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Further Evaluating the Relationship Between Adverse Childhood Experiences, Antisocial Behavior, and Violent Victimization: A Sibling-Comparison Analysis

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Abstract

A developing line of research suggests that adverse childhood experiences (ACEs) increase the risk for antisocial behavior and future victimization. However, the mechanisms that underlie this association remain largely speculative. To address this gap in the existing body of research, data on full siblings from a large population-based sample of youth were analyzed to evaluate the direct effect of ACEs on child antisocial behavior, adolescent delinquency, and young adult violent victimization after controlling for familial confounders. Traditional between-family analyses revealed that ACEs were significantly associated with higher levels of childhood antisocial behavior, adolescent delinquent behavior, and risk for violent crime victimization. After controlling for unmeasured common genetic and shared environmental confounds using fixed-effect sibling comparisons, siblings exposed to more ACEs did not demonstrate higher levels of antisocial behavior, delinquent behavior, or risk for future victimization. The implications of these results for future ACEs research are discussed.

Keywords

adverse childhood experiences, antisocial behavior, violent victimization, familial confounds, family-based research designs, NLSY

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> We may be through with the past, but the past is not through with us.
>
> Evans (1946)

Research regarding the effect of adverse childhood experiences (ACEs) on later-life health and human development has grown at a rapid rate over the past decade. This growing body of research can be traced back to the landmark Center for Disease Control-Kaiser ACE study conducted by Felitti and colleagues (1998), which examined the impact of ACEs on health-related outcomes in adulthood. Results from Felitti et al. (1998) showed, for the first time, that ACEs increase risk for cancer, heart disease, lung disease, liver disease, and early death. Recently, criminologists have begun to apply the ACE framework to the study of individual differences in antisocial behavior and criminal victimization. Several studies have documented a dose–response relationship between ACEs and a wide range of antisocial behaviors including childhood externalizing problems (Hunt, Slack, & Berger, 2017), preadolescent delinquent behavior (Hambrick, Rubens, Brawner, & Taussig, 2017), violent delinquent behavior (Fox, Perez, Cass, Baglivio, & Epps, 2015), juvenile arrest (Fagan & Novak, 2017), juvenile offending trajectories (Baglivio, Wolff, Piquero, & Epps, 2015), juvenile recidivism (Wolff & Baglivio, 2016; Wolff, Baglivio, & Piquero, 2017), and life-course offending (Craig, Piquero, Farrington, & Ttofi, 2017). Studies also report a positive association between ACEs and future victimization (Ports, Ford, & Merrick, 2016; Widom, Czaja, & Dutton, 2008). Taken together, accumulated evidence indicates that while individuals may be done with ACEs, such experiences may not be done with them and continue to exert an effect on their health and behavior.

While evidence of a robust association between ACEs, antisocial behavior, and future victimization is mounting, there are some important limitations that warrant further examination. First, evidence from contemporary research on ACEs within criminology is largely based on data from at-risk samples of delinquent youth, making it difficult to establish whether findings are generalizable to more representative youth samples. As a result, it is currently unknown whether reported results can be generalized to nonoffending populations in order to inform evidence-based intervention/prevention programing efforts. Second, while existing research has used a range of multivariate statistical techniques to control for measurable confounds, no research within criminology has used a quasi-experimental, genetically informed research design to control for unobservable genetic and environmental confounds that may partly explain the relationship between ACEs, antisocial behavior, and future victimization. Of primary theoretical significance to this point, a long line of quantitative behavioral genetic research has now shown that correlations between environmental exposure and individual's genetic risk for antisocial behavior and victimization are pervasive across the life course (Barnes & Beaver, 2012; Connolly & Beaver, 2015; D'Onofrio et al., 2016; Jaffee & Price, 2012; Kendler & Baker, 2007; Schwartz, Solomon, & Valgardson, 2017). A failure to control for familial confounds may therefore inflate associations between early-life exposures (i.e., ACEs) and life outcomes (i.e., antisocial behavior and victimization). Thus, genetically informed research designs are greatly needed in criminology to allow researchers to move from identifying risks to examining mechanisms (genetic and/or environmental) by which a risk factor may potentially *cause* future antisocial behavior or risk for victimization.

With this in mind, the current study seeks to address the abovementioned limitations by (1) analyzing longitudinal data from a population-based sample of youth from the United States to examine the prevalence of ACEs and compare rates to previous research and (2) using traditional between-family analyses and sibling-comparison analyses to help disentangle co-occurring genetic and environmental processes between ACEs, antisocial behavior in childhood, delinquent behavior in adolescence, and violent victimization in young adulthood. The overall rationale for this study is to use a rigorous quasi-experimental method, such as the sibling-comparison design, to help specify the processes behind commonly observed associations between ACEs, antisocial behavior,

delinquency, and violent crime victimization. A better understanding of these underlying processes will help to advance criminological theory and inform intervention/prevention programming.

ACEs, Antisocial Behavior, and Violent Victimization

Few topics in criminology have garnered as much empirical attention, theoretical interest, and debate as the study of child maltreatment and antisocial behavior. Some of the most prominent criminological theories posit that early-life exposure to abusive and inconsistent parenting may lead to maladaptive behavior in children, which in turn increases risk for antisocial behavior (Agnew, 1992, 2001; Gottfredson & Hirschi, 1990). Agnew's (1992) general strain theory holds that long-term exposure to stressful stimuli (e.g., emotional, physical, verbal, or sexual abuse) increases a child's risk for engaging in delinquent behavior to cope with this stress, especially if a child has problems regulating their emotions, such as anger. Gottfredson and Hirschi's *General Theory of Crime* (1990) posits that poor parenting techniques before age 8 aimed at teaching children how to properly regulate their emotional impulses will eventually lead to lower levels of self-control and behavior that is dictated more by emotional impulse than rational calculation, thus increasing the likelihood of criminal and/or delinquent offending. The common thread that ties these two theoretical frameworks together is an emphasis on the formative role of early-life familial factors on child behavior. Felitti et al.'s (1998) ACE framework along with recent conceptualizations (Baglivio et al., 2015) has extended this line of hypothesizing by suggesting that the following 10 ACEs experienced before age 18 increase the probability of developing problematic health and human behaviors later in life: (1) physical abuse, (2) emotional abuse, (3) sexual abuse, (4) physical neglect, (5) emotional neglect, (6) household substance use, (7) family violence, (8) parental separation or divorce, (9) household mental illness, and (10) having a household member incarcerated.

To date, several studies have reported that ACEs tend to be highly intercorrelated (Cicchetti, 2013) and, when factored together, are a robust predictor of chronic disease (Dong, Dube, Felitti, Giles, & Anda, 2003; Dong et al., 2004; Felitti et al., 1998), substance abuse (Dube, Anda, Felitti, Edwards, & Croft, 2002; Dube, Felitti, Dong, Chapman, et al., 2003), and several other negative life outcomes (Dube, Felitti, Dong, Giles, & Anda, 2003). Given the increasing support for the detrimental effect of ACEs, recent research within criminology has begun to examine the unique impact of ACEs on delinquency and chronic offending across the life course. For example, an analysis of 22,575 adjudicated delinquents from the Florida Department of Juvenile Justice by Fox, Perez, Cass, Baglivio, and Epps (2015) found that delinquents with more ACEs were more likely to become serious, violent, and chronic juvenile offenders. Another study analyzing the same sample found that the number of ACEs a juvenile was exposed to distinguished between both early onset and chronic patterns of offending from other patterns of offending (Baglivio et al., 2015). Other research examining a sample of males drawn from the Cambridge Study in Delinquent Development showed that ACEs increased the likelihood of offending over time (Craig et al., 2017), while another study analyzing youth from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN) sample reported that the number of ACEs was positively associated with the likelihood of arrest at age 16 among African American adolescents (Fagan & Novak, 2017).

Other studies have reported that ACEs are positively associated with different forms of victimization (Desai, Arias, Thompson, & Basile, 2002; Dube et al., 2001; Ports et al., 2016; Whitfield, Anda, Dube, & Felitti, 2003; Widom, 1995). A recent study by Ports, Ford, and Merrick (2016) found that ACEs before age 18 increased the risk for reporting sexual victimization in adulthood in a large sample of Health Maintenance Organization members, while another analysis by Voith, Anderson, and Cahill (2017) found that different types of child abuse were associated with victimization and perpetration in adulthood in a convenience sample of college males. Based on this body of evidence, it is possible that exposure to ACEs may make youth more susceptible to future

victimization outside of the household environment through the development of antisocial behavior and other forms of psychopathology. Evidence for this possibility is beginning to emerge with studies showing that high levels of anxiety (Fisher et al., 2012), poor self-control (Avakame, 1998), and depression (Fisher et al., 2012) partly mediate the effect of ACEs on future victimization. However, no study to date has examined the extent to which ACEs are associated with risk for serious violent crime victimization in emerging adulthood—a life-course period when violent victimization as been shown to increase in the United States (Truman & Morgan, 2017).

Conceptual Models for the Relationship Between ACEs, Antisocial Behavior, and Violent Victimization

The premise behind most arguments for the long-term consequences associated with ACEs is that exposure to ACEs increases the probability of developing a range of negative behavioral, physical, and psychological outcomes. Indeed, hypotheses from many criminological theories hold that childhood or adolescent experiences exert a direct effect on later-life functioning because the experience occurs during a sensitive developmental period (Agnew, 1992, 2001; Gottfredson & Hirschi, 1990). These experiences can exert direct effects on future growth which may impact the development of a range of factors that increase the risk for antisocial behavior and future environmental exposure to violent victimization. A diagram of this model—designated the *direct effect model*—is presented in Section A of Figure 1. To date, observational studies examining the impact of ACEs have used a range of multivariate statistical techniques to control for measured covariates that are associated with both ACEs and examined outcomes to provide less biased estimates of the direct effect of ACEs. The inclusion of covariates increases the likelihood that observed associations will not be the product of "backdoor paths" (D'Onofrio et al., 2016), where common causes partly or entirely explain the observed link and lead to a spurious association (Kendler, 2017). As such, measured covariates are included into multivariate statistical models to block backdoor paths and increase confidence in reported findings. However, one key limitation of this approach is that generated results are based only on controlling for confounds that are measured. Only taking into account measured confounds may inflate the possibility of detecting statistically significant associations by masking alternative pathways between unmeasured variables that may exist, but are not able to be examined because they are not measured. Therefore, it is important to explore plausible alternative explanations using methodologies capable of controlling for unmeasured confounds in order to further isolate and assess the impact of early-life exposures on behavior.

The *familial model* (depicted in Section B of Figure 1) posits that familial confounds (i.e., common genetic and shared environmental factors) that directly influence both exposure to ACEs and antisocial behavior as well as future victimization may account for much, if not all, of observed associations. The *familial model* suggests that ACEs do not exert a direct environmentally mediated effect on antisocial behavior and victimization, but rather are explained by unmeasured familial factors that correlate with both ACEs and adverse behavioral outcomes. This model is largely based on an extensive line of quantitative behavioral genetic research showing that differential exposure to early-life events, such as ACEs, are partly heritable (Pezzoli, Antfolk, Hatoum, & Santtila, 2018) and that a substantial amount of the covariance between parenting, household conditions, and child behavior are accounted by common genetic influences (Cleveland, Wiebe, Van Den Oord, & Rowe, 2000; Jaffee & Price, 2007; Neiderhiser, Reiss, Hetherington, & Plomin, 1999), creating a phenomenon known as gene–environment correlation (Scarr & McCartney, 1983). This may not come as much of a surprise given that parents create household environments and relationships with their children based on their emotional and behavioral propensities, which they also pass down to their children via biological/genetic transmission and reinforce through socialization. At present, however, no study has conducted a quasi-experimental analysis to evaluate the validity of the *direct*

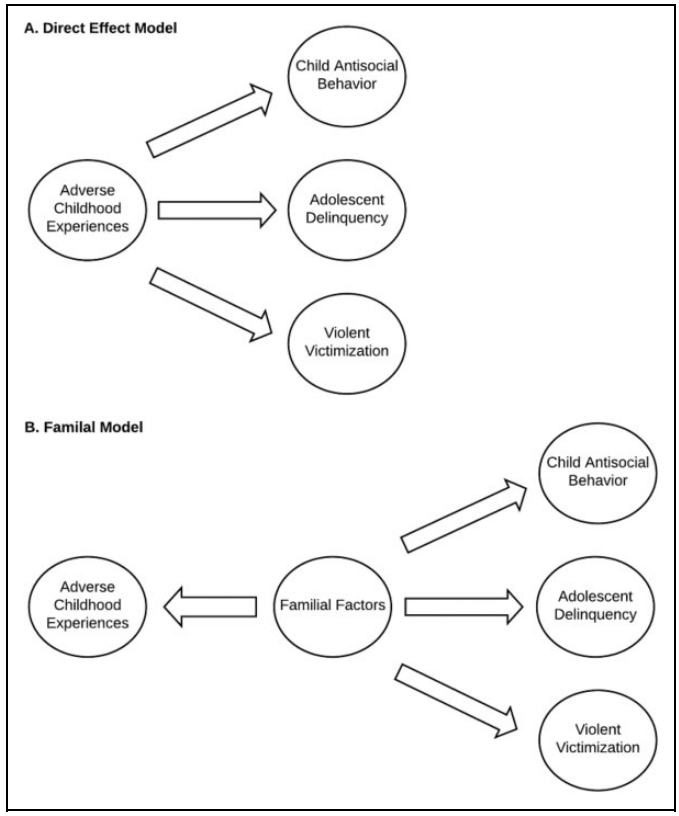

Figure 1. Conceptual models for adverse childhood experiences, antisocial behavior, delinquency, and victimization.

effect model against the *familial model* to assess which one offers a more accurate explanation of the relationship between ACEs, antisocial behavior, delinquency, and victimization.

The Current Study

The aim of the current study was to explore whether ACEs were significantly related to antisocial behavior during childhood, delinquent behavior during adolescence, and violent victimization in young adulthood after taking into account both observed and unobserved confounds. Thus, the current study has three main goals. First, the analysis focuses on examining if there is a dose–response relationship between ACEs, antisocial behavior in childhood, delinquency in adolescence, and victimization in young adulthood, as found in previous research. Second, a series of baseline and multivariate models are estimated to explore the impact of measured covariates on the direct association between ACEs, antisocial behavior, delinquency, and violent victimization. Third, fixed-effect sibling-comparison models are estimated to examine whether full siblings exposed to more ACEs are significantly more likely to demonstrate higher levels of antisocial behavior in childhood, delinquent behavior in adolescence, and risk for violent victimization in adulthood, over and above the influence of genetic and shared environmental confounds. If ACEs exert a direct effect on antisocial behavior, delinquency, and violent victimization, the association will be statistically significant across all levels of analysis.

Method

Data

The current study analyzed data from the National Longitudinal Survey of Youth (NLSY79). The NLSY79 is a nationally representative sample of 6,111 youth and an oversample of 3,652 African American and Hispanic youth between the ages of 14 and 21 as of December 31, 1978 (Baker, 1993). Participants have been assessed annually from 1979 to 1994 and biennially from 1994 to 2014. Data collection efforts have been funded by the Bureau of Labor Statistics. Retention rates have ranged from 73% to 95%.

Beginning in 1986, all children born to women from the NLSY79 sample were assessed on a range of behaviors and social experiences. Children of women from the NLSY79 have been assessed biennially from 1986 to 2014. This sample is commonly referred to as the Children of the National Longitudinal Survey of Youth (CNLSY). As of the 2012 survey wave, close to 5,000 women from the NLSY79 have given birth to over 11,500 children. Since multiple children from the same mother and/or household were included in the CNLSY, researchers have created validated kinship links to explicitly identify the sibling status shared between siblings growing up in the same family (Rodgers et al., 2016). Based on self-reported questionnaire items from both mothers and children, 16,083 sibling pairs (e.g., twins, full siblings, half siblings, cousins, and adoptive siblings) have been identified. As expected, 43% of the sibling sample are full siblings since the CNLSY consists of children born to a nationally representative sample of U.S. women, and twins were not oversampled. The full sibling sample was analyzed in the current study to examine full sibling differences in ACEs, antisocial behavior, and violent victimization between siblings who share, on average, the same amount of additive genetic material and family/household environment growing up. In total, the analytic sample included 4,844 full siblings ($\sim$70%) of the possible 6,953 full siblings for the between-family analyses and 4,009 full siblings from 993 families for the sibling-comparison analyses.

Preliminary analyses indicated that there was not a significant difference in the proportion of male and female siblings in the analytic sample compared to those excluded from the analytic sample (odds ratio [*OR*] = 1.03, 95% confidence interval [95% CI] = [0.98, 1.08]). There was also no significant difference in the proportion of African American (*OR* = .99, 95% CI [0.98, 1.02]) or Hispanic (*OR* = 1.01, 95% CI [0.99, 1.03]) siblings in the analytic sample compared to siblings excluded from the sample. Moreover, siblings in the analytic sample did not differ from excluded siblings in the number of ACEs (mean = 0.65 vs. mean = 0.59, $t = 1.63$, $p = .19$), childhood antisocial behavior (mean = 29.53 vs. mean = 28.10, $t = 1.99$, $p = .15$), adolescent delinquency (mean = 2.38 vs. mean = 2.41, $t = 1.01$, $p = .23$), or violent victimization (*OR* = 1.02, 95% CI [0.99, 1.05]).

Measures

ACEs. ACEs were measured during the 2012 and 2014 waves of the CNLSY by asking participants how often they had experienced a range of adversities before the age of 18. Participants who did not answer these questions during the 2012 assessment period were asked the same questions during the 2014 assessment period. Responses from 2012 and 2014 were combined to capture as much as data as possible on ACEs. Participants were asked if they had ever lived with anyone who was depressed, mentally ill, or suicidal (0 = *no*, 1 = *yes*) or anyone who was a problematic drinker or alcoholic (0 = *no*, 1 = *yes*). Participants were also asked to report how often, before age 18, a parent hit, beat, kicked, or physically harmed them (0 = *never*, 1 = *once*, 2 = *more than once*) and how often parental love and affection was shown towards them growing up (0 = *none at all*, 1 = *a little*, 2 = *quite a lot*, 3 = *a great deal*). Additionally, participants were asked to select one of the following

descriptions that best explained their food availability and quality situation before age 18 (1 = *we could always afford to eat good nutritious meals*, 2 = *we could always afford to eat but not always the kinds of foods we should have eaten*, 3 = *sometimes we could not afford enough to eat*, 4 = *often we could not afford enough to eat*). To stay consistent with previously used measures of ACEs (Craig et al., 2017; Fagan & Novak, 2017; Felitti et al., 1998; Wolff & Baglivio, 2016), categorical response categories were recoded into binary categories to create a count measure of ACEs experienced before age 18. As such, responses to physical abuse were recoded into categories where 0 = *never* and 1 = *once* and *more than once*, and responses to parental affection were recoded into categories where 0 = *a great deal* and *quite a lot* and 1 = *a little* and *none at all*. Responses to food availability and quality were recoded into categories where 0 = *we could always afford to eat good nutritious food* and *we could always afford to eat but not always the kinds of foods we should have eaten* and 1 = *sometimes we could not afford enough to eat* and *often we could not afford to eat*. After recoding, item responses were summed together to create a count measure of ACEs, which demonstrated adequate internal reliability (Cronbach's $\alpha = .68$). Scores ranged from 0 to 5, with higher scores representing more ACEs.

Childhood antisocial behavior. Childhood antisocial behavior was measured by a 6-item subscale of antisocial behavior from the Behavior Problems Index (Peterson & Zill, 1986). Items used to measure antisocial behavior were drawn from the well-validated Achenbach Behavior Problems Checklist (Achenbach & Edlelbrok, 1981). Mothers were asked during each wave to report how often (1 = *often true*, 2 = *sometimes true*, and 3 = *often true*) their child (1) cheats or tells lies, (2) bullies or is cruel/mean to others, (3) does not seem to feel sorry after misbehaving, (4) breaks things deliberately, (5) is disobedient at school, and (6) has trouble getting along with teachers. Mothers were asked to report on their child's antisocial behavior from ages 4 to 9. Cronbach's α for the Childhood Antisocial Behavior Scale ranged from .73 to .78 across ages. The mean of childhood antisocial behavior across ages 4–9 was calculated by taking the mean of z-transformed scores for antisocial behavior at each age interval (i.e., 4–5, 6–7, 8–9), which were also standardized within each age interval. A Blom transformation was used to reduce nonnormality in the mean of the childhood antisocial behavior measure. Childhood antisocial behavior at each age interval was highly correlated with the overall mean score ($r = .78$–$.82$).

Adolescent delinquency. Adolescent delinquency was measured by a 6-item Self-Report Scale adopted from the widely used Self-Report of Delinquency (SRD; Elliott & Huizinga, 1983). Beginning in 1988, respondents between the ages of 10 and 13 were asked to report how often (0 = *never*, 1 = *once*, 2 = *twice*, and 3 = *more than twice*) in the past year they had (1) hurt someone bad enough to need bandages or a doctor, (2) lied to a parent about something important, (3) took something from a store without paying for it, (4) intentionally damaged or destroyed property that did not belong to you, (5) had to bring your parent(s) to school because of something you did wrong, and (6) skipped a day of school without permission. Response categories to these items were dichotomized (0 = *never*, 1 = *once or more*) to create a symptom count measure of adolescent delinquency from age 10 to 13. Cronbach's α for the 6-item SRD Scale ranged from .66 to .68 from age 10 to 13. The mean of delinquent behavior between ages 10 and 13 was calculated by taking the mean of z-transformed scores for delinquency at each age interval (i.e., 10–11, 12–13), which were also standardized by age. Because the mean score of adolescent delinquency was skewed, a Blom transformation was used to reduce nonnormality.

Violent victimization. Violent victimization was measured by asking respondents during the 2008, 2010, 2012, and 2014 waves if they had been the victim of a violent crime (i.e., physical or sexual assault, robbery or arson) since the date of their last interview. Response categories were binary

(0 = *no*, 1 = *yes*). Respondents' age from 2008 to 2014 ranged from 14 to 30 years old, with an average age of 22 years old at the 2008 wave, 23 years old at the 2010 wave, 24 years old at the 2012 wave, and 26 years old at the 2014 wave.

Family-level covariates. A range of family-level covariates were controlled for in the analysis. Maternal age at first birth was measured by a continuous variable capturing the age (in years) during which each mother gave birth to their first child. Maternal education was measured by the total amount of years of education completed by mothers, while maternal intelligence was measured by percentile scores on the Armed Forces Vocational Aptitude Battery for mothers. Family income was measured by reports of the total amount of net family income reported by mothers when they were 30 years old. Income was log-transformed and then z-transformed. Maternal delinquency was measured by a 10-item Self-Report Scale capturing involvement in aggressive and delinquent behavior during adolescence. Responses were dichotomized (0 = *no*, 1 = *yes*) and summed together to create a count measure of maternal delinquent behavior (Cronbach's $\alpha = .78$). Neighborhood disadvantage was measured by an 8-item scale that asked mothers to report the extent to which each of the following items were a problem in their neighborhood (1 = *big problem*, 2 = *somewhat of a problem*, 3 = *not a problem*): (1) people don't have enough respect for rules and laws; (2) crime and violence; (3) abandoned or run-down buildings; (4) not enough police protection; (5) not enough public transportation; (6) too many parents who don't supervise their children; (7) people keep to themselves, don't care about the neighborhood; and (8) lots of people who can't find jobs. Items were reverse-coded so that higher scores indicate higher levels of neighborhood disadvantage. The maternal-reported measure of neighborhood disadvantage demonstrated good internal reliability (Cronbach's $\alpha = .85$) and was Blom transformed and converted to a z-score to facilitate interpretation. Mothers were also asked to report if the biological father of their children lived in the household. A measure was therefore created to capture whether children grew up in a single-parent household (0 = *no*, 1 = *yes*). Beginning in 2006, child respondents were asked whether their mother or father had been sent to jail or prison since the date of their last interview. A variable was created to capture parental incarceration where respondents who reported having a mother or father who had served time in jail or prison were assigned a value of "1," while respondents who reported not having a mother or father who had served time in jail or prison were assigned a value of "0." Child race was measured by a binary variable (0 = *African American/Hispanic*, 1 = *Caucasian*).

Child-specific covariates. Covariate influences unique to each child were also controlled for in the analysis. Birth order was measured by the birth order of each child within their respective family. Maternal age at birth was measured by the age of a mother (in years) at the time of giving birth to each child. Child sex was measured by a binary variable (0 = *female*, 1 = *male*).

Sample weights. Because mothers in the current study were recruited to participate in the NLSY79 in geographic clusters, many women and, in turn, their children come from the same geographic location. To adjust for geographic similarities, probability weights at the family-level were incorporated into all models to generate more representative estimates of children born to a nationally representative sample of women in the United States.

Plan of Analysis

The analyses were carried out in a series of interconnected steps. First, mean estimates of childhood antisocial behavior, adolescent delinquency, and percentages of violent victimization were calculated across prevalence rates of ACEs to explore whether there was a dose–response relationship between ACEs, antisocial behavior, and victimization. Second, a series of hierarchical linear models

(HLMs) were used to examine the associations between ACEs, antisocial behavior, delinquency, and violent victimization. Traditional HLMs were used to assess the effect of ACEs on antisocial behavior and delinquency, while a modified logistic HLM was used to explore the effect of ACEs on the odds of violent victimization. Overall, four HLMs were estimated to evaluate the association between ACEs, antisocial behavior, delinquency, and violent victimization. Model 1 calculated the unadjusted direct association between ACEs and antisocial behavior, delinquency, and violent victimization. Model 2 examined the direct association after accounting for measured family-level covariates that vary between families. Model 3 examined the direct association after controlling for measured family-level covariates and measured child-specific covariates unique to each child. Model 4 then examined the direct association between ACEs and antisocial behavior, delinquency, and victimization after taking into account measured child-specific covariates, unmeasured shared environmental confounds, and unmeasured genetic confounds. To control for genetic and shared environmental confounds between siblings (i.e., familial confounds), Model 4 compared full siblings differentially exposed to ACEs. The fixed-effect sibling-comparison model provides a more stringent test of the direct effect of ACEs on antisocial behavior, delinquency, and violent victimization because unmeasured genetic and shared environmental confounds that influence exposure to ACEs, the development of antisocial behavior, and violent victimization are taken into account (D'Onofrio et al., 2016). To control for familial confounds, full sibling–specific ACE scores were compared to average ACE scores for children from the same household. The deviation of sibling-specific ACE scores from the family-level aggregate score was then used to examine whether sibling-specific deviations were associated with differences in antisocial behavior, delinquency, and risk for violent victimization.

Before analyses were performed, the prevalence of missing data was examined. Missing values ranged from 6.54% (childhood antisocial behavior) to 18.03% (violent crime victimization) and were addressed by multiple imputation via chained equation procedures that included measured covariates to generate 20 imputed data sets to account for missing values (Graham, Olchowski, & Gilreath, 2007). The analyses analyzed all available data instead of only data from respondents with complete data on all covariates. This approach has been shown to generate standard errors that account for uncertainty due to missing values and avoids bias that would arise from only examining respondents with complete data (Rubin, 2004).

Results

Table 1 presents the descriptive statistics for ACEs, childhood antisocial behavior, adolescent delinquency, violent victimization, family-level covariates, and child-specific covariates. As presented, 61.74% of participants reported no ACEs, while 21.37% reported one ACE, 9.54% reported two ACEs, 4.74% reported three ACEs, 2.02% reported four ACEs, and less than 1.00% reported five ACEs. Participants reported, on average, two delinquent behaviors (mean = 2.38, standard deviation [*SD*] = 2.13) during adolescence, while 13.65% of participants reported a violent victimization. Descriptive analysis also revealed that 50.90% of the sample was male, 56.00% of the sample was Caucasian, 27.00% of the sample was African American, and 17.00% of the sample was Hispanic. Over 7.00% of the sample reported having a mother or father who had served time in jail or prison and 10.64% of the sample reported growing up in a single-parent household.

Table 2 provides the average score of antisocial behavior in childhood, the average number of self-reported delinquent behavior in adolescence, and the prevalence of self-reported violent victimization in young adulthood by the number of ACEs. As shown, there was evidence of a dose–response relationship between reported ACEs, antisocial behavior, and victimization. Participants who reported no ACEs had the lowest levels of maternal-reported antisocial behavior in childhood, adolescent delinquent behavior in adolescence, and violent victimization in adulthood compared to

Table 1. Descriptive Statistics.

Variables	*N*	%	Mean (*SD*)	Range
Adverse childhood experiences (ACEs)	4,884	—	0.65 (1.02)	0–5
Zero ACEs	2,852	61.74	—	0–1
One ACE	1,206	21.37	—	0–1
Two ACEs	466	9.54	—	0–1
Three ACEs	232	4.74	—	0–1
Four ACEs	99	2.02	—	0–1
Five ACEs	29	0.59	—	0–1
Family-level covariates				
Age at first birth	2,231	—	22.92 (5.32)	16–34
Education	2,231	—	13.39 (2.64)	2–20
Family income	2,231	—	US$33,593 (US$39,202)	$0–$97,480
Maternal delinquency	2,231	—	1.01 (1.43)	0–10
Maternal intelligence	2,231	—	35.65 (27.31)	1–99
Neighborhood disadvantage	2,231	—	11.93	0–24
Parental incarceration	2,231	7.30	—	0–1
Single-parent household	2,231	10.64	—	0–1
Race	4,884	—	—	0–1
Caucasian	2,735	56.00	—	1–1
African American	1,319	27.00	—	0–0
Hispanic	830	17.00	—	0–0
Child-specific covariates				
Birth order	4,884	—	1.90 (1.10)	1–5
Maternal age at birth	4,884	—	24.31 (5.63)	16–39
Sex	4,884	—	—	0–1
Male	2,486	50.90	—	1–1
Female	2,398	49.10	—	0–0
Childhood antisocial behavior (age 4–9)	4,884	—	29.53 (18.40)	18–49
Adolescent delinquency (age 10–13)	4,884	—	2.38 (2.13)	0–12
Violent victimization (age 14–30)	4,884	13.68	—	0–1

respondents with one, two, three, four, or five ACEs. Participants with no ACEs reported, on average, two delinquent acts during adolescence (mean = 2.22), while respondents with five ACEs reported, on average, three delinquent acts (mean = 3.35). Results also showed that while 13.01% of participants with no ACEs reported experiencing a violent victimization, the percentage of victimization increased with the number of ACEs. Prevalence rates indicated that 21.86% of participants with one ACE reported experiencing a violent victimization in young adulthood, while 29.79% of participants with two ACEs reported experiencing a violent victimization, 38.72% of participants with three ACEs, 41.59% of participants with four ACEs, and 66.67% of participants with five ACEs. Taken together, there was clear support for a dose–response relationship between ACEs, antisocial behavior in childhood, delinquent behavior in adolescence, and risk for violent victimization in young adulthood.

The next step in the analysis focused on examining whether and to what extent ACEs were directly associated with childhood antisocial behavior after measured and unmeasured confounds were taken into account. The intraclass correlation (ICCs), which measures the degree to which observations are similar to one another within clusters, showed that sibling correlations for ACEs were expectedly high (ICC = .81, 95% CI [0.75, 0.86]) but that there was significant within-family variation (16.05%, $p < .01$) and between-family variation (83.95%, $p < .001$). Based on these results, HLMs were estimated to examine the between- and within-family effects of ACEs on antisocial

Table 2. Average of Antisocial Behavior and Prevalence of Violent Victimization by Adverse Childhood Experiences (ACEs).

ACEs	Childhood Antisocial Behavior (Mean)	Adolescent Delinquency (Mean)	Violent Victimization (%)
Zero ACEs	19.38	2.22	13.01
One ACE	26.05	2.33	21.86
Two ACEs	31.59	2.76	28.79
Three ACEs	33.61	2.92	38.72
Four ACEs	36.00	3.25	41.59
Five ACEs	40.14	3.35	66.67

Table 3. Parameter Estimates From Multilevel Models for Childhood Antisocial Behavior.

	Model 1		Model 2		Model 3		Model 4	
Variables	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Adverse childhood experiences	.30***	.02	.26***	.02	.23**	.02	.04	.03
Age at first birth	—	—	−.02**	.01	−.02**	.01	—	—
Maternal education	—	—	−.02**	.01	−.02**	.01	—	—
Maternal delinquency	—	—	.06***	.01	.05**	.01	—	—
Maternal intelligence	—	—	.01	.02	.01	.01	—	—
Neighborhood disadvantage	—	—	.14***	.02	.10***	.02	—	—
Parental incarceration	—	—	.08*	.02	.07*	.02	—	—
Single-parent household	—	—	.05*	.01	.02	.01	—	—
Race	—	—	−.19***	.02	−.14**	.02	—	—
Family income	—	—	−.06**	.02	−.05**	.02	—	—
Birth order	—	—	—	—	.02	.01	.01	.01
Maternal age at birth	—	—	—	—	.01	.01	.01	.01
Sex	—	—	—	—	.21***	.02	.20***	.02
N	4,844		4,844		4,844		4,009	

Note. All parameters are unstandardized.
*$p < .05$. **$p < .01$. ***$p < .001$.

behavior, delinquency, and violent victimization. Table 3 presents the unstandardized parameter estimates from all estimated HLMs for childhood antisocial behavior. Model 1 shows that for each ACE, participants had an increase of 0.16 *SD* ($p < .001$) in antisocial behavior from age 4 to 9. Model 2 explored the association between ACEs and childhood antisocial behavior while controlling for measured family-level covariates. Including the family-level covariates only slightly reduced the main effect of ACEs on childhood antisocial behavior ($b = .14$, $p < .001$). Model 3 also indicated that ACEs continued to have a significant, albeit attenuated, direct effect on childhood antisocial behavior after controlling for measured family-level and child-specific covariates ($b = .11$, $p < .001$). Model 4 shows estimates from the sibling-comparison model controlling for measured child-specific covariates and unmeasured genetic and shared environmental confounds. The estimates from Model 4 indicate that after familial confounds were taken into account, including characteristics unique to each child, there was no significant direct effect of ACEs on childhood antisocial behavior ($b = .04$, $p = .19$).

Table 4 presents estimates from a similar series of HLMs examining the effect of ACEs on adolescent delinquency from age 10 to 13. As can be seen, Model 1 indicated that for each ACE,

Table 4. Parameter Estimates From Multilevel Models for Adolescent Delinquency.

	Model 1		Model 2		Model 3		Model 4	
Variables	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Adverse childhood experiences	.24***	.02	.22***	.02	.19***	.01	.02	.01
Age at first birth	—	—	−.01**	.01	−.01*	.01	—	—
Maternal education	—	—	−.02**	.01	−.01	.01	—	—
Maternal delinquency	—	—	.05**	.01	.04**	.01	—	—
Maternal intelligence	—	—	.01	.02	.01	.02	—	—
Neighborhood disadvantage	—	—	.12***	.02	.11**	.02	—	—
Parental incarceration	—	—	.15***	.01	.10**	.01	—	—
Single-parent household	—	—	.03	.01	.01	.01	—	—
Race	—	—	−.17**	.03	−.12*	.04	—	—
Family income	—	—	−.05**	.01	−.03*	.01	—	—
Birth order	—	—	—	—	.03	.01	.01	.01
Maternal age at birth	—	—	—	—	.01	.01	.01	.01
Sex	—	—	—	—	.32***	.02	.31***	.02
N	4,844		4,844		4,844		4,009	

Note. All parameters are unstandardized.
*$p < .05$. **$p < .01$. ***$p < .001$.

participants had an increase of 0.14 *SD* in delinquent behavior ($b = .14$, $p < .001$) when not controlling for any covariates or unmeasured confounds. Models 2 and 3 showed that ACEs continued to exert a significant, although weaker, direct effect on adolescent delinquent behavior after controlling for family-level covariates ($b = .13$, $p < .001$) and family-level and child-specific covariates ($b = .10$, $p < .001$). However, Model 4 revealed that ACEs no longer exerted a significant direct effect on adolescent delinquency after controlling for genetic and shared environmental confounds ($b = .02$, $p = .30$), suggesting that siblings with more ACEs were no more likely to report higher levels of adolescent delinquent behavior compared to co-siblings with less ACEs.

Table 5 provides unstandardized parameter estimates from a series of logistic HLMs examining the association between ACEs and the odds of violent victimization in young adulthood. Model 1 showed that for each ACE, participants were 58% more likely to experience a violent victimization in young adulthood ($OR = 1.58$, 95% CI [1.50, 1.68]). After controlling for family-level covariates in Model 2, the association between ACEs and violent victimization was slightly attenuated where for each ACE, participants were 54% more likely to experience a violent victimization ($OR = 1.54$, 95% CI [1.42, 1.65]). Model 3 still revealed a positive and significant association between ACEs and violent victimization after controlling for family-level and child-specific covariates ($OR = 1.49$, 95% CI [1.37, 1.71]). Model 4, however, revealed that ACEs were no longer significantly associated with increased probability of experiencing a violent victimization in young adulthood after controlling for common genetic and shared environmental confounds ($OR = 1.01$, 95% CI [0.92, 1.13]).

Sensitivity Analyses

Similar to all modeling strategies, there are assumptions about the generalizability of findings from comparing differentially exposed full siblings (D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013). First, this approach assumes that results from full siblings will generalize to families with half siblings. To assess whether the reported results were robust to this assumption, half siblings from the CNLSY were included in the sample and similar analyses were conducted. The results from this set of sensitivity analyses did not reveal substantively different results from the reported results.

Table 5. Parameter Estimates From Multilevel Models for Violent Victimization.

	Model 1		Model 2		Model 3		Model 4	
Variables	*OR*	95% CI	*OR*	95% CI	*OR*	95% CI	*OR*	95% CI
Adverse childhood experiences	1.58***	[1.50, 1.68]	1.54***	[1.42, 1.65]	1.49***	[1.37, 1.71]	1.01	[0.92, 1.13]
Age at first birth	—	—	0.97**	[0.93, 0.99]	0.97**	[0.94, 0.99]	—	—
Maternal education	—	—	0.98*	[0.94, 0.99]	0.99	[0.97, 1.01]	—	—
Maternal delinquency	—	—	1.08*	[1.02, 1.11]	1.07*	[1.02, 1.10]	—	—
Maternal intelligence	—	—	0.99	[0.98, 1.01]	0.99	[0.98, 1.01]	—	—
Neighborhood disadvantage	—	—	1.17**	[1.09, 1.28]	1.14*	[1.05, 1.21]	—	—
Parental incarceration	—	—	1.09*	[1.01, 1.17]	1.02	[0.99, 1.03]	—	—
Single-parent household	—	—	1.00	[0.99, 1.01]	1.00	[0.94, 1.01]	—	—
Race	—	—	0.92**	[0.87, 0.97]	0.97*	[0.95, 0.99]	—	—
Family income	—	—	1.00	[0.99, 1.00]	1.00	[0.99, 1.00]	—	—
Birth order	—	—	—	—	1.00	[0.99, 1.00]	1.00	[0.99, 1.00]
Maternal age at birth	—	—	—	—	1.00	[0.99, 1.00]	1.00	[0.99, 1.00]
Sex	—	—	—	—	1.03	[0.99, 1.05]	1.01	[0.99, 1.03]
N	4,844		4,844		4,844		4,009	

Note. All parameters are unstandardized. *OR* = odds ration; CI = confidence interval.
*$p < .05$. **$p < .01$. ***$p < .001$.

Second, sibling-comparison analyses assume no carryover effects between siblings (i.e., the possibility that the amount of exposure for one sibling influences the outcome for another sibling; Frisell, Öberg, Kuja-Halkola, & Sjölander, 2012; Lahey & D'Onofrio, 2010; Susser, Eide, & Begg, 2010). To examine this possibility, an interaction term was created between birth order and ACEs and entered into each HLM equation examining childhood antisocial behavior, adolescent delinquent behavior, and young adult violent victimization. The results revealed that birth order did not moderate the association between ACEs and any of the examined outcomes. Results from these models are available upon request.

Discussion

In an effort to further identify early-life exposures that may exert a direct effect on the development of antisocial behavior and other adverse life outcomes, criminologists have begun to examine the unique influence of ACEs. This burgeoning line of research has found that ACEs are positively associated with criminal offending, different forms of victimization, and a wide range of adverse health outcomes (Chapman et al., 2004; Desai et al., 2002; Dube, Felitti, Dong, Chapman, et al., 2003; Edwards, Holden, Felitti, & Anda, 2003; Felitti et al., 1998; Fox et al., 2015; Widom, 1995). Yet, research has been largely limited by examining only at-risk samples of youth and not controlling for backdoor paths that may inflate the possibility of observing statistically significant direct associations between ACEs and deleterious outcomes (D'Onofrio et al., 2016). The current study aimed to address this limitation by analyzing a large sample of full sibling pairs from a population-based sample of U.S. youth to evaluate the potential direct effect of ACEs on childhood antisocial behavior, adolescent delinquency, and young adult violent victimization. Three key findings emerged from the analysis that warrant further discussion.

First, as expected, the average prevalence rate of ACEs in the current sample (mean = 0.65) was much smaller than the average rate observed in other at-risk samples such as the LONGSCAN

sample (mean = 3.15; Fagan & Novak, 2017) that consists of children from high-risk families. However, the average ACE score in the CNLSY was closer to the average ACE score found in the Cambridge Study in Delinquent Development (mean = 1.87; Craig et al., 2017), which consists of a less at-risk sample of 411 males from a middle-income working-class neighborhood in South London. While the analyses were based on a sample of children born to a nationally representative sample of U.S. females, thus increasing the possibility that they may not come from the same type of backgrounds, some possible reasons for observed differences in the number of reported ACEs could be due to differences in the number and types of ACEs examined across studies. While previous studies have included measures of parental separation or divorce and family member incarceration in ACE scores, this was not possible in the current study since such measures do not vary between siblings from the same family and would make it difficult to examine differences between siblings. However, self-report items measuring parental incarceration and single-parent household were controlled for in all between-family HLMs. Nevertheless, measures of parental incarceration and single-parent household were added to ACE scores to see if including such items would substantially alter the reported average prevalence rate of ACEs in the CNLSY. After including these items, the average prevalence rate increased from .65 to .81. Moreover, additional models were estimated that included both measures in the ACEs score to examine whether this produced any substantial changes. The pattern of results remained the same. Future research should focus on creating standardized measures of ACEs to aid in the replication of findings across studies.

Second, bivariate analyses revealed a dose–response relationship between ACEs, childhood antisocial behavior, adolescent delinquent behavior, and risk for violent victimization. This finding is in line with previous evidence from other studies that have found that risk for poor health, psychopathology, and victimization incrementally increases as the number of ACEs increases (Dong et al., 2003; Dong et al., 2004; Dube et al., 2002; Dube, Felitti, Dong, Chapman, et al., 2003; Dube, Felitti, Dong, Giles, et al., 2003; Felitti et al., 1998; Port et al., 2016). In all between-family HLMs statistically controlling for measured covariates, ACEs were found to exert a positive statistically significant effect on antisocial behavior, delinquency, and violent crime victimization. These results provide additional support for the pervasive association between ACEs and antisocial behavior during childhood and adolescence as well as risk for violent victimization later in life. While these findings contribute to an ever-growing body of observational research on the consequences of ACEs (Baglivio et al., 2015; Craig et al., 2017; Fagan & Novak, 2017; Fox et al., 2015; Hambrick et al., 2017; Hunt et al., 2017; Ports et al., 2016; Wolff & Baglivio, 2016; Wolff et al., 2017), they should be interpreted with caution since observed associations may be attributable to unmeasured common genetic and shared environmental factors that are account for the correlation between ACEs, antisocial behavior, and violent victimization.

Third, to address the issue of plausible backdoor paths between ACEs and examined outcomes where associations may be overestimated due to familial confounding, sibling-comparison models were estimated to control for unobserved genetic and shared environmental factors. A major benefit of using a co-sibling design to test familial model is that these approaches are able to closely resemble randomized controlled trials since they are able account for the influence of both observed and unobserved confounders. After controlling for genetic and shared environmental confounders (i.e., single-parent household and parental incarceration) in the sibling-comparison models, as well as observed child-specific characteristics (i.e., birth order, maternal age at birth, and sex) to better isolate the environmental influence of ACEs, all direct associations were no longer statistically significant. Accordingly, generated findings from the current study indicate that while traditional between-family analyses report that ACEs are linked to antisocial behavior and victimization, the direct effect of ACEs on these outcomes may be overestimated due to familial factors (Beckley et al., 2018; Connolly & Kavish, 2018). Some readers may reasonably question how this could be the case, given that a wealth of research has reported statistically significant associations between child

maltreatment, antisocial behavior, and future victimization. In fact, however, the assumption—commonly referred to as the "nurture assumption" (Harris, 2011)—that social experiences are responsible for largely molding a person into whom they will become has been empirically scrutinized (see also, Pinker, 2003). Contemporary research using quasi-experimental, family-based research designs are beginning to show that after controlling for factors that influence the nonrandom selection of children into families, child abuse/neglect (Stern et al., 2018), early sexual abuse (Dinwiddie et al., 2000), child maltreatment (Dinkler et al., 2017), parenting (Wright & Beaver, 2005; Wright, Beaver, DeLisi, & Vaughn, 2008), adolescent victimization (Schaefer et al., 2017), and lone motherhood (Dinescu, Haney-Claus, Turkheimer, & Emery, 2018) have marginal, and sometimes nonsignificant, effects on antisocial behavior and psychopathology. Consequently, the reported results from the current study alongside findings from other studies highlight the need for future research to use family-based research designs to isolate the impact of these environments on behavior and increase confidence in the conclusions on the direct link between early-life experiences, such as ACEs, and the development of problem behaviors.

It is important to note at this juncture that evidence from the current study does not indicate that parenting or early-life victimization has no effect at all on later-life functioning. The interplay between genetic liability and environmental experiences across the life course (i.e., gene-environment interplay) is pervasive and the rule rather than the exception (Knafo & Jaffee, 2013; Scarr & McCartney, 1983). It is possible that perspectives such as Scarr's (1992) *average and expectable environments hypothesis* where experienced environments that fall outside the realm of "average expectable" conditions may impact genetic expression and exert cascading effects on later-life human development. However, while this is a possibility, it is important to keep in mind that biological set points between families or members of the same family may condition the impact of exposure to environments that deviate from "average expectable" conditions where some individuals may be more resilient to ACEs, while others are more vulnerable (Rowe, 2001). Unfortunately, the sad reality is that children with increased genetic liability for developing antisocial behavior and psychopathology are often exposed to more ACEs as a result of their contact with abusive parents and/or family members and unhealthy life circumstances (i.e., food insecurity, parental incarceration, parent substance use, or parent mental illness). These factors create a nonrandom process of selection into at-risk family environments that must be taken into account to strengthen causal inference between ACEs and human behavior.

The current study has several strengths. Most importantly, the study used a quasi-experimental, family-based research design to control for genetic and shared environmental liability shared between siblings for ACEs, antisocial behavior, delinquency, and violent victimization. The reported results also accounted for measured family-level and child-specific covariates to examine the influence of these confounds on the association and to better isolate the direct effect of ACEs on antisocial behavior, delinquency, and violent victimization. In addition, the reported results are also more generalizable than those of previous studies since the results are based on a sample of children born to a national sample of women, which was weighted to be representative of all women in the United States.

Despite these strengths, there are several limitations that must also be acknowledged. First, retrospective reports of ACEs were used to measure ACEs in the current study. This may have increased risk of recall problems, thus biasing reporting. Contemporary research has assessed whether using retrospective or prospective reports of ACEs influence observed associations and show that retrospective ACE measures are more strongly associated with self-reported life outcomes, but agreeable and neurotic proclivities may bias retrospective ACE measures toward overestimating the impact of ACEs on self-reported outcomes (Reuben et al., 2016). Supplemental analyses revealed that ACE items that varied most between full siblings were items about living

with anyone who was a problematic drinker or alcoholic, having been shown little to no parental love and affection, and having been physically abused before age 18. These items varied significantly more than items asking about exposure to food insecurity ($ps < .01$), but not other ACEs ($ps > .05$). Based on prior research suggesting that many adults may fail to report or recall their childhood experiences accurately (Widom & Courtious, 1997), it is unclear whether these differences are actual differences or differences in reporting accuracy. Future research should focus on using both self-report and official records to better capture child abuse experiences to evaluate whether different forms of reporting alter observed associations between ACEs, antisocial behavior, and victimization. Moreover, it is important to note that because participants were asked to report ACEs before age 18, it was not possible to establish temporal order between ACEs, child antisocial behavior measured at ages 4–9, and adolescent delinquency measured at ages 10–13. In light of this limitation, findings from these portions of the current study should be interpreted with caution. Second, because the CNLSY is a sample of children born to a nationally representative sample of U.S. women, there were very few twins assessed in the sample since twins constitute a small fraction of the overall youth population. While examining differences between full siblings helps to control for shared genetic and environmental influences, it does not entirely control for all differences between full siblings that may influence the association under examination (Frisell et al., 2012; McGue, Osler, & Christensen, 2010). Several strategies were used to reduce this possibility such as including child-specific covariates into sibling-comparison models and assessing the degree of carryover effects. Still, future research should attempt to replicate the reported results with large twin samples to better control for genetic and shared environmental confounding. In light of this, it is worth noting that research has reported a similar degree of genetic and shared environmental influence on criminal and delinquent behavior in both twin and sibling samples (Connolly & Beaver, 2014; Kendler, Lönn, Maes, Sundquist, & Sundquist, 2015). Third, sexual abuse and family conflict, which are included in recent conceptualizations of ACEs, were not assessed in the CNLSY before age 18 and therefore not included in the analysis. Future research should aim to replicate the results with measures of sexual abuse and family conflict using sibling comparisons to assess whether these items alter any conclusions from the current study.

Conclusion

Recently, there has been a proliferation of research aimed at evaluating the effect of ACEs on future antisocial behavior and victimization. The current study, which was based on a population-based sample of youth, a rigorous quasi-experimental design with longitudinal data, and a sophisticated analytic approach to control for possible unmeasured confounds, did not find evidence which would strengthen the inference that ACEs have a direct effect on antisocial behavior, delinquency, or violent crime victimization. Findings that unmeasured familial factors related to ACEs may be responsible for higher levels of antisocial behavior and later-life violent victimization emphasize the need for criminologists, and social scientists alike, to use family-based designs to help identify plausible causal environmental risk factors associated with ACEs to provide more effective targets for environmental intervention/prevention. Although criminological research on early-life exposure and later-life offending and victimization has advanced in many fruitful ways in recent years, the amount of research using twin or sibling designs to control for documented genetic and shared environmental confounding has been stagnant. This has made it increasingly difficult to determine whether examined exposures have an effect on outcomes net of familial confounding. One must be hopeful, however, that such designs will be used more frequently in the coming years to aid in moving from examining correlates to causal mechanisms.

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