age}^2)]^2 + [e + E_1(\text{age}) + E_2(\text{age}^2)]^2$. The variance component estimates calculated in this way are then presented in Figure 3, separately by subtype. Bold font indicates that the estimate is significant at $p < .05$.

Table 4

Sibling Intraclass Correlations for Aggressive (AGG) and Delinquent (DEL) Antisocial Behavior at Time 1 and Time 2

Time	DEL	AGG	*N*
Time 1[a]			
MZ	.84**	.62**	46
DZ	.65**	.08	49
Full-siblings	.30**	.22**	77
Half-siblings	.40**	.26*	37
Step-siblings	.35**	.08	31
Time 2[b]			
MZ	.78**	.61**	40
DZ	.44**	*.08	43
Full-siblings	.26**	.03	62
Half-siblings	*.02	.29*	28
Step-siblings	.14	*.05	21

Note. Monozygotic (MZ) twins share 100% of their genetic material, dizygotic (DZ) twins and full-siblings share an average of 50% of their genetic material, half-siblings share an average of 25% of their genetic material, and step-siblings are genetically unrelated. *N* corresponds to the number of sibling pairs. Time 1 correlations are also presented in Table 1 (see "younger siblings" section). Time 2 assessments took place 2–4 years after the intake assessment (all Time 2 participants had participated at Time 1).

[a] Age range = 10–13 years (average age = 11.4 years).

[b] Age range = 12–17 years (average age = 14.0 years).

* Correlation is significantly larger than zero at $p < .05$.

** Correlation is significantly larger than zero at $p < .01$.

Table 5

Model Fit Statistics for Longitudinal Models

Model	−2lnL	*df*	χ^2 (*df*)	AIC
	AGG			
Baseline	2,277.31	820		
Assessment-differences	2,368.08	860	90.77 (40)	10.77
No-assessment-differences	**2,368.93**	**863**	**91.62 (43)**	**5.62**
	DEL			
Baseline	2,201.70	820		
Assessment-differences	**2,286.46**	**860**	**84.76 (40)**	**4.76**
No-assessment-differences	2,293.91	863	92.21 (43)	6.21

Note. AGG and DEL represent aggression and delinquency, respectively. We initially estimated variances, covariances, and means for the raw data to get a baseline index of fit for each subtype (−2lnL). The −2lnL under the baseline model is then compared with −2lnL under more restrictive biometric models to provide a likelihood-ratio chi-square test of goodness of fit for the model, which is then converted to the Akaike information criterion (AIC). Better fitting models have lower AIC values. Biometric models were fit allowing for differences in parameter estimates across assessments (i.e., the assessment-differences model) and constraining parameter estimates to be equal across assessments (i.e., the no-assessment-differences model). The better fitting model is highlighted in bold font.

Table 6

Genetic and Environmental Contributions to Aggressive (AGG) and Delinquent (DEL) Antisocial Behavior at Time 1 and Time 2

Model	Time 1	Time 2
	AGG	
A	**.62 (64%)**	**.62 (64%)**
C	.00 (0%)	.00 (0%)
E	**.35 (36%)**	**.35 (36%)**
	DEL	
A	**.59 (64%)**	**.87 (82%)**
C	**.19 (20%)**	.00 (0%)
E	**.15 (16%)**	**.19 (18%)**

Note. A, C, and E represent genetic, shared, and nonshared environmental influences, respectively. To more precisely examine absolute changes in genetic and environmental variance over time, we present unstandardized estimates above (followed by standardized proportions in parentheses). On average, participants were 11.4 years of age at Time 1, and 14.0 years of age at Time 2. Estimates in bold font are statistically significant at $p < .05$.