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EMPIRICAL RESEARCH

The Causal Relationship between Childhood Adversity and Developmental Trajectories of Delinquency: A Consideration of Genetic and Environmental Confounds

Eric J. Connolly[1] · Nicholas Kavish[2]

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Abstract
An extensive line of research has found that children exposed to multiple forms of early life adversity are more likely to engage in high levels of delinquent behavior during adolescence. Several studies examining this association have used a range of multivariate statistical techniques capable of controlling for observable covariates. Fewer studies have used family-based research designs to additionally control for unobservable confounds, such as genetic and shared environmental influences, that may be associated with exposure to childhood adversity and delinquency. The current study analyzes self-report data on 2534 full-siblings (50% female) from the National Longitudinal Survey of Youth 1997 to conduct a sibling-comparison analysis to provide a rigorous test of the causal hypothesis that exposure to childhood adversity causes differences in developmental patterns of delinquent behavior. Results from multivariate latent growth curve models revealed that childhood adversity was associated with higher starting levels of delinquency during adolescence and slower rates of decline from adolescence to emerging adulthood. Results from multivariate sibling-comparison models, however, revealed that siblings exposed to higher levels of childhood adversity reported higher starting levels of delinquent behavior, but not slower declines over time, suggesting that childhood adversity may not be directly associated with long-term patterns of delinquent behavior after genetic and shared environmental factors are taken into account. Implications of these results for future research are discussed.

Keywords Childhood adversity · Delinquency · Siblings · Genetic · Environmental · NLSY97

Introduction

It is estimated that over 1 billion children—half of all children in the world—are exposed to some form of violence every year (Slutkin 2017). In the United States, estimates suggest that approximately 60% of children under the age of 17 are exposed to primary or secondary violence each year (Finkelhor et al. 2009). Children exposed to violence and other adverse experiences early in life are at an elevated risk for developing a host of deleterious life outcomes including cognitive problems (Graham-Bermann et al. 2010), poor school performance (Hardaway et al. 2014; Sherr et al. 2015), substance abuse issues (Zimmerman and Kushner 2017), internalizing problems (Hardaway et al. 2014; Heleniak et al. 2018), externalizing problems (Darawshy and Haj-Yahia 2018; Hardaway et al. 2014), and long-terms patterns of serious delinquent and criminal behavior (Zimmerman and Posick 2016). In recent years, a vast amount of research has documented that children exposed to multiple forms of adversity are more likely to report higher levels of delinquent and externalizing behaviors during adolescence compared to children exposed to less adversity (Kretschmar et al. 2017; Turner et al. 2016). As a result, many scholars contend that adversity experienced during childhood may exert a long-term influence on developmental patterns of offending across adolescent development (Farrell and Zimmerman 2017; Obsuth et al. 2017), thus making childhood adversity an important, potentially causal, risk factor for adolescent delinquency.

✉ Eric J. Connolly
ejc025@shsu.edu

[1] Department of Criminal Justice and Criminology, Sam Houston State University, Huntsville, TX 77340, USA

[2] Department of Psychology and Philosophy, Sam Houston State University, Huntsville, TX 77340, USA

While our understanding of the association between childhood adversity and adolescent delinquency has expanded in recent years, there are two existing gaps in the literature. First, it is difficult to determine whether observed associations between childhood adversity and delinquent behavior reflect direct effects or arise because children who are more likely to report multiple forms of adversity are also more likely to engage in higher levels of delinquency because of a third underlying common variable. This "third variable problem" is important because it limits our understanding of whether childhood adversity exerts a direct effect on subsequent delinquent behavior and limits the ability of researchers and policymakers to determine whether interventions that target a specific type of adversity will reduce the occurrence of adolescent delinquency. The problem is compounded by the fact that previous behavior genetic research reports that exposure to different levels of childhood victimization and adversity is correlated with genetic differences, whereby some children are more likely to experience multiple forms of adversity, and are more affected by such experiences, because of genetic vulnerabilities (Ball et al. 2008; Connolly and Beaver 2016; Fisher et al. 2015; Kavish et al. 2018; Schaefer et al. 2018). As such, reported associations may be influenced by "familial" confounds, which are confounding variables that are substantially correlated in siblings who grow up together. A second and related gap is that few studies have used longitudinal, genetically informed research designs to evaluate the short- and long-term effects of childhood adversity on initial levels and rates of change in delinquent behavior over time, while controlling for familial confounds and other theoretical covariates. One research design that can be used to address this gap in the literature is the sibling-comparison design (Lahey and D'Onofrio 2010). This design contrasts siblings exposed to higher levels of childhood adversity to their less exposed siblings, a design that controls for genetic and shared environmental risks associated with childhood adversity. Results from this type of design can help evaluate the quasi-causal effect of early life experiences on later life behavior. With this in mind, the current study aimed to explore whether and to what extent childhood adversity was related to starting levels in delinquent behavior during adolescence and rates of change from adolescence to emerging adulthood after taking into account genetic and shared environmental confounds.

Childhood Adversity and Adolescent Delinquency

Traditionally, developmental research on risk factors for offending have tended to examine factors individually, such as focusing on single types of victimization (e.g., bullying or child sexual abuse; Obsuth et al. 2017). More recently, researchers have begun to develop more inclusive profiles of participants by assessing their experiences with multiple forms of victimization (Cudmore et al. 2017; Finkelhor et al. 2007a; Kretschmar et al. 2017; Turner et al. 2016) and other adverse experiences (e.g., witnessing domestic violence, having an incarcerated parent; Felitti et al. 1998). The inclusion of these multiple types of adverse childhood experiences (ACEs) in empirical research has allowed for a more comprehensive understanding of the associations between childhood adversity and different types of psychopathology across the life course including mental health problems (Ford et al. 2018; Obsuth et al. 2017; Turner et al. 2016), anger and aggression (Ford et al. 2018; Obsuth et al. 2017), substance use (Ford et al. 2018), and delinquency (Cudmore et al. 2017; Farrell and Zimmerman 2017; Turner et al. 2016).

Within this growing body of research, studies examining the association between early life victimization and adolescent delinquent behavior have provided strong evidence for the victim–offender overlap. For example, in a nationally representative sample of over 2000 youth ages 10–17, Turner and colleagues (2016) found that children exposed to multiple forms of victimization had higher probabilities of engaging in many types of delinquent behavior, compared to non-victims and several other classes of youth victimized in only one or two domains (e.g., at home or school). Similarly, in a study of over 1600 youth from the Zurich Project on the Social Development of Children and Youths sample, Obsuth et al. (2017) found that non-parental poly-victimization (but not parental poly-victimization) was associated with increases in self-reported delinquency. Similarly, in another study by Cudmore et al. (2017) examining a sample of 1525 Latino youth, higher levels of victimization were significantly and positively associated with delinquency, even after controlling for age, gender, and socioeconomic status. In their study, Cudmore and colleagues (2017) found that multiple victimizations, termed 'poly-victimization', increased the odds of engaging in delinquent behavior by approximately 53%, and that the association was only marginally attenuated after controlling for anger and social support. Kretschmar and colleagues (2017) also investigated patterns of poly-victimization and delinquency in a sample of almost 500 at-risk youth and found that youth who reported both repeated exposure to violence and exposure to more types of violence had higher probabilities of multiple types of violence, including making threats, hitting someone before and after that person hit them, beating someone up, and attacking someone with a knife. Finally, using a nationally representative sample of over 12,000 adolescents and young adults, Farrell and Zimmerman (2017) found that poly-victimization was associated with a higher probability of engaging in both property crime and violent offending. They reported that the highest levels of poly-victimization (2 or more incidents, 2

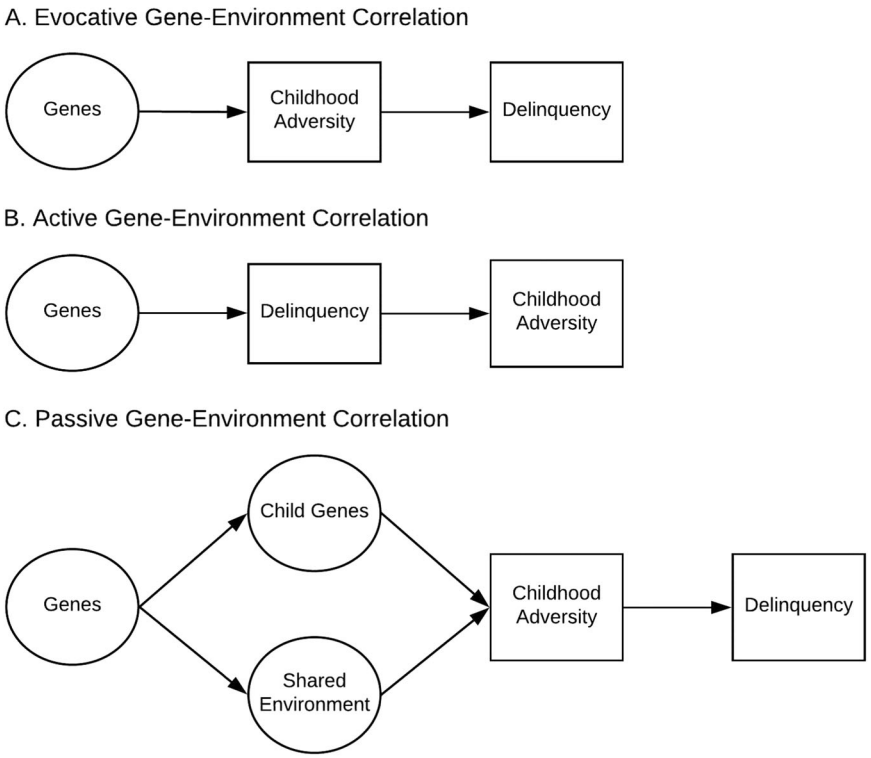

Fig. 1 Models of childhood adversity and delinquency due to gene–environment correlation

or more types) were associated with the highest probabilities of both property crime and violent offending, even after controlling for age, gender, race, family structure, depression, neighborhood bond, and impulsivity. Furthermore, poly-victimization was significantly more strongly associated with violent offending compared to repeated exposure to violence of one type (Farrell and Zimmerman 2017).

Specific victimizations and poly-victimization are not the only adverse life experiences a child or adolescent can experience, however. Recently, studies have begun to examine the relationship between scores on broader measures of adverse childhood experiences (ACEs) and offending. Baglivio et al. (2014) found that serious, violent, and chronic (SVC) juvenile offenders were more likely to have experienced an ACE and experience more ACEs than non-SVC juveniles referred to a juvenile justice facility. Fox and colleagues (2015) also reported that each additional ACE increased the risk of becoming an SVC offender by age 35, even after controlling for other risk factors. Related research has continued to provide supporting evidence suggesting that high levels of ACEs are associated with early onset offending and recidivism (e.g., Baglivio et al. 2015; Wolff et al. 2017).

Taken together, contemporary research suggests that experiencing multiple ACEs are strongly associated with delinquent behavior. This association is consistently found and remains even after controlling for a wide range of statistical covariates. Of particular note, the association appears to be robust to controls for both individual-level factors (e.g., trait anger, impulsivity) and demographic confounds such as race/ethnicity and socioeconomic status (Cudmore et al. 2017; Farrell and Zimmerman 2017; Fox et al. 2015), and the association has been found in at least one large study outside of the United States (Osbuth et al. 2017). Moreover, research suggests that, although it is not fully clear why, experiencing multiple types of victimization, or ACEs, is particularly impactful. While repeated exposure to violence (chronic victimization) is associated with delinquent behavior, recent studies suggest that exposure to multiple types of violence (poly-victimization) has an even stronger relationship with engaging in delinquency, including both property and violent offenses (Farrell and Zimmerman 2017; Turner et al. 2016). Taken together, adolescents who experience adversity in multiple settings (e.g., at home *and* at school) and/or in multiple ways (e.g., being threatened with *and* experiencing violence, or being bullied *and* having a parent incarcerated) are at an increased risk of serious delinquent behavior.

Theoretical Models for Childhood Adversity and Adolescent Delinquency

The impressive amount of evidence demonstrating that ACEs are related to higher levels of delinquent behavior from various different samples using different measurement techniques has started to suggest that perhaps ACEs are a causally linked to later life delinquent behavior. This

theoretical model, which is often tested in contemporary research examining ACEs and delinquency, contends that childhood adversity is directly associated with the development of delinquent behavior in adolescence. Indeed, several studies provide evidence for this theoretical model (Baglivio et al. 2015; Cudmore et al. 2017; Farrell and Zimmerman 2017).

Yet, there is one critical challenge to causal inference when examining ACEs and adolescent delinquent behavior. This challenge centers on familial confounding. Particularly, the fact that familial confounds (genetic and shared environmental factors that make siblings similar to one another) contribute to the non-random selection of children into social environments whereby they are more (or less) likely to be exposed to ACEs. Indeed, a long line of behavioral genetic research has found that variation in childhood victimization and adolescent delinquency are under genetic and environmental influence (Connolly et al. 2015; Ball et al. 2008; Fisher et al. 2015), with considerable genetic overlap between the two (Barnes and Beaver 2012; Beckley et al. 2018; Vaske et al. 2012). It is possible then that genetic factors may partly explain the association between childhood adversity and adolescent delinquent behavior leading to a phenomenon known as gene–environment correlation (Kendler and Eaves 1986), whereby common genetic factors influence an individual's exposure to a given environment. To further illustrate how this could take place, Fig. 1 outlines three commonly used models of gene–environment correlation that are applied to explain the link between childhood adversity and adolescent delinquency. As can be seen, in Segment A, children with genetically influenced characteristics may be more likely to be victimized or confronted with a wide range of childhood adversities because they evoke negative responses from friends or family members, which in turn, lead to an increase in delinquency. This process is defined as evocative gene–environment correlation. Active gene–environment correlation, presented in Segment B, may also play a role whereby some children with genetically influenced characteristics are more likely to engage in delinquent behavior, which in turn, increases their risk for experiencing adversity because they have selected into unsafe environments where they commonly commit their delinquent behaviors. The last plausible model presented in Segment C is passive gene–environment correlation, which has accumulated a long line of support with regards to early life adversity and youth antisocial behavior (Jaffee et al. 2004). This model contends that observed associations between parents and children are often confounded by the fact that parents share genes with their children, which ultimately influence their personality and exposure to a variety of environments during childhood (i.e., household, neighborhood, and school setting). As a result, some children may have elevated risk for exposure to specific types of adversity during childhood and delinquent behavior not because of the direct effect of adversity, per se, but because of genetic factors they share with their parents. However, no research to date has used a longitudinal genetically informed research design to test whether genetic and shared environmental factors (i.e., familial factors) account for any portion of the association between childhood adversity and longitudinal patterns of delinquent behavior.

The Current Study

Given the paucity of research examining the link between childhood adversity and developmental patterns of delinquent behavior while controlling for familial confounds, the current study focused on evaluating this relationship using a longitudinal genetically informed research design capable of controlling for genetic and shared environmental confounds. Drawing on previous research and theoretical models of gene–environment correlation, we hypothesize that the direct association between childhood adversity and longitudinal patterns of delinquent behavior will be attenuated after genetic and shared environmental confounds are taken into account. However, we are not able to put forth a prediction at this time regarding the degree of attenuation given the limited amount of research on this association using a longitudinal genetically informed design. To evaluate these hypotheses of attenuation in a step-wise fashion, traditional latent growth curve models (LGCMs) are first estimated to assess the direct relationship between childhood adversity and starting levels and changes in delinquency while controlling for measured covariates. This first step is then followed by a series of sibling-comparison LGCMs to assess this relationship after taking into account both measured covariates and unmeasured familial confounds.

Methods

Data

The current study analyzes longitudinal data from the National Longitudinal Survey of Youth 1997 (NLSY97). The NLSY97 is a nationally representative sample of approximately 9000 U.S. youths between the ages of 12 and 16 as of December 31, 1996. Eligible youth have been assessed annually from 1997 to 2012 with retention rates over 80% since the first assessment period (U.S. Bureau of Labor Statistics 2018). Over time, youth have been assessed on their attitudes, behaviors, household environments, school experiences, substance use habits, workforce

involvement, and victimizations. All youth between ages 12 and 16 living in a sampled household during 1996 were eligible for participation during the initial assessment period. As such, there are several biologically related sibling pairs nested within the NLSY97 that can be used for genetically informative analysis. Because the NLSY97 does not include an oversample of twin pairs and is a nationally representative sample of youth, close to 90% of the sibling sample are full-siblings. This makes it possible to examine differences in childhood adversity between full-siblings while partly controlling for additive genetic confounds since full-siblings share, on average, 50% of their segregating additive genetic material and 100% of their shared environment (D'Onofrio et al. 2013). The current study takes advantage of these data to conduct a multi-method analysis using both traditional between-family modeling and sibling-comparison modeling. All full-siblings with a valid childhood adversity score and at least one valid delinquency score were included in the analytic sample. The final analytic sample included 2534 full-siblings from 994 families.

Measures

Child-specific characteristics

Childhood adversity Childhood adversity was assessed by a 9-item scale from the self-report youth questionnaire. Items were specifically designed by NLS staff to evaluate whether participants had experienced different forms of adversity and/or victimization during childhood (a sensitive period of human development). Participants were asked to report if before age 12: (1) their house or apartment had been broken into while they were home (0 = *no*, 1 = *yes*), (2) they were the victim of repeated bullying (0 = *no*, 1 = *yes*), or if, (3) they had seen someone get shot or shot at with a gun (0 = *no*, 1 = *yes*). Participants were also asked to report if they had ever had something stolen from school (0 = *no*, 1 = *yes*) or were threatened with being hurt at school (0 = *no*, 1 = *yes*). In addition to asking about school experiences, participants reported on whether their mother (0 = *no*, 1 = *yes*) or father (0 = *no*, 1 = *yes*) had served a prison sentence for a conviction before their 16th birthday. Lastly, participants were asked to report the style of parenting they received from their mother and father. Response categories ranged from 1 = *uninvolved* to 4 = *authoritative*. Participants that reported having a mother or father whom were not involved were given a value of "1" for each measure, while participants that reported having parents with other parenting styles were given a value of "0". Responses to all adversity items were summed together to create a variety scale of childhood adversity (Cronbach's alpha = .69). The strengths of this variety scale for capturing childhood adversity are that: (1) it asks participants to report on experience with both direct and indirect forms of adversity and victimization, (2) it asks participants to report on exposure to adversity in different life domains (i.e., house, school, and neighborhood) and, (3) it asks participants to report on adverse experiences that may vary between siblings from the same family. Table 1 reports the prevalence of childhood adversity in the sample. As can be seen, 40.26% of participants reported never being exposed to any type of childhood adversity, 30.27% reported experiencing one type of childhood adversity, 16.75% reported experiencing two types of childhood adversity, 8.08% reported experiencing three types of childhood adversity, 3.01% reported experiencing four types of childhood adversity, and 1.62% reported experiencing five or more types of childhood adversity.

Table 1 Descriptive statistics

	Mean/ percent	SD	Min	Max
Child-specific characteristics				
Childhood adversity	1.10	1.21	0	7
0 Adversities	40.26%	–	–	–
1 Adversity	30.27%	–	–	–
2 Adversities	16.75%	–	–	–
3 Adversities	8.08%	–	–	–
4 Adversities	3.01%	–	–	–
5 Adversities	1.18%	–	–	–
6 Adversities	.29%	–	–	–
7 Adversities	.15%	–	–	–
$\text{Delinquency}_{\text{Wave 1}}$	.99	1.01	0	6
$\text{Delinquency}_{\text{Wave 2}}$	.58	1.04	0	6
$\text{Delinquency}_{\text{Wave 3}}$	.42	.84	0	6
$\text{Delinquency}_{\text{Wave 4}}$	.37	.83	0	6
$\text{Delinquency}_{\text{Wave 5}}$	.26	.73	0	6
$\text{Delinquency}_{\text{Wave 6}}$	.22	.65	0	6
$\text{Delinquency}_{\text{Wave 7}}$	.18	.59	0	6
Substance using peers	7.10	3.30	3	15
Gang membership	5.20%	.22	0	1
Gangs in neighborhood or school	46.38%	.49	0	1
Age	14.34	1.48	12	18
Sex	50.68%	.50	0	1
Household–specific characteristics				
Family size	2.62	.65	2	4
Mother education	12.15	2.99	1	20
Father education	12.27	3.36	2	20
Household income	$41,572	$49,847	0	$251,300
Race	–	–	–	–
Non-Black/Non-Hispanic	49.67%	–	–	–
African-American	25.96%	–	–	–
Hispanic	23.41%	–	–	–
Mixed race	.95%	–	–	–

Self-reported delinquency Self-reported delinquency was assessed by a 6-item scale from the youth questionnaire administered from Wave 1–7. Participants were asked during the first wave if they have ever committed the following acts, and since the date of their last interview at follow-up waves: (1) carried a handgun; (2) purposely damaged property that did not belong to them; (3) stolen something from a store, person or house, or something that did not belong to them worth 50 dollars or more; (4) stolen something from a store or something that did not belong to them worth less than 50 dollars; (5) committed other property crimes such as fencing, selling or receiving stolen property, or cheating someone, and; (6) attacked someone with the idea of seriously hurting them or had a situation end up in a serious fight or assault of some kind. Response categories for each question were 0 = *no* and 1 = *yes*. Items were summed together to create a variety score of delinquent behavior at each wave (Cronbach's alphas = .64 – .70).

Substance using peers Substance using peers was assessed by 3 self-report items during the first wave of data collection by asking participants to report what percent of their peers smoke cigarettes, get drunk more than once a month, and/or use marijuana, inhalants, or other drugs. Response categories for all questions were 1 = *almost none (less than 10%)*, 2 = *about 25%*, 3 = *about half (50%)*, 4 = *about 75%*, and 5 = *almost all (more than 90%)*. Items were summed together to create a scale of substance using peers (Cronbach's alpha = .78).

Gang membership Gang membership was assessed during the first wave of data collection by asking participants to report if they had ever belonged to a gang. A gang was defined as "a group that hangs out together, wears gang colors or clothes, has set clear boundaries of its territory or turf, protects its members and turf against other rival gangs through fighting or threats" (U.S. Bureau of Labor Statistics 1997, p. 276). Participants were asked to respond 0 = *no* or 1 = *yes*. Approximately 5.20% of the analytic sample reported having belonged to a gang at Wave 1.

Gangs in neighborhood/school Participants were asked during Wave 1 to report if there were any gangs - as defined by the above mentioned definition - in their neighborhood or school. Response categories were 0 = *no* and 1 = *yes*. As shown in Table 1, approximately 46.38% of the sample reported having a gang in their neighborhood or school.

Age Participant age was assessed by a continuous measure of years measured during the initial survey wave. Participants were between ages 12–18 at Wave 1 and 18–24 at Wave 7.

Sex Participant sex was assessed by a binary measure where 0 = *female* and 1 = *male*. Approximately 50.68% of the analytic sample was male and 49.32% was female.

Household-specific characteristics

Family size Participant family size was assessed by the number of siblings from the same nuclear family. Family size ranged from 2 to 4 full-siblings with an average of 2.62 full-siblings per family.

Mother and father education Mother and father education levels were assessed by maternal and paternal self-reports of the highest grade completed by 1997. Response categories ranged from 1 = 1^{st} *grade* to 20 = 8^{th} *college or more*. The average amount of education for biological mothers and fathers was 12.15 and 12.27 respectively, which was equivalent to completing the 12^{th} grade or graduating high school.

Household income Household income was measured by reports of total gross household family income between 1997 and 1998. The average household family income for families in the analytic sample was $41,572.

Race Race was assessed by a binary variable where 0 = *African-American, Hispanic, Mixed Race* and 1 = *Non-Black/Non-Hispanic*.

Plan of analysis

The analysis for the current study was carried out in a series of sequential steps focused on further evaluating the environmentally mediated effect of childhood adversity on the development of delinquent behavior from adolescence to young adulthood. First, descriptive analyses were conducted to examine differences in child-specific covariates across participants who reported varying levels of childhood adversity. To examine this, a series of one-way analysis of variance (ANOVA) tests were conducted to examine statistically significant group mean differences. Bivariate associations were then examined by pearson, polychoric, and tetrachoric correlations between variables based on the distributional properties of each variable.

Second, unconditional LGCMs were estimated to evaluate the functional form of growth in delinquent behavior from Wave 1 to Wave 7. Three different growth patterns were tested: (1) linear growth characterized by steady and continual growth in one direction over time; (2) quadratic growth characterized by non-linear growth where the strength or direction of change alters over time, and; (3) latent basis growth where slope factor loadings are freed and based on observed patterns of change over time. Model

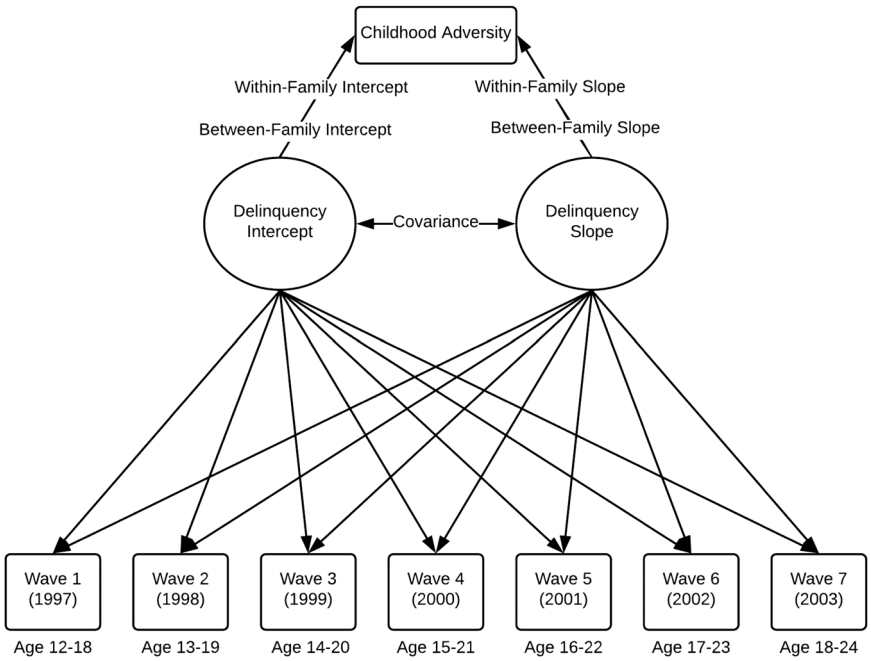

Fig. 2 Path diagram for the sibling-comparison latent growth curve model for childhood adversity and delinquency. *Notes:* All intercept loadings for Waves 1–7 were fixed to 1. Slope loadings for Wave 1 and Wave 2 were fixed to 0 and 1. Slope loadings for Waves 3–7 were allowed to freely estimate

fit for all conditional models was evaluated using the comparative fit index (CFI), Tucker–Lewis index (TLI), root mean square error of approximation (RMSEA), and standardized root mean residual (SRMR). The following model fit cut-off points were used to evaluate model fit (Hu and Bentler 1999): CFI > .90 (satisfactory fit), TLI > .90 (satisfactory fit), RMSEA < .05 (good fit), SRMR < .05 (good fit).

Third, traditional between-family LGCMs were estimated to examine whether between-family differences in childhood adversity were associated with starting levels and rates of change in delinquency from adolescence to young adulthood. The first model was a baseline model including only childhood adversity as a predictor. This was done to establish a baseline estimate of the direct link between childhood adversity and intercept and slope factors. The second model was a multivariate LGCM where child- and household-specific covariates were introduced into the LGCM to assess how much of the observed baseline association was attenuated due to measured confounds. Results from this stage of the analysis were able to provide evidence on whether childhood adversity was independently associated with initial levels and rates of change in delinquency across adolescence after controlling for a host of observable theoretical covariates.

The fourth and final stage of analysis focused on examining whether within-family variation in sibling adversity was associated with variation in starting levels and changes in delinquency over time after taking into account between-family variation. To test this possibility, sibling scores were created for each respondent. Each sibling score was created by taking their adversity score and subtracting it from the family-wide average (i.e., between-family average) of childhood adversity reported by all siblings from the same family. To illustrate, a sibling that reported experiencing 1 form of adversity and who comes from a family with 2 other siblings that reported 5 adverse experiences between them (family-wide average = 1.66 [1 + 2 + 3 = 5/3 = 2]) that sibling would have a score of −1 (sibling score = −1 [1 − 2 = −1]). The sibling with 2 adversities would have a score of 0 and the sibling with a score of 3 would have a score of 1. A graphical depiction of this modeling strategy is displayed in Fig. 2.

Results

Table 2 reports differences in child-specific characteristics based on the amount of adversity experienced during childhood. As can be seen, participants exposed to higher levels of childhood adversity reported, on average, higher levels of delinquent behavior from Wave 1 to 7, compared to participants with lower levels of childhood adversity. Participants with higher levels of childhood adversity also reported having more substance using peers during adolescence, a higher likelihood of gang membership, and a higher likelihood of having a gang in their neighborhood or school during adolescence. The results from this initial step of the analysis are in line with many other studies reporting that children exposed to multiple forms of adversity are more likely to report having risk factors for delinquent behavior (e.g., Dong et al. 2013; Finkelhor et al. 2005; Ford

Table 2 Differences in child-specific characteristics by frequency of childhood adversity

	0 Adversities (n = 1020) (40.26%)	1 Adversity (n = 767) (30.27%)	2 Adversities (n = 424) (16.75%)	3 Adversities (n = 206) (8.08%)	4 Adversities (n = 76) (3.01%)	5+ Adversities (n = 41) (1.62%)	F / χ^2
	Mean/Percent	Mean/Percent	Mean/Percent	Mean/Percent	Mean/Percent	Mean/Percent	
$Delinquency_{Wave\ 1}$	.57	.87	1.20	1.54	1.95	2.50	30.77**
$Delinquency_{Wave\ 2}$	.33	.47	.70	.80	1.09	1.41	10.01**
$Delinquency_{Wave\ 3}$	.29	.38	.40	.61	.92	1.36	6.90**
$Delinquency_{Wave\ 4}$	.22	.34	.35	.58	.60	.70	5.70**
$Delinquency_{Wave\ 5}$	.18	.24	.26	.39	.41	.54	3.07**
$Delinquency_{Wave\ 6}$	.16	.19	.23	.25	.46	.51	3.01**
$Delinquency_{Wave\ 7}$	.11	.19	.14	.22	.29	.38	2.18*
Substance peers	6.36	6.62	7.15	7.85	7.97	8.72	4.22**
Gang membership	2.19%	1.94%	6.14%	9.09%	9.98%	27.27%	54.35**
Neighborhood/ school	35.10%	42.75%	55.26%	59.09%	60.04%	81.82%	52.63**
Age	14.27	14.24	14.21	14.25	14.00	14.72	.75
Male	45.07%	51.21%	53.07%	58.18%	63.41%	54.55%	12.50*

* $p < .05$; ** $p < .01$

Table 3 Correlations for childhood adversity, delinquency, and child-specific characteristics

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Childhood adversity	–												
2. $Delinquency_{Wave\ 1}$	.32**	–											
3. $Delinquency_{Wave\ 2}$	.19**	.40**	–										
4. $Delinquency_{Wave\ 3}$	.14**	.34**	.37**	–									
5. $Delinquency_{Wave\ 4}$	.13**	.31**	.34**	.43**	–								
6. $Delinquency_{Wave\ 5}$	.11**	.21**	.24**	.34**	.46**	–							
7. $Delinquency_{Wave\ 6}$	.09**	.21**	.22**	.26**	.35**	.39**	–						
8. $Delinquency_{Wave\ 7}$	.07**	.19**	.21**	.25**	.31**	.35**	.40**	–					
9. Substance using peers	.17**	.27**	.10**	.05**	.09**	.05**	.04*	.03	–				
10. Gang membership	.34**	.44**	.20**	.16**	.18**	.10**	.10**	.11**	.16**	–			
11. Gangs in neighborhood or school	.24**	.19**	.12**	.10**	.09**	.08**	.09**	.11**	.20**	.15**	–		
12. Age	−.01	.08**	−.02	−.09**	−.03	−.02	−.04	−.02	.48**	.05**	.08**	–	
13. Sex	.12**	.22**	.17**	.15**	.16**	.16**	.14**	.14**	−.12**	.08**	.01	−.02	–

* $p < .05$; ** $p < .01$

et al. 2010) and recurring victimization (e.g., Finkelhor et al. 2007b; Ford et al. 2013).

Table 3 presents the bivariate correlations between childhood adversity, delinquency, and child-specific covariates. Childhood adversity was positively and significantly associated with delinquency across all waves ($rs = .07$–$.32$, $p < .01$), substance using peers ($r = .17$, $p < .01$), gang membership ($r = .34$, $p < .01$), and gangs in adolescents' neighborhood or school ($r = .24$, $p < .01$). Wave-to-wave correlations for delinquency also revealed that delinquent behavior was moderately correlated over time ($rs = .37$–$.46$, $p < .01$) and that gang membership ($rs = .10$–$.44$, $p < .01$), gang presence in neighborhood or school ($rs = .09$–$.19$, $p < .01$), and sex ($rs = .14$–$.22$, $p < .01$) were significantly associated with delinquency from Wave 1 to 7. Having substance using peers was also positively and significantly associated with delinquent behavior from Wave 1 to 6 ($rs = .04$–$.27$, $p < .01$), but not at Wave 7 ($r = .03$, $p > .05$).

The next step in the analysis focused on fitting an unconditional LGCM to the data to evaluate the pattern of change in delinquent behavior in the sample. Table 4 presents the values from all model fit indices for a linear, quadratic, and latent basis growth model. As presented, all variations adequately fit the data and suggested that there was significant variation in starting levels (intercept) and rates of change (slope) among adolescents. Among the

Table 4 Parameter estimates and model fit statistics for unconditional latent growth curve models for delinquency

	Intercept		Slope		Covariance	Model fit indices				
	Mean	Variance	Mean	Variance		$\Delta\chi^{2\ (\Delta df)}$	CFI	TLI	RMSEA	SRMR
Linear growth	.99**	.92**	−.47**	.15**	−.39**	–	.93	.90	.06	.04
Quadratic growth	.99**	.92**	−.37**	.18**	−.40**	41.03** (7)	.95	.92	.05	.03
Latent basis growth	.99**	.92**	−.40**	.21**	−.35**	36.54** (7)	.96	.92	.05	.02

CFI comparative fit index, *TLI* Tucker–Lewis index, *RMSEA* root mean square error of approximation, *SRMR* standardized root mean square residual

**$p < .01$

Table 5 Baseline and multivariate latent growth curve models for delinquency

	Baseline model				Multivariate model				Baseline sibling-comparison model				Multivariate sibling-comparison model			
	Intercept		Slope		Intercept		Slope		Intercept		Slope		Intercept		Slope	
	b	SE	*b*	SE	*b*	SE	*b*	SE	*b*	SE	*b*	SE	*b*	SE	*b*	SE
Childhood adversity	.43**	.02	.11**	.02	.39**	.02	.07*	.02	.18*	.03	.02	.01	.16*	.03	.02	.01
Substance using peers	–	–	–	–	.09**	.01	−.10*	.05	–	–	–	–	.04*	.01	−.04	.02
Gang membership	–	–	–	–	2.23**	.10	.17**	.09	–	–	–	–	1.09*	.07	.04	.02
Gangs in Neighborhood/school	–	–	–	–	.24**	.05	.02	.01	–	–	–	–	.02	.01	.01	.01
Age	–	–	–	–	−.04	.01	−.05*	.02	–	–	–	–	−.01	.01	−.03*	.01
Sex	–	–	–	–	.53**	.04	.31**	.01	–	–	–	–	.15**	.01	.20*	.02
Family size	–	–	–	–	.08*	.02	.02	.01	–	–	–	–	–	–	–	–
Mother education	–	–	–	–	−.01	.01	−.01	.01	–	–	–	–	–	–	–	–
Father education	–	–	–	–	−.02	.01	−.01	.01	–	–	–	–	–	–	–	–
Household Income	–	–	–	–	−.08*	.03	.02	.01	–	–	–	–	–	–	–	–
Race	–	–	–	–	.25**	.05	.10**	.03	–	–	–	–	–	–	–	–

Notes: Unstandardized coefficients presented

*$p < .05$; **$p < .01$.

estimated LGCMs, model fit statistics revealed that a latent basis LGCM provided a better fit to the data ($\Delta\chi^2 = 36.54$; CFI = .96, TLI = .92, RMSEA = .05, SRMR = .02) compared to a linear LGCM (CFI = .93, TLI = .90, RMSEA = .06, SRMR = .04) or quadratic LGCM ($\Delta\chi^2 = 41.03$; CFI = .95, TLI = .92, RMSEA = .05, SRMR = .03). Parameter estimates from the best-fitting latent basis model revealed that there was significant variation in starting levels of delinquency (Intercept var. = .92, $p < .01$) and both mean decreases (Slope mean = -.40, $p < .01$) and variation in the rate of decreases from adolescence to young adulthood (Slope var. = .21, $p < .01$). The negative and significant correlation between the intercept and slope factor (r = -.35, $p < .01$)—which is often observed with LGCMs—suggests that adolescents with higher levels of delinquent behavior at Wave 1 demonstrated, on average, faster decreases in delinquent behavior over time. In addition, preliminary multi-level analyses examining the degree of between- and within-family variation in childhood adversity in the analytic sample found statistically significant between-family (79.89%, $p < .001$) and within-family (20.11%, $p < .01$) variation, thus allowing for the examination of sibling comparisons.

After establishing that there were significant individual differences in starting levels and rates of change in delinquency, the next step in the analysis was to examine whether childhood adversity was associated with differences in starting levels and changes over time. Table 5 presents the estimates from four estimated LCGMs with each subsequent model controlling for additional confounders. The baseline model in Table 5 revealed that childhood adversity was positively and significantly associated with both starting levels ($b = .43$, $p < .01$) and rates of change ($b = .11$, $p < .01$) suggesting that adolescents with higher levels of childhood adversity reported higher starting levels of delinquent behavior during adolescence and demonstrated slower rates of decline in delinquent behavior from adolescence to young adulthood. Estimates from the subsequent

multivariate model, which included measured child-specific and household-specific covariates, revealed that the effect of childhood adversity on starting levels of delinquent behavior ($b = .39$, $p < .01$) and change in delinquent behavior ($b = .07$, $p < .01$) was slightly attenuated, but remained statistically significant. The third model introduced sibling-comparisons of childhood victimization as a predictor of intercept and slope factors for delinquency. For comparison purposes, this model did not include child-specific covariates that vary between siblings (i.e., percentage of substance using peers, gang membership, self-perceptions of gangs in neighborhood or school, age, and sex), but did take into account unobserved genetic and shared environmental confounds shared between full-sibling pairs. The results from this model showed that after controlling for unobserved familial confounders, childhood adversity was still associated with higher starting levels of delinquent behavior during adolescence ($b = .18$, $p < .05$), but not rates of change in delinquent behavior from adolescence to emerging adulthood ($b = .02$, $p > .05$). The fourth and final model was a multivariate sibling-comparison model that included controls for genetic and shared environmental confounders as well as child-specific covariates. As shown, the inclusion of child-specific covariates slightly attenuated the effect of childhood adversity on initial levels of delinquent behavior, but the association remained statistically significant ($b = .16$, $p < .05$).

Discussion

A long line of research has reported a positive relationship between childhood adversity and later life delinquent behavior (e.g., Brumley et al. 2017; Duke et al. 2010). However, few studies have controlled for unobservable genetic and shared environmental effects when examining this association. The goal of the current study was to address this gap in the existing body of literature by examining the relationship between childhood adversity and developmental patterns of delinquent behavior after controlling for observable confounds and unobservable genetic and shared environmental confounds. Specifically, our study contributed to the literature by examining this relationship using full-sibling data from a nationally representative sample of youth. The structure of sibling relationships within families in this sample provided a unique opportunity to conduct a quasi-experimental test of the causal hypothesis that childhood adversity causes developmental patterns of delinquent behavior across different stages of the life course net of genetic and shared environmental confounding. The employed analysis produced two key findings that warrant further discussion.

First, the results from estimated baseline LGCMs replicated previous results that childhood adversity is a risk factor for delinquency during adolescence (Cudmore et al. 2017; Farrell and Zimmerman 2017; Turner et al. 2016). Childhood adversity remained a significant predictor of starting levels of delinquent behavior despite the inclusion of measured and unmeasured confounds. Although far from definitive, this result is in line with the quasi-causal interpretation of the association between childhood adversity and delinquent behavior during adolescent development, albeit to a more modest degree than prior research has suggested. This statistically significant finding in the multivariate sibling-comparison model implies that after trying to remove the influence of shared genetic and environmental factors, childhood adversity exerted an environmentally mediated effect on delinquent behavior, suggesting a short or contemporaneous effect of adversity on levels of adolescent delinquency. This finding coincides with arguments from criminological theories such as general strain theory (Agnew 2001), which holds that recent adversities are more likely to be associated with maladaptive behavior such as delinquency, substance abuse, or criminal behavior because victims are using these methods to cope with traumatic stress. It is important to note, however, that childhood adversity and starting levels of delinquency were assessed at the same time-point. Thus, our results cannot establish temporal ordering, and it must be considered equally possible that our findings might be due to reverse causation whereby individuals that demonstrate high levels of delinquent behavior are more likely to be experience multiple forms of adversity.

Second, despite evidence of a statistically significant association between childhood adversity and slower rates of decline in delinquency in traditional multivariate LGCMs, the addition of controls for unobserved genetic and environmental confounds rendered this effect non-significant. While we found that childhood adversity appears to have a robust effect on starting levels of delinquent behavior during adolescence, the results suggest that the effect stemming from childhood adversity did not necessarily carry over to developmental patterns of delinquent behavior. This finding therefore suggests that the effect of childhood adversity on delinquent behavior may be time-limited whereby as youth mature they are influenced more directly by other environmental sources of influence that correlate with declines in delinquent involvement such as college attendance, peer association, employment, and intimate partner relationships; all partly influenced by genetics and self-selection (Kendler and Baker 2007). Another possible and complimentary explanation for this finding could be that as youth progress through adolescence and enter adulthood they begin to have more control over their exposure to environmental contexts, thus allowing for a greater impact of active gene–

environment correlation on this developmental process across the life course. Future research should begin to explore the unique role of active gene-environment correlation and gene × environment interaction on this association using longitudinal twin and sibling research designs (Burt 2015).

The current study has limitations that must be noted. First, while the quasi-experimental, sibling-comparison approach aims to approach causality by controlling for unobserved familial confounding, it cannot prove causality. As with every social science research design, the analyses do not account for every possible variable that may confound the association. It is possible that another unmeasured factor that varied between full-siblings and was correlated with variations in childhood adversity is the causal environmental risk factor. Future research using sibling-comparisons or other family-based research designs will need to explore the role of additional risks associated with childhood adversity and delinquent behavior. Second, the present study could not determine whether the confounding factors in the association between childhood adversity and rates of change in delinquency were genetic or environmental in origin. While previous analyses have found that genetic and nonshared environmental influences often account for variation in childhood adversity, antisocial behavior, and delinquency (McCrory et al. 2012; Rhee and Waldman 2002), future research should continue to employ family-based research models such as the discordant twin design or sibling-comparison research design to assess if different forms of adversity are important nonshared environmental influences in the development of delinquent behavior (see Beaver 2008 and Jaffee et al. 2012). Failure to use such designs make it impossible to capture nonshared environmental effects, which a long line of research suggests explains more variation in delinquent behavior than shared environmental effects (Boisvert et al. 2012; Connolly and Beaver 2014; Rodgers et al. 2001; Wright et al. 2008). Third, smaller studies of childhood adversity with more targeted sample populations can conduct more intensive assessments of adverse life experiences that occur not only during childhood, but also infancy and the prenatal period. The large scale sample analyzed in the current study does not replace more focused and targeted studies on early life adversities and their relation to maladaptive behavioral development.

Conclusion

While a great deal of research has focused on examining the potential impact of traumatic childhood experiences on offspring problem behavior, far less attention has been given to assessing this association using family-based research designs to rule out familial confounds. Our findings, based on a nationally representative sample of American youths and a rigorous quasi-experimental, family-based research design, found evidence that strengthens the inference that childhood adversity has a direct effect on delinquent behavior during adolescence, but not on developmental trajectories of delinquent behavior across the life course. The finding that unmeasured familial confounds related to childhood adversity may be responsible for individual differences in rates of decline in delinquency from adolescence to emerging adulthood suggests that members of the research and treatment community need to focus more on examining the underlying biological/genetic and environmental factors associated with exposure and response to childhood adversity, particularly factors that may vary across siblings. Future research with a focus on identifying key unique environmental factors related to maladaptive behaviors, such as delinquency, will help to create more targeted and effective early intervention programs for at-risk youth. Findings from the current study support this argument that familial factors play an important role in the development of delinquent behavior and highlight the need for more researchers in fields such as criminal justice, criminology, psychology, and sociology to employ family-based designs to better understand the mechanisms involved in creating individual differences in criminal and delinquent behavior.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

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References

Agnew, R. (2001). Building on the foundation of general strain theory: Specifying the types of strain most likely to lead to crime and delinquency. *Journal of Research in Crimean and Delinquency*, *38*, 319–361.

Baglivio, M. T., Jackowski, K., Greenwald, M. A., & Howell, J. C. (2014). Serious, violent, and chronic juvenile offenders: A statewide analysis of prevalence and prediction of subsequent recidivism using risk and protective factors. *Criminology & Public Policy*, *13*, 83–116.

Baglivio, M. T., Wolff, K. T., Piquero, A. R., & Epps, N. (2015). The relationship between adverse childhood experiences (ACE) and juvenile offending trajectories in a juvenile offender sample. *Journal of Criminal Justice*, *43*, 229–241.

Ball, H. A., Arseneault, L., Taylor, A., Maughan, B., Caspi, A., & Moffitt, T. E. (2008). Genetic and environmental influences on victims, bullies and bully-victims in childhood. *Journal of Child Psychology and Psychiatry*, *49*, 104–112.

Barnes, J. C., & Beaver, K. M. (2012). Extending research on the victim–offender overlap: Evidence from a genetically informative analysis. *Journal of Interpersonal Violence*, *27*, 3299–3321.

Beaver, K. M. (2008). Nonshared environmental influences on adolescent delinquent involvement and adult criminal behavior. *Criminology*, *46*, 341–369.

Beckley, A. L., Caspi, A., Arseneault, L., Barnes, J. C., Fisher, H. L., Harrington, H., & Moffitt, T. E. (2018). The developmental nature of the victim-offender overlap. *Journal of Developmental and Life-Course Criminology*, *4*, 24–49.

Boisvert, D., Wright, J. P., Knopik, V., & Vaske, J. (2012). Genetic and environmental overlap between low self-control and delinquency. *Journal of Quantitative Criminology*, *28*, 477–507.

Brumley, L. D., Jaffee, S. R., & Brumley, B. P. (2017). Pathways from childhood adversity to problem behaviors in young adulthood: The mediating role of adolescents' future expectations. *Journal of Youth and Adolescence*, *46*, 1–14.

Burt, S. A. (2015). Evidence that the gene–environment interactions underlying youth conduct problems vary across development. *Child Development Perspectives*, *9*, 217–221.

Connolly, E. J., & Beaver, K. M. (2014). Examining the genetic and environmental influences on self-control and delinquency: Results from a genetically informative analysis of sibling pairs. *Journal of Interpersonal Violence*, *29*, 707–735.

Connolly, E. J., & Beaver, K. M. (2016). Considering the genetic and environmental overlap between bullying victimization, delinquency, and symptoms of depression/anxiety. *Journal of Interpersonal Violence*, *31*, 1230–1256.

Connolly, E. J., Schwartz, J. A., Nedelec, J. L., Beaver, K. M., & Barnes, J. C. (2015). Different slopes for different folks: Genetic influences on growth in delinquent peer association and delinquency during adolescence. *Journal of Youth and Adolescence*, *44*, 1413–1427.

Connolly, E. J., & Beaver, K. M. (2015). Assessing the salience of gene-environment interplay in the development of anger, family conflict, and physical violence: A biosocial test of General Strain Theory. *Journal of Criminal Justice*, *43*, 487–497.

Cudmore, R. M., Cuevas, C. A., & Sabina, C. (2017). The impact of polyvictimization on delinquency among Latino adolescents: A general strain theory perspective. *Journal of Interpersonal Violence*, *32*, 2647–2667.

Darawshy, N. A. S., & Haj-Yahia, M. M. (2018). Internalizing and externalizing symptoms among Palestinian adolescents from Israel as consequences of their exposure to community violence: Are they moderated by their self-efficacy and collective efficacy? *Child Abuse & Neglect*, *79*, 61–73.

Dong, F., Cao, F., Cheng, P., Cui, N., & Li, Y. (2013). Prevalence and associated factors of poly-victimization in Chinese adolescents. *Scandinavian Journal of Psychology*, *54*, 415–422.

D'Onofrio, B. M., Lahey, B. B., Turkheimer, E., & Lichtenstein, P. (2013). Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. *American Journal of Public Health*, *103*, S46–S55.

Duke, N. N., Pettingell, S. L., McMorris, B. J., & Borowsky, I. W. (2010). Adolescent violence perpetration: Associations with multiple types of adverse childhood experiences. *Pediatrics*, *125*, 778–786.

Farrell, C., & Zimmerman, G. M. (2017). Does offending intensify as exposure to violence aggregates? Reconsidering the effects of repeat victimization, types of exposure to violence, and poly-victimization on property crime, violent offending, and substance use. *Journal of Criminal Justice*, *53*, 25–33.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) study. *American Journal of Preventive Medicine*, *14*, 245–258.

Finkelhor, D., Ormrod, R. K., & Turner, H. A. (2007a). Poly-victimization: A neglected component in child victimization. *Child Abuse & Neglect*, *31*(1), 7–26.

Finkelhor, D., Ormrod, R. K., & Turner, H. A. (2007b). Re-victimization patterns in a national longitudinal sample of children and youth. *Child Abuse & Neglect*, *31*, 479–502.

Finkelhor, D., Ormrod, R. K., Turner, H. A., & Hamby, S. L. (2005). Measuring poly-victimization using the Juvenile Victimization Questionnaire. *Child Abuse & Neglect*, *29*, 1297–1312.

Finkelhor, D., Turner, H., Ormrod, R., & Hamby, S. L. (2009). Violence, abuse, and crime exposure in a national sample of children and youth. *Pediatrics*, *124*(5), 1411–1423.

Fisher, H. L., Caspi, A., Moffitt, T. E., Wertz, J., Gray, R., Newbury, J., & Odgers, C. L. (2015). Measuring adolescents' exposure to victimization: The environmental risk (E-Risk) longitudinal twin study. *Development and Psychopathology*, *27*, 1399–1416.

Ford, J. D., Charak, R., Modrowski, C. A., & Kerig, P. K. (2018). PTSD and dissociation symptoms as mediators of the relationship between polyvictimization and psychosocial and behavioral problems among justice-involved adolescents. *Journal of Trauma & Dissociation*, *19*(3), 325–346.

Ford, J. D., Elhai, J. D., Connor, D. F., & Frueh, B. C. (2010). Polyvictimization and risk of posttraumatic, depressive, and substance use disorders and involvement in delinquency in a national sample of adolescents. *Journal of Adolescent Health*, *46*, 545–552.

Ford, J. D., Grasso, D. J., Hawke, J., & Chapman, J. F. (2013). Polyvictimization among juvenile justice-involved youths. *Child Abuse & Neglect*, *37*, 788–800.

Fox, B. H., Perez, N., Cass, E., Baglivio, M. T., & Epps, N. (2015). Trauma changes everything: Examining the relationship between adverse childhood experiences and serious, violent and chronic juvenile offenders. *Child Abuse & Neglect*, *46*, 163–173.

Graham-Bermann, S. A., Howell, K. H., Miller, L. E., Kwek, J., & Lilly, M. M. (2010). Traumatic events and maternal education as predictors of verbal ability for preschool children exposed to intimate partner violence (IPV). *Journal of Family Violence*, *25*, 383–392.

Hardaway, C. R., Larkby, C. A., & Cornelius, M. D. (2014). Socioemotional adjustment as a mediator of the association between exposure to community violence and academic performance in low-income adolescents. *Psychology of Violence*, *4*, 281–293.

Heleniak, C., King, K. M., Monahan, K. C., & McLaughlin, K. A. (2018). Disruptions in emotion regulation as a mechanism linking community violence exposure to adolescent internalizing problems. *Journal of Research on Adolescence*, *28*, 229–244.

Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*, 1–55.

Jaffee, S. R., Caspi, A., Moffitt, T. E., & Taylor, A. (2004). Physical maltreatment victim to antisocial child: evidence of an environmentally mediated process. *Journal of Abnormal Psychology*, *113*, 44.

Jaffee, S. R., Strait, L. B., & Odgers, C. L. (2012). From correlates to causes: Can quasi-experimental studies and statistical innovations bring us closer to identifying the causes of antisocial behavior? *Psychological Bulletin*, *138*, 272.

Kavish, N., Connolly, E. J., & Boutwell, B. B. (2018). Genetic and environmental contributions to the association between violent victimization and major depressive disorder. *Personality and Individual Differences*. https://doi.org/10.1016/j.paid.2018.05.034

Kendler, K. S., & Baker, J. H. (2007). Genetic influences on measures of the environment: a systematic review. *Psychological Medicine*, *37*, 615–626.

Kendler, K. S., & Eaves, L. J. (1986). Models for the joint effect of genotype and environment on liability to psychiatric illness. *The American Journal of Psychiatry*, *143*, 279–289.

Kretschmar, J. M., Tossone, K., Butcher, F., & Flannery, D. J. (2017). Patterns of poly-victimization in a sample of at-risk youth. *Journal of Child & Adolescent Trauma*, *10*, 363–375.

Lahey, B. B., & D'Onofrio, B. M. (2010). All in the family: Comparing siblings to test causal hypotheses regarding environmental influences on behavior. *Current Directions in Psychological Science*, *19*, 319–323.

McCrory, E., De Brito, S. A., & Viding, E. (2012). The link between child abuse and psychopathology: A review of neurobiological and genetic research. *Journal of the Royal Society of Medicine*, *105*, 151–156.

Obsuth, I., Mueller Johnson, K., Murray, A. L., Ribeaud, D., & Eisner, M. (2017). Violent poly-victimization: The longitudinal patterns of physical and emotional victimization throughout adolescence (11–17 years). *Journal of Research on Adolescence*. https://doi.org/10.1111/jora.12365.

Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin*, *128*, 490.

Rodgers, J. L., Buster, M., & Rowe, D. C. (2001). Genetic and environmental influences on delinquency: DF analysis of NLSY kinship data. *Journal of Quantitative Criminology*, *17*, 145–168.

Schaefer, J. D., Moffitt, T. E., Arseneault, L., Danese, A., Fisher, H. L., Houts, R., & Caspi, A. (2018). Adolescent victimization and early-adult psychopathology: Approaching causal inference using a longitudinal twin study to rule out noncausal explanations. *Clinical Psychological Science*, *6*, 352–371.

Sherr, L., Hensels, I. S., Skeen, S., Tomlinson, M., Roberts, K. J., & Macedo, A. (2015). Exposure to violence predicts poor educational outcomes in young children in South Africa and Malawi. *International Health*, *8*, 36–43.

Slutkin, G. (2017). Reducing violence as the next great public health achievement. Nature Human Behaviour, *1*, s41562-41016-40025.

Turner, H. A., Shattuck, A., Finkelhor, D., & Hamby, S. (2016). Polyvictimization and youth violence exposure across contexts. *Journal of Adolescent Health*, *58*, 208–214.

U.S. Bureau of Labor Statistics (1997). National longitudinal survey of youth 1997 cohort round one questionnaire. http://www.nlsinfo.org/ordering/display_db.php3#NLSY97. Accessed 13 Aug 2018.

U.S. Bureau of Labor Statistics (2018). Retention & reasons for non-interview. https://www.nlsinfo.org/content/cohorts/nlsy97/intro-to-the-sample/retention-reasons-non-interview/page/0/0/#retention. Accessed 13 Aug 2018.

Vaske, J., Boisvert, D., & Wright, J. P. (2012). Genetic and environmental contributions to the relationship between violent victimization and criminal behavior. *Journal of Interpersonal Violence*, *27*, 3213–3235.

Wolff, K. T., Baglivio, M. T., & Piquero, A. R. (2017). The relationship between adverse childhood experiences and recidivism in a sample of juvenile offenders in community-based treatment. *International Journal of Offender Therapy and Comparative Criminology*, *61*, 1210–1242.

Wright, J., Beaver, K., Delisi, M., & Vaughn, M. (2008). Evidence of negligible parenting influences on self-control, delinquent peers, and delinquency in a sample of twins. *Justice Quarterly*, *25*, 544–569.

Zimmerman, G. M., & Kushner, M. (2017). Examining the contemporaneous, short-term, and long-term effects of secondary exposure to violence on adolescent substance use. *Journal of Youth and Adolescence*, *46*, 1933–1952.

Zimmerman, G. M., & Posick, C. (2016). Risk factors for and behavioral consequences of direct versus indirect exposure to violence. *American Journal of Public Health*, *106*, 178–188.

Eric J. Connolly is an Assistant Professor in the Department of Criminal Justice and Criminology at Sam Houston State University. His research interests include biosocial criminology, criminological theory, family-based research designs, life-course/developmental criminology, and victimology.

Nicholas Kavish is a doctoral student in the Clinical Psychology program at Sam Houston State University. His research interests include the assessment, etiology, and correlates of personality psychopathology, psychopathic traits, and antisocial behavior.