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Differential Genetic and Environmental Influences on Developmental Trajectories of Antisocial Behavior from Adolescence to Young Adulthood

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Abstract

Little research has investigated differential genetic and environmental influences on different developmental trajectories of antisocial behavior. This study examined genetic and environmental influences on liabilities of being in life-course-persistent (LCP) and adolescent-limited (AL) type delinquent groups from adolescence to young adulthood while considering nonviolent and violent delinquency subtypes and gender differences. A genetically informative sample ($n = 356$, 15–16 years) from the first three waves of In-Home Interview of the National Longitudinal Study of Adolescent to Adult Health was used, with 94 monozygotic and 84 dizygotic pairs of same-sex twins (50% male). Biometric liability threshold models were fit and found that the male-specific LCP type class, *chronic*, showed more genetic influences, while the AL type classes, *decliner* and *desister*, showed more environmental influences. Genetic liability and shared environment both influence the persistence of antisocial behavior. The development of female antisocial behavior appears to be influenced more by shared environment.

Keywords

genetic influence; environmental influence; developmental taxonomic theory; antisocial behavior; adolescent; gender difference

Introduction

Developmental taxonomic theory posits that adolescence-limited (AL) delinquents manifest antisocial behavior mostly during adolescence and desist thereafter, whereas life-course persistent (LCP) delinquents continue to engage in moderate levels of antisocial behavior from early childhood into adulthood (Moffitt, 1993). AL delinquents primarily engage in nonviolent delinquency (e.g., shop lifting) and are mainly influenced by temporary environmental factors (e.g., deviant peer affiliation), whereas LCP delinquents engage more in violent delinquency (e.g., physical fights) and are more influenced by genetic liability,

neuropsychological problems, and criminogenic environment (DiLalla & Gottesman, 1989; Moffitt, 1993; 2006; 2008). In addition to these two groups that have been widely supported empirically (e.g., Barnes & Beaver, 2010; Moffitt, 2006; 2008; Piquero & Brezina, 2001), a group of individuals who are never or rarely delinquent (i.e., abstainer) has also been consistently identified (e.g., Boutwell & Beaver, 2008; Jennings & Reingle, 2012; Piquero, 2008; Piquero, Brezina, & Turner, 2005).

Substantial research has examined genetic and environmental influences on the development of antisocial behavior. Meta analyses and reviews reveal moderate additive genetic influences, modest shared environmental influences, and substantial non-shared environmental influences (Ferguson, 2010; Miles & Carey, 1997; Moffitt, 2005; Rhee & Waldman, 2002). Meta analyses also show decreasing familial influences (genetic and shared environmental influences) and increasing non-familial influences with increasing age (Ferguson, 2010; Rhee & Waldman, 2002), but others show increasing genetic influences and decreasing shared environmental influences (Miles & Carey, 1997).

Large scale population-based longitudinal twin studies have typically found that a common set of genetic factors and shared environmental factors can explain the persistence of antisocial behavior. For example, Tuvbald and colleagues (2011) found that a common genetic factor explained 67% of the variance of a latent persistent antisocial behavior factor, and 26% by a common shared environmental factor from childhood to young adulthood. Bartels et al. (2004) reported 60% genetic and 34% shared environmental influences on the stability of externalizing behavior from age 3 to 12 years. Silberg et al. (2007) found a common genetic factor explaining antisocial behavior from age 10 to 21 years, and a common shared environmental factor from age 14 to 21 years. Similar results were reported from age 10 to 17 years by Van Hulle et al. (2009) and from 8 to 20 years by Wichers et al. (2013). Lastly, using retrospective reports, Jacobson and colleagues (2002) reported that a single set of genetic factors influenced antisocial behavior from childhood (prior to 15 years) to adulthood (18 years and older).

The above-mentioned longitudinal studies primarily focused on changes in genetic and environmental influences across time, rather than differences in genetic and environmental influences on different developmental trajectories of antisocial behavior. The latter would require person-centered analyses that examine group differences, such as latent class growth analysis (LCGA; Nagin & Tremblay, 2005a; 2005b). Few studies have directly examined genetic and environmental influences on antisocial behavior between AL and LCP delinquents to test the developmental taxonomic theory (Fairchild, van Goozen, Calder, & Goodyer, 2013; Moffitt, 2006; 2008). An earlier study using a small sample of same-sex male twins reported .54 genetic influences and .30 shared environmental influences among early starters showing antisocial behavior, while no significant genetic influences among late starters (Taylor, Iacono, & McGue, 2000). Using the first 3 waves of In-Home Interview data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), Barnes, Beaver, and Boutwell (2011) reported .70 genetic influences on LCP delinquent membership, in contrast to .35 on AL delinquent and .56 on abstainer membership. Shared environmental influences did not significantly contribute to membership in either group.

Using all 4 waves of Add Health data, Barnes (2013) later reported .51 genetic influences and .49 non-shared environmental influences on LCP group membership.

Although demonstrating different genetic and environmental influences across different developmental trajectories, previous studies mostly classified different groups based on subjective cut-off criteria (e.g., top 10% or 20% for LCP in Barnes et al., 2011; onset of antisocial behavior before or after 12 years in Taylor et al., 2000) rather than adopting person-centered group-based modeling like LCGA. Second, many studies (e.g., Bartels et al., 2004; Tuvblad et al., 2011) did not consider different subtypes of antisocial behavior (e.g., violent vs. nonviolent delinquency), which have been found to influence trajectory identification (Fontaine et al., 2009; Jennings & Reingle, 2012; Zheng & Cleveland, 2013). Twin studies have also found violent/aggressive delinquency to be more heritable than nonviolent/nonaggressive delinquency (Burt, 2009; Burt & Neiderhiser, 2009), as well as different genetic and environmental influences in the development of aggressive and nonaggressive delinquency (Eley, Lichtenstein, & Moffitt, 2003). Third, some previous analyses were based on samples drawn from a wide age range (e.g., grades 7–12 in wave 1 in Barnes et al., 2011). Given the well-grounded age-graded development of antisocial behavior (Moffitt, 1993; Rutter, Giller, & Hagell, 1998), analyzing longitudinal patterns in a sample with such a broad range of ages potentially confounds the identification of developmental trajectories by allowing age-related differences and changes across waves to be incorrectly interpreted as between-individual differences. Estimates of genetic and environmental influences may also be inaccurate given their changes across ages (Burt & Neiderhiser, 2009; Ferguson, 2010; Miles & Carey, 1997; Rhee & Waldman, 2002).

Fourth, many previous studies either used a male-only sample (e.g., Taylor et al., 2000), or did not examine gender differences (e.g., Barnes et al., 2011). Despite males generally committing more antisocial behaviors than females, and being more likely to engage in violent delinquency (Moffitt, 2001; Rutter et al., 1998), little attention has been paid to gender differences in developmental trajectories of antisocial behavior. Studies have found that males and females differ in the prevalence of different trajectories, and some groups may be gender-specific (Fontaine et al., 2009). For example, Moffitt & Caspi (2001) reported that males are more likely to be in LCP group than females (10:1 ratio), but no major gender difference in the prevalence of AL group (1.5:1 ratio). Other studies have found the prevalence of the low/abstainer group to be higher in females than in males (e.g., Odgers et al., 2008). Particularly, Silverthorn and Frick (1999) reported a female-specific group, the adolescence-delayed-onset trajectory who shared similar risk factors to the male LCP group but did not demonstrate antisocial behavior until adolescence. These gender differences in developmental trajectories of antisocial behavior could be explained by two processes: socialization process that discourages females from antisocial behavior and higher levels of protective factors (e.g., parental supervision) in females (Fontaine et al., 2009; Silverthorn & Frick, 1999). Therefore, the decrease of parental supervision in adolescence may be linked with the emergence of antisocial behavior, particularly in females.

Findings are inconsistent regarding gender differences in genetic and environmental influences on antisocial behavior, with some supportive findings (Meier, Slutske, Heath &

Martin, 2011; Miles & Carey, 1997; Van Hulle et al., 2007; 2009) and null findings (Ferguson, 2010; Rhee & Waldman, 2002). Males and females appear to differ regarding the changes in age-specific genetic and environmental influences on antisocial behavior (Jacobson et al., 2002; Meier et al., 2011; Tuvblad et al., 2011; Van Hulle et al., 2009), and regarding the development of nonviolent and violent delinquency subtypes (Eley, Lichtenstein, & Stevenson, 1999; Tuvbald et al., 2005). For example, Jacobson et al. (2002) found higher genetic influences in females and stronger shared environmental influences in males in childhood, which however disappeared in adolescence and adulthood. Tuvbald et al. (2005) found that violent developmental pathway was genetically mediated in females, whereas non-violent developmental pathway was environmentally mediated in males from childhood to adolescence.

A recent study investigated developmental trajectories of antisocial behavior across the first 3 waves of Add Health data while also considering nonviolent and violent delinquency subtypes and gender differences using a group-based modeling on a homogeneous age group (15–16 years during the 1st wave) (Zheng & Cleveland, 2013). Four trajectories were identified: *low*, *desister*, *decliner*, and *chronic*. The *low* class resembled the low/abstainer class with very low levels of nonviolent and violent delinquency. Females had a higher prevalence (59%) than males did (50%). The *chronic* class was similar to LCP delinquents and was male-specific (13%). The *decliner* and *desister* classes resembled the AL pattern with similar prevalence in males (12% and 25%) and females (11% and 29%), although *decliner's* overall levels of antisocial behavior stayed relatively high compared to *desister*.

The Present Study

Building on the findings of Zheng & Cleveland (2013), the current study drew a genetically informative subsample and modeled genetic and environmental influences on liabilities of being in different trajectories described above. Specifically, the current study aimed to investigate gender difference in differential genetic and environmental influences on different developmental trajectories of antisocial behavior from adolescence to young adulthood while also considering nonviolent and violent delinquency subtypes. Informed by developmental taxonomic theory (Moffitt, 1993), we expected the LCP type delinquents, *chronic*, to be more influenced by genetic factors than other classes, whereas the AL type delinquents, the *desister* and *decliner*, to be more influenced by environmental factors. Second, given previous findings on gender differences in age-specific genetic and environmental influences (Jacobson et al., 2002; Tuvblad et al., 2011; Van Hulle et al., 2009) and in the development of nonviolent and violent delinquency subtypes (Eley et al., 1999; Tuvbald et al., 2005), we expected gender differences in genetic and environmental influences in different developmental trajectories of antisocial behavior. Specifically, as socialization processes and family protective factors discourage females from antisocial behavior (Fontaine et al., 2009; Silverthorn & Frick, 1999), we expected females to be more influenced by shared environment.

Methods

Sample

In the original study by Zheng and Cleveland (2013), 15–16 year-old participants ($n = 6,244$, 49.5% male, 53.4% non-Hispanic Whites) in the 1st wave (1994–1995) of Add Health In-Home Interviews were selected. Their developmental trajectories of nonviolent and violent delinquency from adolescence to young adulthood (16–17 years in 1996, 2nd wave, and 21–22 years in 2001–2002, 3rd wave) were modeled. The full Add Health twin data included 307 and 452 pairs of monozygotic (MZ) and dizygotic (DZ) twins (mean age = 15.3 years, range = 11–21 years, 51% males, 49% non-Hispanic Whites) in wave 1 with high retention rates in wave 2 (> 92%) and wave 3 (> 89%) (Harris, Halpern, Smolen, & Haberstick, 2006). The twin sample has been shown to be representative of the full Add Health sample with regard to age, ethnicity, maternal education (Jacobson & Rowe, 1999), urbanicity, and antisocial outcomes (Barnes & Boutwell, 2013). The current study drew a genetically informative subsample from the 15–16 years participants, which included 94 (male = 46) MZ and 84 (male = 44) DZ pairs of same-sex twins ($n = 356$, 50% male). Opposite-sex twins were excluded to avoid potential gender differences, and full/half siblings were excluded to avoid potential age confounds. More information on the design of Add Health study and its twin data are described in Harris et al. (2009), Harris et al. (2006), and Jacobson & Rowe (1999).

Measures

Nonviolent and violent delinquency—Nonviolent and violent delinquency measures used in Zheng & Cleveland (2013) covered a variety of behaviors typically measured in self-reported scale of delinquency and violence (Thornberry & Krohn, 2000), and were very similar to the measures used in other Add Health studies that have demonstrated acceptable reliability and validity (e.g., Guo, Roettger, & Cai, 2008; Hagan & Foster, 2003). However, only items consistently asked across all three waves were used to retain the comparability of nonviolent and violent delinquency measures across waves. Specifically, nonviolent delinquency was measured as the average score of the same 5 items (e.g., how often did you steal something worth less than 50 dollars in the past year?), and violent delinquency was measured as the average score of the same 3 items (e.g., how often did you take part in a fight where a group of your friends was against another group in the past year?) at each wave. All items' response ranged from 0 “never”, 1 “once or twice”, 2 “three or four times” to 3 “five times or more.” Cronbach was between .66 and .74 for nonviolent delinquency, and between .58 and .63 for violent delinquency, across three waves.

Class identification and liabilities—The subscale scores of nonviolent and violent delinquency over three waves (therefore a total of 6 scores) were analyzed with longitudinal latent class analysis (LLCA, Collins & Lanza, 2009) to identify latent homogenous classes or groups of similar developmental patterns of antisocial behavior. LLCA provides Maximum Likelihood (ML) estimates using the Expectation-Maximization (EM) algorithm, which includes participants in the analysis as long as one subscale score at one time is available (Collins & Lanza, 2009). Akaike's information Criterion (AIC) and Bayesian Information Criterion (BIC) was used to choose the most parsimonious model (for more

details in latent class modeling and identification, see Zheng & Cleveland, 2013). LLCA assigns each participant posterior probabilities (ranging from 0 to 1) of belonging to each identified class, which were used in the current study as outcomes to indicate class liability. Therefore, every male participant had a posterior probability for the *chronic*, *decliner*, *desister*, and *low* class, respectively. Each female participant had a posterior probability for the *decliner*, *desister*, and *low* class, respectively.

Analytic Strategy

Because of the male-specific *chronic* class and the gender differences in levels of nonviolent and violent delinquency in *desister* and *decliner* classes, models were fit separately by gender. Given the skewed nature of the posterior probabilities, consistent with previous studies (Fontaine, Rijdsdijk, McCrory, & Viding, 2010), each probability was first ordinalized into 4 ordinal values (0–3) using its three quartiles points which divided the distribution into 4 equal groups (e.g., values falling below 25% percentile were coded as 0; values falling between 50% and 75% were coded as 2). One benefit of using the posterior probability to construct class liability as opposed to directly assigning an individual to one class is that the uncertainty of class classification (e.g., an individual has a probability of 0.8 of being in one class) is retained (Clark & Muthén, 2009). Liability threshold model was fit to each ordinalized posterior probability with 3 thresholds, which assumes an underlying normally distributed liability (Neale & Maes, 2004). Figure 1 provides the diagram for univariate ACE liability threshold model. To briefly introduce the ACE model, or biometric model, variance of the latent liability (L) is partitioned into three independent components: A, additive genetic influences that make twins similar; C, shared environmental influences that make twins similar, such as the family context in which twins live together; and E, non-shared environmental influences that make twins different from each other, as well as measurement errors. The ordinalized posterior probabilities were modeled as manifest variables that loaded on the latent liability through 3 thresholds. The latent liability has factor loadings on three latent factors—A, C, and E. The three factor loadings (a, c, and e) were set to be equal across twins, requiring that each component's influence to be the same for each twin in a pair. The three liability thresholds were also constrained to be the same across twins. There were correlations between A and C of the same twin pair. The former was set at 1 for MZ twins because they share all of their segregating genes and 0.5 for DZ twins because on average they share half of their segregating genes. The latter correlation was set to 1 for both groups. The correlation of E was 0 because conceptually these non-shared environments make twins differ from one another and thus should not be correlated. Because r_{DZ} in the *chronic* class is less than half of r_{MZ} , an ADE model was also fit to this class, which models the dominant genetic influences (D; interactions of alleles at a locus). The correlation between D was 1 for MZ and 0.25 for DZ twins. The variance of each latent liability L was scaled to 1. Therefore, A, C, and E are a^2 , c^2 , and e^2 , respectively. In addition to the full ACE/ADE model, two reduced models, AE/DE and CE models, were also fit to choose more parsimony model. AIC (the lower the better) and χ^2 tests ($-2\log\text{-likelihood}$) between full and reduced models were used to compare nested models. A non-significant χ^2 test suggests a better fit of the reduced model over the full model. All biometric models were fit in R 3.1.1 (R Development Core Team, 2013) using package OpenMx 2.0 (Boker et al., 2011).

Results

Trajectory Results and Preliminary Analyses

To briefly summarize the patterns of identified trajectories, the *low* class had very low levels of nonviolent (e.g., .02 in wave 1 for both genders) and violent delinquency (e.g., .01 in wave 2 for both genders) across waves. The *chronic* class had moderate levels of nonviolent (e.g., .19 and .12 in wave 2 and 3) and violent delinquency (e.g., .45 and .59 in wave 1 and 2) across waves. The *desister* class had high levels of antisocial behaviors that decreased over time (e.g., .49 to .27 from wave 1 to 3 for male nonviolent delinquency; .38 to .05 for female violent delinquency). Similar pattern held for the *decliner* class, whose overall levels of antisocial behavior however were higher compared to the *desister* class (e.g., .97 to .36 from wave 1 to 3 for male nonviolent delinquency; .43 to .07 for female violent delinquency). While *decliner* males reported similar levels of nonviolent and violent delinquency (e.g., .97 and .95 in wave 1), *decliner* females reported more nonviolent delinquency than violent delinquency (e.g., .75 and .43 in wave 1). In contrast, *desister* females reported more violent delinquency than nonviolent delinquency (e.g., .38 and .20 in wave 1), whereas *desister* males reported the opposite pattern (e.g., .31 and .49 in wave 1) (for more details, see Zheng & Cleveland, 2013).

The posterior probabilities of each class by gender in the current subsample (see Table 1) are comparable to those of the original sample (e.g., .25 for *desister* males vs. .25 in the original sample; .52 for *low* females vs. .59 in the original sample; .11 for *chronic* males vs. .13 in the original sample). As the polychoric cross-twin correlations show (see Table 2), in general, except for male *desister* class (but *ns*), MZ twins had higher correlations than DZ twins did. However, the difference in correlations was minimal except for the *chronic* class, which suggested modest, if any, genetic influences on the liability of being in all classes but substantial genetic influences for the *chronic* class.

Biometric Model Results

The full ACE model estimated modest A (males: .07; females: .18), moderate C (males: .35; females: .55) and E (males: .59; females: .28) on the liability of being in the *low* class (see Table 3). Compared to the full model, the CE model fit the best with the lowest AIC for both males (AIC = 132.09) and females (AIC = 102.71), and did not significantly decrease the model fit, $\chi^2(1) = .03$ and .41, *ns*, respectively. The estimated C and E were .40 and .60 for males, .68 and .32 for females. A further test constraining C and E to be the same across gender was marginally significant, suggesting a worse fit, $\chi^2(2) = 4.81$, $p = .08$ and a higher AIC (235.61 vs. 234.80).

For the male-specific class, the liability of being in the *chronic* class was best characterized by the DE model with the lowest AIC (111.66). The DE model did not significantly decrease the model fit from the ADE model, $\chi^2(1) = .00$, *ns*, and provided the same point estimates as in the ADE model. The estimated D and E were .31 and .69.

For females, the ACE model estimated negligible A (.02), substantial C (.60) and E (.38) on the liability of being in the *decliner* class. The CE model had the lowest AIC (112.65), and did not fit the data significantly worse than the full model, $\chi^2(1) = .00$, *ns*. Estimated C

and E were close: .61 and .39, respectively. For males, the liability of being in the *decliner* class was also fit best by the CE model (AIC = 129.75), $\chi^2(1) = .32$, *ns*. Estimated C and E were .42 and .58, respectively. However, it is worth noting that the AIC for the AE model was very close as well (129.92), also with a non-significant decrease of model fit, $\chi^2(1) = .49$, *ns*. A more conservative look into the full model revealed A, C, and E to be .23, .25, and .53, respectively. However, the 95% CIs of A and C cover zero. Constraining C and E to be the same across gender did not provide a worse fit, $\chi^2(2) = 2.12$, *ns*, with a lower AIC (240.53 vs. 242.40). The estimated C and E across gender were .52 and .48, respectively.

Lastly, the full ACE model estimated negligible A (males: .00; females: .06), moderate C (males: .30; females: .32) and E (males: .70; females: .62) on the liability of being in the *desister* class. The CE model had the lowest AIC for both males (AIC = 103.38) and females (AIC = 103.38), $\chi^2(1) = .00$ and .02, *ns*, respectively. The estimated C and E were .30 and .70 for males, .36 and .64 for females. Constraining C and E to be the same across gender did not provide a worse fit, $\chi^2(2) = .14$, *ns*, with a lower AIC (202.9 vs. 206.76). The estimated C and E across gender were .33 and .67, respectively.

Discussion

Despite previous studies of genetic and environmental influences on the development of antisocial behavior, few studies have directly examined how these influences may differentially contribute to different developmental trajectories. Consistent with expectations derived from the developmental taxonomic theory (Moffitt, 1993), the liability of being in the *chronic* class, which was similar to LCP delinquents or “continuous antisocials” (DiLalla & Gottesman, 1989) in that they continuously committed moderate levels of antisocial behaviors and mostly engaged in violent delinquency, was more influenced by genetic factors than were other classes. In fact, in the most parsimonious model, the *chronic* class was the only one under significant genetic influences. This finding indicates that genetic predispositions play an important role in the continuous commission of delinquent and violent acts from adolescence to young adulthood (Moffitt, 1993; 2006; 2008), and is consistent with prior studies showing violent delinquency to be more heritable (Burt, 2009; Burt & Neiderhiser, 2009). It is important to note that the strong genetic influences on the *chronic* class are driven in part by a low and non-significant DZ correlation rather than a particularly high MZ correlation. The low DZ correlation for the *chronic* class suggests that DZ twins are highly discordant and different from each other regarding this particular class that is male-specific and the smallest among all classes.

As expected, the liability of being in the *desister* and *decliner* classes, which were similar to the AL delinquents or “transitory delinquents” (DiLalla & Gottesman, 1989), was more influenced by environmental factors than was the *chronic* class. The absence of substantial genetic influences on liability for being in *desister* and *decliner* classes suggests that environmental influences have a major role in temporary adolescent delinquency, including both non-shared environmental influences such as deviant peer affiliation possibly due to maturity gap (e.g., Barnes & Beaver, 2010), and shared environmental influences (e.g., inconsistent or harsh parenting, disruptive family life, and poor neighborhood quality) (Moffitt, 1993; 2006; 2008; Patterson, Reid, & Dishion, 1992; Rutter et al. 1998).

Liability of being in the *low* class, which resembled the low/abstainer class (e.g., Boutwell & Beaver, 2008; Piquero, 2008; Piquero et al., 2005) with very low levels of nonviolent and violent delinquency, was primarily explained by environmental influences for both genders. However, the primary source of environmental influences varied, with males due to non-shared environmental influences and females due to shared environmental influences. Perhaps the more normative nature of male delinquency, particularly during adolescence, increases the salience of males' peer influences—a major source of non-shared experience (Patterson et al., 1992). Among females, however, the abstinence from delinquency from adolescence to young adulthood is tied more closely to family processes (Fontaine et al., 2009; Silverthorn & Frick, 1999).

It is interesting to note that across all three classes common to males and females, the estimates of shared environmental influences in females were consistently higher than in males: .68 vs. .40 for the *low* class, .61 vs. .42 for the *decliner* class, and .36 vs. .30 for the *desister* class. However, this gender difference in shared environmental influences was only marginally significant ($p = .08$) in the *lower* class and non-significant in the other two classes. The findings differ from some previous studies that found more shared environmental influences in males (Tuvbald et al., 2005; 2011), or no difference (Jacobson et al., 2002) during adolescence, but are consistent with some others (Bartels et al., 2004). The findings add preliminary evidence to the literature that genetic and environmental influences can differentially (at least in the *lower* class) contribute to the developmental patterns of antisocial behavior across gender (Meier et al., 2011; Van Hulle et al., 2007; 2009), and are congruent with the ideas that socialization process and family protective factors (e.g., parental supervision) are especially relevant in the development of antisocial behavior in females (Fontaine et al., 2009; Silverthorn & Frick, 1999).

Current findings are generally consistent with previous large scale longitudinal twins studies that found common genetic and shared environmental influences in the persistence of antisocial behavior from childhood to young adulthood (e.g., Bartels et al., 2004; Jacobsen et al., 2002; Silberg et al., 2007; Tuvbald et al., 2011; Wichers et al., 2013; Van Hulle et al., 2009). Additionally, the current study found that genetic influences play a relatively more important role in the continuously moderate antisocial behavior that defines the *chronic* class, whereas shared environmental influences are more relevant in the continuously low antisocial behavior in the *low* class. It is important to note that both *decliner* and *chronic* delinquents continued to commit antisocial behavior into young adulthood in the sample. Despite decreasing, *decliner* delinquents actually committed the most delinquent and violent acts and were therefore as problematic as the *chronic* class (Zheng & Cleveland, 2013). The findings that liability for *chronic* class was more influenced by genetic factors while liability for *decliner* class was more influenced by shared environmental factors suggest that both genetically predisposed and shared environmentally mediated pathways can lead to the development of problematic patterns of antisocial behavior from adolescence to young adulthood (e.g., Tuvbald et al., 2005). Taken together, this study furthered our understandings of gender differences in genetic and environmental influences on developmental trajectories of antisocial behavior. Genetic and environmental influences both matter in the development of antisocial behavior, but to different degrees in different developmental trajectories. Environmental influences on developmental trajectories differ

across gender. This study is novel in important ways. Although prior work have used behavioral genetic techniques to examine genetic and environmental influences on antisocial behaviors (Barnes, 2013; Barnes et al., 2011), and have used sophisticated approaches to empirically estimate developmental trajectories while distinguishing violent and nonviolent delinquency and considering gender differences (Zheng & Cleveland, 2013), the current study is the first to apply genetically informed analyses to developmental trajectories empirically derived. As analyses were built upon the study by Zheng & Cleveland (2013), they also considered violent and nonviolent delinquency subtypes and gender differences.

Although current study's findings are generally consistent with prior studies that used similar approaches, there are notable differences. For example, Taylor et al. (2000) also found some evidence of shared environmental influences (.30, but marginally significant) for LCP pattern delinquency. Barnes et al. (2011) reported genetic influences in the AL delinquents and abstainers but no shared environmental influences. Methodological differences most likely lead to these different results. Despite these differences in the results, the current study's finding of significant shared environmental influences on the liability of being in each class, except for the *chronic* class, is to some extent assuring, as meta-analyses and reviews generally show modest shared environmental influences in antisocial behavior (Ferguson, 2010; Miles & Carey, 1997; Moffitt, 2005; Rhee & Waldman, 2002). Finally, our results are very similar to an earlier report by Jacobson and colleagues (2001) using 1070 adult same-sex male twin pairs. Genetic influences on adolescent antisocial behavior (prior to 18 years) were significantly higher among those with adult antisocial behavior (18 years and older; $a^2 = .38$; $c^2 = .10$) than those who were not antisocial in adulthood ($a^2 = .00$; $c^2 = .35$).

Strengths, Limitations, and Suggestions for Future Studies

The current study has several methodological advantages over previous studies. Estimation of developmental trajectories for the twin subsamples were based on the original Add Health sample, ensuring the generalizability of the twin results to the general population (Barnes & Boutwell, 2013; Jacobson & Rowe, 1999). By using the results of Zheng and Cleveland's (2013) person-centered group-based modeling, this study was able to compare genetic and environmental influences on developmental trajectories based on empirically-derived criteria as opposed to subjective cut-offs. Using posterior probabilities to construct class liability rather than dichotomously assigning individuals to one class accounts for the uncertainty of class classification. Using a homogenous age group and separately examining trajectory across gender avoided potential trajectory identification issues (Fontaine et al., 2009; Jennings & Reingle, 2012; Zheng & Cleveland, 2013), and enabled examination of gender differences in genetic and environmental influences. Considering both nonviolent and violent delinquency provided the opportunity to investigate genetic and environmental influences at a more refined level on different trajectories jointly defined by the levels of nonviolent and violent delinquency.

The current study has limitations that should be addressed in future studies. First, group-based model results are data-driven and individuals' group memberships are not immutable (Nagin & Tremblay, 2005b). Therefore, the meaning of identified groups depends on sample

characteristics. In this regard, results using subjective cut-offs have the advantage of being theoretically-informed (see Moffitt, 1993), and thus potentially better able to directly inform theory. Relatedly, fitting liability threshold models to ordinalized posterior probability only represents one way to utilize latent class analysis results. Alternative techniques such as probability and probability-weighted regression may also be considered (Clark & Muthén, 2009). Second, although using a larger sample size compared to Taylor et al. (2000), the power of this study to detect relative small effect sizes is limited. The small sample may partly explain the null finding of genetic influences in other classes except for the *chronic* class, and null finding of shared environmental influences in the *chronic* class which should not be trivial according to the developmental taxonomic theory. The issue of power could partly be seen in the results for male *decliner* class where the full model suggested comparable but non-significant genetic and shared environmental influences, and the two reduced models provided comparable model fit. Large scale prospective longitudinal studies are needed to test and replicate the current findings. Third, the Add Health study started when participants were in adolescence. No information on childhood antisocial behavior was available. Therefore, we only examined persistent and continuous delinquents from adolescence to young adulthood, and were not able to directly examine LCP and AL delinquent trajectories, which require data starting from childhood. This was one of the reasons identified trajectories were labeled *chronic*, *desister*, and *decliner* instead. Future studies following the development of antisocial behavior from childhood to young adulthood can provide a more rigorous examination. Fourth, the validity of developmental taxonomic theory has been recently challenged (Fairchild et al., 2013). More studies are needed to look into the childhood-only and late-onset types of delinquents, and to examine how genetic liability and environmental risk factors interact to influence the development of antisocial behavior. Fifth, the current study used the same items across waves to ensure that measures of nonviolent and violent delinquency and their meanings are comparable across waves. However, choosing to use all available items at each wave, while disregarding the fact that different items being used may create difficulties in interpreting some results, may also provide opportunities for future analyses to consider different delinquent behaviors as they vary across development.

Conclusion

The current study contributes to our understanding of genetic and environmental influences on different developmental trajectories of antisocial behavior from adolescence to young adulthood, and highlights the importance of considering gender difference and nonviolent and violent delinquency subtypes when studying the development of antisocial behavior. Genetic liability and shared environmental influences both influence the persistence of antisocial behavior. Genetic influences are relatively more important in the continuously moderate antisocial behavior that defines the *chronic* class, whereas shared environmental influences are more relevant in the continuously low antisocial behavior as in the *low* class, and in temporary adolescent delinquent patterns as in the *desister* and *decliner* classes. The development of antisocial behavior in females appears to be tied more closely to influences associated with differences between families.

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Fig. 1. univariate ACE liability threshold model

A: additive genetic influences. C: shared environmental influences. E: non-shared environmental influences. L: latent liability of being in one class. MZ: monozygotic twins. DZ: dizygotic twins.

Table 1Means (*SD*) of latent trajectory probabilities by gender and zygosity

		DZ	MZ	overall
class		<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
		<i>n</i> = 88	<i>n</i> = 92	<i>n</i> = 180
males	low	0.51 (0.42)	0.55 (0.42)	0.53 (0.42)
	decliner	0.12 (0.27)	0.10 (0.25)	0.11 (0.26)
	desister	0.22 (0.31)	0.28 (0.34)	0.25 (0.33)
	chronic	0.15 (0.28)	0.08 (0.20)	0.11 (0.24)
		<i>n</i> = 80	<i>n</i> = 96	<i>n</i> = 176
females	low	0.48 (0.42)	0.55 (0.40)	0.52 (0.41)
	decliner	0.20 (0.34)	0.13 (0.26)	0.16 (0.30)
	desister	0.33 (0.34)	0.31 (0.32)	0.32 (0.33)

Table 2

Polychoric cross-twin correlations (95% confidence intervals) by gender and zygosity

	class	MZ	DZ
males	low	0.41 (0.11–0.64)	0.38 (0.03–0.64)
	decliner	0.47 (0.18–0.68)	0.36 (0.01–0.62)
	desister	0.23 (–0.13–0.52)	0.37 (0.04–0.62)
	chronic	0.33 (–0.01–0.59)	–0.03 (–0.36–0.31)
females	low	0.72 (0.49–0.86)	0.64 (0.36–0.81)
	decliner	0.62 (0.34–0.79)	0.61 (0.32–0.79)
	desister	0.38 (0.01–0.65)	0.35 (–0.01–0.63)

Table 3

Estimates of genetic and environmental influences (95% confidence intervals) and AICs from univariate liability threshold model by gender

Class	Model	A	C/D	E	AIC
Males					
low	ACE	0.07 (0.00–0.64)	0.35 (0.00–0.59)	0.59 (0.36–0.82)	134.07
	AE	0.46 (0.19–0.66)	--	0.54 (0.34–0.81)	133.01
	CE	--	0.40 (0.18–0.59)	0.60 (0.41–0.83)	132.09
chronic	ACE	0.26 (0.00–0.53)	0.00 (0.00–0.34)	0.74 (0.47–1.00)	114.29
	ADE	0.00 (0.00–0.51)	0.31 (0.00–0.58)	0.69 (0.42–1.00)	113.66
	AE	0.26 (0.00–0.53)	--	0.74 (0.47–1.00)	112.29
	CE	--	0.15 (0.00–0.38)	0.85 (0.62–1.00)	113.46
decliner	DE	--	0.31 (0.00–0.58)	0.69 (0.42–1.00)	111.66
	ACE	0.23 (0.00–0.68)	0.25 (0.00–0.60)	0.53 (0.32–0.79)	131.43
	AE	0.50 (0.24–0.69)	--	0.50 (0.31–0.76)	129.92
	CE	--	0.42 (0.20–0.60)	0.58 (0.40–0.80)	129.75
desister	ACE	0.00 (0.00–0.52)	0.30 (0.00–0.51)	0.70 (0.47–0.94)	105.38
	AE	0.32 (0.03–0.56)	--	0.68 (0.44–0.97)	104.97
	CE	--	0.30 (0.07–0.51)	0.70 (0.49–0.94)	103.38
Females					
low	ACE	0.18 (0.00–0.79)	0.55 (0.00–0.80)	0.28 (0.14–0.48)	104.30
	AE	0.76 (0.57–0.87)	--	0.25 (0.13–0.43)	105.91
	CE	--	0.68 (0.51–0.80)	0.32 (0.20–0.49)	102.71
decliner	ACE	0.02 (0.00–0.69)	0.60 (0.00–0.76)	0.38 (0.21–0.58)	114.65
	AE	0.67 (0.46–0.81)	--	0.33 (0.19–0.54)	116.54
	CE	--	0.61 (0.42–0.76)	0.39 (0.25–0.58)	112.65
desister	ACE	0.06 (0.00–0.65)	0.32 (0.00–0.58)	0.62 (0.35–0.89)	105.36
	AE	0.43 (0.12–0.67)	--	0.57 (0.33–0.88)	104.04
	CE	--	0.36 (0.11–0.58)	0.64 (0.42–0.89)	103.38

Note. The most parsimony models with the lowest Akaike's Information Criterion (AIC) were bolded. A: additive genetic influences. D: dominant genetic influences. C: shared environmental influences. E: non-shared environmental influences.