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Considering the Genetic and Environmental Overlap Between Bullying Victimization, Delinquency, and Symptoms of Depression/Anxiety

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Abstract

Emerging evidence from longitudinal research suggests that bullied children are more likely to develop antisocial tendencies and mental health problems later in life. Less research, however, has used genetically sensitive research designs to control for genetic confounding and examine whether the well-supported association between bullying victimization and maladaptive development is partially accounted for by common genetic and environmental influences. Using sibling data from the National Longitudinal Survey of Youth 1997, the current study used a series of bivariate liability-threshold models to disentangle the genetic and environmental influences on observed covariance between repeated bullying victimization, delinquent involvement, and symptoms of depression/anxiety. Results revealed that common additive genetic and nonshared environmental effects accounted for the covariance in liability between bullying victimization and delinquent involvement as well as bullying victimization and symptoms of depression/anxiety. The results

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suggest the presence of genotype–environment correlation (*r*GE) between repeated victimization and maladaptive development.

Keywords
bullying, youth violence, criminology

Introduction

In recent years, bullying victimization has emerged as a serious health concern among children and adolescents. Part of the reason for the widespread focus on bullying and the consequences associated with it stems, in part, from the relative frequency of bullying. Prevalence rates of bullying victimization in the United States, for instance, range from 15% to 28% among elementary and high school students (National Center for Education Statistics, U.S. Department of Education, 2011). Empirical evidence suggests that bullied youth are at an increased risk of developing a wide range of maladaptive behaviors including low self-esteem (Callaghan & Joseph, 1995), depression (Neary & Joseph, 1994), violent behavior (Kim, Leventhal, Koh, Hubbard, & Boyce, 2006; Nansel, Overpeck, Haynie, Ruan, & Scheidt, 2003), and under some circumstances, suicidal thoughts (Cleary, 2000). Compared with children who are bullied once or twice, chronically bullied children tend to report higher levels of psychiatric problems during childhood (Winsper, Lereya, Zanarini, & Wolke, 2012) and poor health in later adulthood (Wolke, Copeland, Angold, & Costello, 2013). Taken together, further understanding the etiological origins of being bullied and maladaptive behavioral development can inform intervention strategies, public health initiatives, and quite possibly, the criminal justice system.

Mounting evidence on bullying victimization has highlighted the detrimental effects of being bullied on children's health and behavior. For example, one study found that children who were bullied at age 5 reported more internalizing and externalizing problems at age 7 compared with children who were not bullied (Arseneault et al., 2006). At the same time, other evidence suggests that repeated bullying victimization is predicted by both internalizing and externalizing problems such as physical aggression (Barker et al., 2008), a low level of assertiveness (Egan & Perry, 1998), and depression (Hodges & Perry, 1999). Results from a recent meta-analysis found that across several different longitudinal studies using different samples, internalizing emotional problems increase both the risk of being bullied and the consequences of bullying victimization (Reijntjes,

Kamphuis, Prinzie, & Telch, 2010; see also Cook, Williams, Guerra, Kim, & Sadek, 2010). Such findings thus suggest that victimization and maladjustment may operate in a reciprocal manner where one increases the risk of another (Hodges & Perry, 1999).

Despite the wealth of research examining the association between early life bullying victimization and maladaptive behavioral development, much evidence supporting these associations is drawn from cross-sectional or short-term longitudinal data and does not control for genetic influences. Consequently, less is known about whether and to what extent genetic effects explain individual differences in being bullied and the development of externalizing and internalizing problems. Using genetically informed methods, however, researchers can help disentangle these unique effects and shed new light on the underlying biosocial factors that may operate to increase the risk of both victimization and maladaptive development. As such, the aim of the current study was to assess whether and to what extent the pathways between repeated bullying victimization, delinquent activity, and symptoms of depression/anxiety were accounted for by common genetic and environmental influences.

Bullying Victimization and Delinquent Behavior

A growing body of research indicates that chronic victims of bullying are more likely to develop externalizing problem behaviors (Arseneault et al., 2006) and engage in various forms of delinquent activity (Rusby, Forrester, Biglan, & Metzler, 2005; Wong & Schonlau, 2013). Using self-reported and official crime data from Finland, Sourander et al. (2007) found that being victimized at age 8 was positively associated with committing property offenses between the ages of 16 and 20. Other research has found positive associations between frequent peer victimization in adolescence and delinquent tendencies such as alcohol use, aggression, street crime, and antisocial behavior (Gunter & Newby, 2014; Rusby et al., 2005). Findings from a recent study analyzing a nationally representative sample of adolescents in the United States showed that peer victimization predicted an increase in delinquent behavior after controlling for exposure to other forms of violence and prior delinquency (Jackson et al., 2013). To better control for potential confounders, Wong and Schonlau (2013) went beyond correlational research and used propensity score matching (PSM) with longitudinal data from the National Longitudinal Survey of Youth 1997 (NLSY97). PSM results indicated that early life bullying victimization predicted a variety of delinquent behaviors including physical assault, theft, holding stolen property, running away, selling drugs, and vandalism.

Although an emerging body of evidence shows that bullying victimization and delinquency are positively associated, few studies have controlled for the potentially confounding effects of genetic factors that may explain variation in personality characteristics that correlate with both victimization and delinquent behavior (but see Ball et al., 2008). The present study therefore extends contemporary quasi-experimental research on bullying victimization and delinquency by using sibling data to tease apart the genetic and environmental contributions to the covariance between repeated bullying victimization and delinquent activity.

Bullying Victimization and Internalizing Problems

An extensive line of research has found that bullying victimization is linked to a wide range of mental health problems (Neary & Joseph, 1994; Veenstra et al., 2005). For example, previous studies have identified strong links between chronic bullying victimization and depression/anxiety among other symptoms of internalizing behaviors (for a review, see Arseneault, Bowes, & Shakoor, 2010). Cross-sectional evidence from a recent study by Turner, Exum, Brame, and Holt (2013) found that various forms of bullying victimization (e.g., cyber, verbal, and physical bullying) were associated with levels of depression and suicidal thoughts. These findings correspond with psychiatric studies that have found similar significant associations between childhood bullying victimization and suicide attempts in preadolescence (Winsper et al., 2012), even after taking into account childhood conduct disorder and depression (Klomek et al., 2009). Moreover, other research has observed a dose–response effect between the persistence of being bullied and demonstrating psychiatric symptoms during early adolescence (Schrier et al., 2009). Considered together, mounting evidence indicates that childhood bullying victimization increases the risk of negative mental health.

Using longitudinal data from the NLSY97, Bouffard and Koeppel (2014) recently examined the long-term association between childhood bullying victimization and reports of both negative mental and physical health in early adulthood. After controlling for a range of potential confounding factors, a series of regression models revealed that chronic victims reported higher levels of negative health, emotional problems, and risky behaviors later in life.[1] Bouffard and Koeppel concluded that repeated victimization early in life may play an important role in the development of mental and physical health problems. Moreover, Wolke and colleagues (2013) analyzed a population-based sample of children from North Carolina and found a positive association between chronic victimization and social-relationship problems in adulthood.

Taking into consideration the wealth of evidence supporting an association between childhood bullying victimization and internalizing problematic development during adolescence and adulthood, understanding the factors that may mediate this association is critical to identifying effective targets for intervention during childhood. However, much of the above-mentioned cross-sectional and longitudinal research does not control for genetic confounding between chronic victimization and mental health development. As a result, it becomes difficult to identify the precise mechanisms, genetic or environmental, that may account for individual differences in chronic victimization and internalizing problems.

Explanations for the Association Between Bullying Victimization, Delinquency, and Internalizing Problems

In recent years, a number of theoretical explanations have been offered to help identify the mechanisms involved in the association between bullying victimization, behavioral problems, and mental health issues. In general, two key theoretical explanations have been advanced more than others. The first comes from general strain theory (GST). According to GST, symptoms of negative health may emerge as a response to being victimized depending on the individual's response to his or her victimization (Agnew, 2002). Specifically, some individuals may respond with anger or aggression generated by feelings of injustice. This type of emotional response (or negative state) may therefore motivate victims to seek corrective action against their perpetrator (or bully) through delinquent or violent means. Alternatively, individuals might turn to internalizing behaviors such as substance use or self-harm to cope with mental health problems that emerge as a result of their victimization. Individuals who choose to internalize their emotions, rather than externalize, may cope in this manner because they have used it before and suffer from low self-esteem, anxiety, or symptoms of depressive disorder. Using cross-sectional data, Hay, Meldrum, and Mann (2010) tested these GST arguments and found that both "traditional" bulling (e.g., physical and verbal abuse) and "cyber" bullying were significantly associated with delinquency, self-harm, and suicidal thoughts. However, given the cross-sectional nature of the analysis, the authors caution interpreting causal order between victimization and self-harm behaviors. Taken together, GST offers a foundation to understanding how both externalizing and internalizing problem behaviors may emerge as a result of being repeatedly bullied.

Second, a body of criminological research suggests that individuals with low levels of self-control (Gottfredson & Hirschi, 1990) are more likely to be victimized than those with high levels of self-control (Schreck, 1999). Given

that individuals with low self-control tend to demonstrate elevated levels of impulsivity and risk-taking behavior, they may also find it difficult to maintain relations with peers and even evoke adverse physical or verbal responses from fellow peers. Consequently, such impulsive behavior may increase children's vulnerability to victimization as bullies can expect to elicit a response from youth with low self-control easier than youth with high self-control and emotional constraint. To illustrate, Unnever and Cornell (2003) analyzed a sample of middle school students and found that students who reported taking medication for attention-deficit/hyperactivity disorder (ADHD) were more likely to be the subject of bullying victimization. As such, self-control (Gottfredson & Hirschi, 1990) provides yet another plausible explanation for the positive association between behavioral problems and bullying victimization.

A complementary explanation to these two commonly offered theories is the biosocial perspective. According to this perspective, a combination of both genetic and environmental effects affects the development of personality characteristics that increase the risk of experiencing bullying victimization and developing antisocial behavior, and symptoms of negative mental health such as depression/anxiety. To understand how this could be the case, consider the type of children most likely to be targeted by bullies. Research commonly indicates that children with introverted personality traits (Egan & Perry, 1998), depression disorder (Arseneault et al., 2006), and externalizing behavioral problems (Barker et al., 2008) are more likely to be bullied by peers compared with children without such personality traits. Importantly, research has also revealed that genetic factors explain a significant amount of variation in these behavioral and mental health outcomes among youth (Hettema, Neale, & Kendler, 2001; Reichborn-Kjennerud et al., 2007; Rhee & Waldman, 2002; Sullivan, Neale, & Kendler, 2000). Moreover, an established line of biosocial research has shown that individual differences in self-control are also explained by a combination of genetic and environmental factors (Beaver, Connolly, Schwartz, Al-Ghamdi, & Kobeisy, 2013; Beaver et al., 2009; Connolly & Beaver, 2014). Taken together, it is possible then that the same genes that increase the risk of bullying victimization also operate to increase the risk of delinquent behavior and internalizing problems such as depression/anxiety. Indeed, one study using cross-sectional data even found that the covariance between bullying victimization and bullying behavior during childhood was solely accounted for by genetic influences (Ball et al., 2008).

Despite the growing amount of research examining the extent to which genetic and environmental factors account for variation in bullying victimization and different domains of maladaptive development, little research has used longitudinal sibling data to evaluate the role genetic factors play in the association between early life bullying victimization and later life

developmental outcomes. To the best of our knowledge, only two recent studies have examined the genetic and environmental influences on chronic bullying victimization over time. One study by Bowes et al. (2013) used data from the Environmental Risk (E-Risk) Longitudinal Twin Study and found that close to half of the correlation (47%) between bullying victimization in primary school and bullying victimization in secondary school was accounted for genetic factors, whereas 41% of the correlation was accounted for by shared environmental factors and the remaining 12% was explained by non-shared environmental factors (plus error). Furthermore, results showed that chronic victims of bullying had significantly higher levels of externalizing and internalizing behavioral problems compared with their non-bullied counterparts. Another study by Boivin et al. (2013) used longitudinal twin data to assess the influence of genetic and environmental effects on persistent peer difficulties during early childhood and found that common genetic factors explained 83% of the stability in peer problems. Although these studies indicate that genetic factors affect patterns of bullying victimization across extended life-course developmental periods, no research has assessed whether common genetic factors that account for variation in repeated bullying victimization during childhood also explain individual differences in delinquent behavior and symptoms of depression/anxiety later in life.

The Current Study

Recent studies using data drawn from the NLSY97 provide some of the most compelling longitudinal evidence on the negative consequences associated with early life repeated bullying victimization. However, as with all studies, there are limitations with the research design and analytic strategy. One of the key areas of concern with this line of research and most social science research is potential confounding. Indeed, Bouffard and Koeppel (2014) acknowledged this limitation by mentioning that previous studies have "largely ignored the role of potential confounding factors in exploring the relationship between victimization and physical and mental health outcomes" (p. 5), while Wong and Schonlau (2013) also commented that "existing research on bullying and victimization uses cross-sectional, quasi-experimental, or descriptive designs and limits analysis to frequency data or bivariate statistics that do not account for confounding among variables" (p. 1186). As such, it is difficult to identify the underlying mechanisms that explain the relation between bullying victimization and maladaptive development when using traditional social science methodologies because they typically confound environmental influences (e.g., bullying) with unmeasured genetic effects (Cleveland, Beekman, & Zheng, 2011).

The current study aims to address this shortcoming in the current literature by using sibling data from the NLSY97 to tease apart the genetic and environmental effects on bullying victimization, delinquency, and negative mental health. To do so, a series of genetically informed bivariate models are used to estimate the shared genetic and environmental effects on the association between early life repeated bullying victimization and subsequent delinquent involvement as well as the association between early life repeated bullying victimization and negative mental health in young adulthood.

Method

Data

The present study used self-report data from the NLSY97. The NLSY97 is a longitudinal data collection effort funded by the Bureau of Labor Statistics designed to examine the life-course transition from adolescence to adulthood among a nationally representative sample of youth. During 1997, the NLSY97 used a household probability sampling design to collect data from 8,984 participants between the ages of 12 and 16. General interviews were conducted using computer-assisted personal interview (CAPI) techniques whereas audio computer-assisted self-interview (ACASI) methods were used when asking respondents sensitive questions on topics such as criminal behavior, victimization, drug use, and sexual behavior. Data used in the present study were collected from 1997 (Wave 1) to 2011 (Wave 15). Retention rates for the NLSY97 during this time period have been above 80%.

During the initial data collection period, the NLSY97 sampled every household resident between the age of 12 and 16. As a result, more than one respondent from 1,862 sampled households was included in the final sample. To accurately identify the nature of the relationship between individual respondents, the NLSY97 asked respondents to select a categorical description that best described their relationship with each household member.[2] The biological sibling categories included identical twin, full-brother, half-brother, full-sister, half-sister, male cousin, and female cousin. In all, 3,690 respondents reported some level of biological relation with one or more NLSY97 respondents.

To identify the sibling subsample nested within the NLSY97, cases were organized by household. After restructuring the data by household identifier, we examined whether respondents reported a biological relation with one or more respondents living within the same household during the 1997 wave. Based on reports of biological relation between siblings within the same home, each sibling was assigned a corresponding genetic coefficient based on the principles of additive genetic theory (Lynch & Walsh, 1998), that

is, monozygotic (MZ) or identical twins were assigned an additive genetic coefficient of $R = 1.00$, because they share 100% of their genetic material; dizygotic (DZ) or non-identical twins and full-sibling pairs were assigned an additive genetic coefficient of $R = .50$ because they share, on average, 50% of their genetic material; and half-sibling pairs were assigned an additive genetic coefficient of $R = .25$ because they share 25% of their genetic material.

To examine the validity of our approach, we conducted a biometric analysis of sex-standardized height between siblings. Behavior genetic research suggests that adult height is largely heritable often accounting for 80% to 90% of variation (Silventoinen et al., 2003). If similar genetic estimates for height are found within the NLSY97 kinship pair data, we can interpret this as support for the validity of the kinship links. Moreover, this method has been used previously by Rodgers and colleagues to validate kinship links for the National Longitudinal Survey of Youth 1979 (NLSY79) and the NLSY79 Child and Young Adult (CNLSY; unpublished manuscript, 2005) sample. Height scores for each respondent were created using the most recent height data from the 2011 wave of the NLSY97 when respondents were between 26 and 32 years of age. Respondent height scores were then standardized by age and sex to create sample norms.

A series of ACE models were then fit to the kinship pair data to estimate the additive genetic, shared environmental, and nonshared environmental (plus error) influences on standardized height by age and sex. Results from the ACE models revealed that over 88% of the variance in male and female height for White/non-Hispanic/non-Black respondents was due to genetic influences, whereas 80% to 86% of the variance in male and female height for Black and Hispanic respondents was attributable to genetic factors (results available on request). The results therefore provide initial support for the NLSY97 kinship links as valid indicators of genetic relatedness.

To eliminate any potential biases, the current study analyzed one sibling pair per household. After selecting one sibling pair per household, full-sibling pairs constituted close to 92% of the sibling sample. To balance the zygosity distribution, a random sample of full-sibling pairs was selected. This procedure resulted in a final analytical sample of $N = 652$ respondents, $n = 326$ sibling pairs, $n = 27$ MZ twin pairs (8.3%), $n = 224$ DZ twin/full-sibling pairs (68.7%), and $n = 75$ half-sibling pairs (23.0%).

Measures

Bullying Victimization

During the initial survey wave, respondents were asked whether they had ever been the victim of repeated bullying before the age of 12. The age of

respondents during this data collection period ranged from 12 to 16 years (*M* age = 14). Although concerns may be raised about the respondent's ability to recall experiences that may have occurred up to 6 or more years ago, survey research suggests that individuals tend to recall traumatic experiences (such as bully victimization) with relative accuracy (Rivers, 2001). In addition, results from a cross-examination of respondents' age and reports of bullying victimization did not reveal any considerable response trends that would suggest the presence of recall problems. Response categories for ever being the victim of repeated bullying were dichotomous (0 = *no*, 1 = *yes*).

Delinquency

NLSY97 participants have been asked several questions at each wave regarding the frequency of their delinquent involvement. Following previous research using the NLSY97, the current study measured delinquency with seven delinquent behaviors (assessed from Wave 2 [1998] to Wave 15 [2011]) that are predicted by repeat bully victimization at Wave 1 (1997; Wong & Schonlau, 2013). Specifically, items that were included asked participants if they had (a) run away from home (assessed only between Waves 2 [1998] and 6 [2002] when participants were 17 or younger at the time of the interview), (b) attacked someone with the intent to seriously hurt them, (c) destroyed property that belonged to someone else on purpose, (d) stole something less than US$50, (e) stole something more than US$50, (f) committed other property offenses, and (g) ever sold drugs. If the respondent reported engaging in one of the delinquent behaviors between Waves 2 and 15, the respondent was given a value of 1 for that item. In contrast, if the respondent did not report engaging in any of the delinquent behaviors between Waves 2 and 15, the respondent was given a value of 0 for that item. All cumulative items were then summed to create a variety index of delinquent involvement (0-7), which demonstrated good internal reliability ($\alpha = .79$). Due to the positive skew of the data, the index was transformed into a categorical variable (0 = *zero delinquent acts*, "no delinquency," 47.26%; 1 = *one to two delinquent acts*, "mild delinquency," 31.00%; 2 = *three to four delinquent acts*, "moderate delinquency," 12.71%; and 3 = *five to seven delinquent acts*, "frequent delinquency," 9.04%).

Symptoms of Depression/Anxiety

Symptoms of depression/anxiety were assessed by the five-item short-version of the Mental Health Inventory (MHI-5) that was administered every 2 years beginning at Wave 4 (2000, Wave 6 [2002], Wave 8 [2004], Wave 10 [2006], Wave 12 [2008], Wave 14 [2010]). The MHI-5 includes questions

referring to depression and anxiety disorder and has been validated by previous experimental interviews as a reliable measure for depression (Berwick et al., 1991; Rumpf et al., 2001). To ask respondents how often in the past month they have felt nervous, calm or peaceful, down or blue, happy, or depressed, 4-point Likert-type scales were used: 1 = *all of the time*, 2 = *most of the time*, 3 = *some of the time*, and 4 = *none of the time*. All five items demonstrated a good internal consistency across waves (2000, $\alpha = .76$; 2002, $\alpha = .76$; 2004, $\alpha = .78$; 2006, $\alpha = .77$; 2008, $\alpha = .79$; 2010, $\alpha = .78$) and after reverse coding three items, confirmatory factor analyses confirmed that a one-factor solution fit the data best at each wave. Each MHI-5 score was then summed together to create a longitudinal measure of negative mental health. Larger values represented a higher frequency of negative feelings (33-101). Due to common convergence issues when using structural equation modeling to decompose covariance between categorical and continuous latent variables, we transformed the continuous measure into a four-quartile categorical variable (1 = *0%-25%*, 2 = *26%-50%*, 3 = *51%-75%*, and 4 = *76%-100%*).

Analysis Plan

The analysis for the present study proceeded in a series of linked steps. First, Stata 12.0, Special Edition (Stata, 1985-2011) was used to examine the frequency of bullying victimization and maladaptive developmental outcomes across sibling pairs. As the sample contained information on respondents from the same household, Huber–White standard errors were used to partially correct for non-independence between observations. Second, tetrachoric and polychoric cross-sibling correlations were calculated to assess within-sibling relationships for bullying victimization, delinquency, and negative mental health. This was done to assess the concordance of each trait within sibling pairs and is a commonly used behavior genetic technique to estimate the relative magnitude of genetic and environmental effects on a given phenotype (Neale & Cardon, 1992). If within-sibling concordance for MZ twins ($R = 1.00$) is higher than within-sibling concordance for DZ twins/full-siblings ($R = 0.50$)—given that certain assumptions are satisfied[3]—this can be interpreted as initial evidence for some degree of genetic influence on the phenotype under investigation. Recent research has found that the underlying assumptions of this method are often satisfied (Barnes et al., 2014).

Third, univariate ACE liability-threshold models were used to estimate the extent to which additive genetic, shared environmental, and nonshared environmental factors accounted for individual differences in liability for each

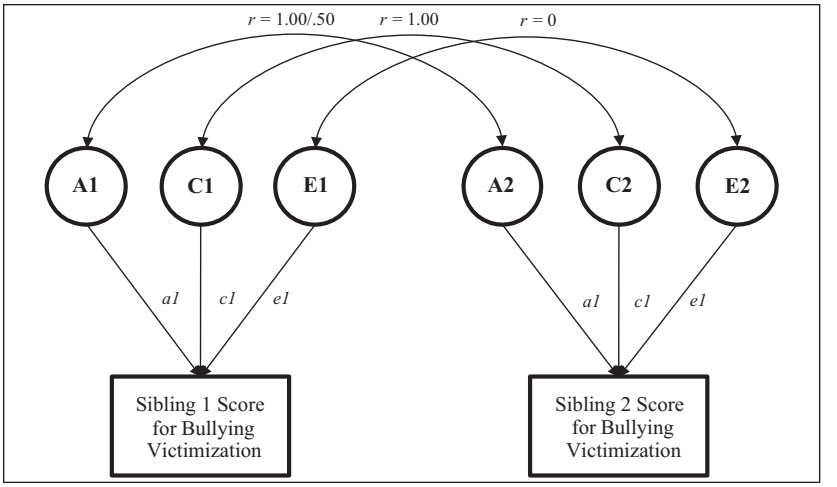

Figure 1. Univariate ACE model for bullying victimization.
Note. *r* values from A1 to A2 represent genetic relatedness model constraints, whereas the *r* value from C1 to C2 represents the shared environment model constraint. Path coefficients (*a1, c1, e1*) from each latent A, C, and E parameter leading to the observed variable (i.e., bullying victimization) will provide information on the amount of variance accounted for by additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences and measurement error (E).

trait. In the univariate model, phenotypic variation between siblings is decomposed into three latent variance components: (a) additive genetic variance (symbolized as A), (b) shared environmental variance (symbolized as C), and (c) nonshared environmental variance (symbolized as E). Shared environmental factors are influences that operate to make siblings similar to one another (e.g., similar parent–child relationships, similar peer groups, similar school experiences), whereas nonshared environmental factors are influences that operate to make siblings different from one another (e.g., different parent–child relationships, different peer groups, different school experiences). Importantly, measurement error is captured by the nonshared environmental component (E) in each model. The relative size of each latent variance component estimate in the model is computed by comparing observed cross-sibling correlations with predicted cross-sibling correlations generated by the model. For a graphical representation of an ACE model decomposing variance in bullying victimization, see Figure 1.

The final step in the analyses involved fitting a series of bivariate liability-threshold models to partition the covariance between bullying victimization, delinquency, and symptoms of depression/anxiety. In contrast to

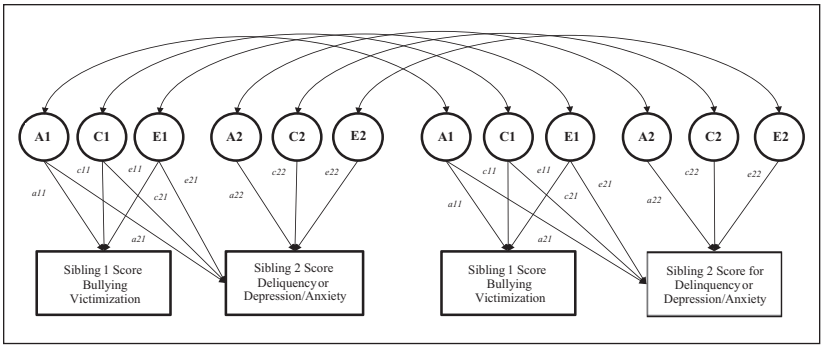

Figure 2. Bivariate ACE model for bullying victimization, delinquency, and depression/anxiety.

Note. Path correlations from A1 to A2 are set to 1.00, .50, or .25 based on genetic relatedness. Path correlations from C1 to C2 are set to 1.00 and path correlations from E1 to E2 are set to 0. Coefficients for *a11, c11, e11, a21, c21*, and *e21* are used to estimate the amount of covariance that is accounted for by additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences and measurement error (E).

univariate biometric models, bivariate biometric models are able to partition the covariance between two phenotypes into A, C, and E latent variance components. Bivariate liability-threshold estimates therefore represent the proportion of explained covariance in liability between two phenotypic outcomes that is explained by additive genetic influences, shared environmental influences, and nonshared environmental influences (plus error). Based on the categorical nature of the variables under examination, liability-threshold models were used instead of traditional ACE and Cholesky decomposition models that are appropriate for continuous data (Prescott, 2004; Smith, 1974). Figure 2 shows a graphical representation of a bivariate ACE model decomposing the covariance between bullying victimization and delinquency or depression/anxiety.

All liability-threshold univariate and bivariate biometric models presented in the current study were estimated using M*plus*, version 7.1 (Muthén & Muthén, 1998-2012) with full information maximum likelihood (FIML) to deal with missing data. As recommended, model fit was assessed using an adjusted chi-square difference test because differences in robust chi-square statistics do not form a normal chi-square distribution (Santorra, 2000). The comparative fit index (CFI) and the root mean square error of approximation (RMSEA) were also used to assess more parsimonious nested models alongside baseline models with the following acceptable

Table 1. Frequency of Bullying Victimization.

	Total Sample	MZ Twins	DZ Twins/Full-Siblings	Half-Siblings
	Yes %	Yes %	Yes %	Yes %
Bullying victimization	17.99	17.86	17.30	25.69

Note. MZ = monozygotic; DZ = dizygotic.

Table 2. Frequency of Delinquency and Symptoms of Depression/Anxiety.

Delinquency	Never %	Mild %	Moderate %	Frequent %
Total sample	47.26	31.00	12.71	9.04
MZ twins	62.50	23.21	1.79	12.50
DZ twins/full-siblings	47.29	31.38	12.89	8.45
Half-siblings	41.22	29.73	14.86	14.19
Depression/Anxiety	0%-25%	26%-50%	51%-75%	76%-100%
Total sample	25.93	25.67	25.24	23.17
MZ twins	35.71	14.29	21.43	28.57
DZ twins/full-siblings	25.89	27.24	25.51	21.37
Half-siblings	23.40	11.70	23.40	41.49

Note. MZ = monozygotic; DZ = dizygotic.

model fit cutoff points: CFI > .90 (satisfactory fit) and RMSEA < .05 (good fit; Hu & Bentler, 1998, 1999).

Results

The analysis began by examining the frequency of repeated bullying victimization, delinquency, and symptoms of depression/anxiety within the total sample and between sibling pairs. As presented in Table 1, around 18% of respondents reported being the victim of repeated bullying before age 12. This frequency estimate is similar to other studies examining repeated bullying victimization (Fekkes, Pijpers, Fredriks, Vogels, & Verloove-Vanhorick, 2006; Jaffee, Caspi, Moffitt, & Taylor, 2004; Rigby, 1998). With respect to sibling pair differences in repeated bullying victimization, results indicate that half-siblings reported being bullied more than MZ twins and DZ twins/full-siblings. Turning to delinquency, Table 2 reveals that half-siblings reported committing more delinquent acts over time compared with MZ twin

Table 3. Cross-Trait Sibling Correlations Between Bullying Victimization, Delinquency, and Symptoms of Depression/Anxiety.

	MZ Twins	DZ Twins/Full-Siblings	Half-Siblings
Bullying victimization	.80**	.42***	.24*
Delinquency	.62**	.31***	.23*
Depression/anxiety	.41**	.25***	.14*

Note. MZ = monozygotic; DZ = dizygotic.
*Significant at $p \leq .10$. **Significant at $p \leq .05$. ***Significant at $p \leq .01$.

pairs and DZ twins/full-sibling pairs. Half-siblings also scored higher on symptoms of depression/anxiety, followed again by MZ twin pairs and DZ twins/full-sibling pairs.

Table 3 contains a series of correlations between sibling pairs for bullying victimization, delinquency, and symptoms of depression/anxiety. Keep in mind that these cross-trait correlation estimates serve to examine the relative magnitude of genetic influences on each trait where higher correlations between siblings who share more genetic material can be interpreted as partial evidence for the presence of genetic effects. As can be seen, MZ twin cross-trait correlation coefficients were higher than other sibling pair correlation coefficients for repeated bullying victimization, delinquent activity, and symptoms of depression/anxiety ($p < .05$). Concordance rates for DZ twins/full-siblings were significant and higher for all three measures under investigation compared with half-sibling pairs as well. Taken together, cross-trait correlational evidence suggests that genetic factors may contribute to liability for bullying victimization, delinquency, and symptoms of depression/anxiety.

Next, univariate liability-threshold models were fitted to the data. Table 4 presents the standardized parameter estimates for each best fitting univariate ACE model. As can be seen, shared environment influences (C) did not account for any observed variation in repeat bullying victimization in the baseline ACE model. As such, the shared environmental component (C) was constrained that modestly improved the overall model fit. According to the best fitting AE model, 70% of the variance in liability to repeat bullying victimization was explained by additive genetic influences, whereas nonshared environmental influences (plus error) accounted for the remaining 30% of the variance in liability. These results align with other standardized additive genetic estimates for bullying victimization (Ball et al., 2008). With respect to delinquency, genetic factors accounted for 56% of the variance in delinquent behavior and 65% of the variance in depression/anxiety. Nonshared environmental influences (plus error) accounted for the remaining 44% of the variance in delinquent

Table 4. Univariate ACE Estimates for Bullying Victimization, Delinquency, and Symptoms of Depression/Anxiety.

	A	C	E	$\Delta\chi^2$	Δdf	CFI	RMSEA
Bullying victimization							
ACE	.70***	.00	.30***	—	—	.94	.04
	[0.43, 0.97]	[−0.002, 0.002]	[0.02, 0.56]				
AE	**.70*** **	**.00**	**.30*** **	**.03**	**1**	**.94**	**.04**
	[0.43, 0.97]	**[0.00, 0.00]**	**[0.02, 0.56]**				
CE	.00	.46***	.54***	32.78***	1	.72	.08
	[0.00, 0.00]	[0.36, 0.58]	[0.43, 0.64]				
E	.00	.00	1.00***	183.80***	2	.23	.14
	[0.00, 0.00]	[0.00, 0.00]	[1.00, 1.00]				
Delinquency							
ACE	.39**	.02*	.59***	—	—	.92	.04
	[0.10, 0.50]	[0.00, 0.24]	[0.50, 0.70]				
AE	**.56*** **	**.00**	**.44*** **	**.09**	**1**	**.95**	**.03**
	[0.38, 0.72]	**[0.00, 0.00]**	**[0.28, 0.62]**				
CE	.00	.35***	.65***	17.12***	1	.73	.08
	[0.00, 0.00]	[0.20, 0.50]	[0.50, 0.80]				
E	.00	.00	1.00***	99.02***	2	.31	.12
	[0.00, 0.00]	[0.00, 0.00]	[1.00, 1.00]				
Depression/anxiety							
ACE	.43**	.01*	.56**	—	—	.92	.05
	[0.25, 0.54]	[0.00, 0.14]	[0.46, 0.67]				
AE	**.65*** **	**.00**	**.35*** **	**.06**	**1**	**.94**	**.05**
	[0.50, 0.80]	**[0.00, 0.00]**	**[0.20, 0.50]**				
CE	.00	.29**	.71***	2.02*	1	.89	.07
	[0.00, 0.00]	[0.13, 0.45]	[0.55, 0.87]				
E	.00	.00	1.00***	85.23***	2	.41	.12
	[0.00, 0.00]	[0.00, 0.00]	[1.00, 1.00]				

Note: The best fitting ACE model is boldfaced. Results are standardized parameter estimates. 95% confidence intervals are presented in brackets. CFI = comparative fit index; RMSEA = root mean square error of approximation.
*Parameter significant at $p \leq .10$. **Parameter significant at $p \leq .05$. ***Parameter significant at $p \leq .01$.

involvement and 35% of the variance in depression/anxiety. In sum, univariate estimates suggest that genetic and nonshared environmental influences largely account for individual differences in repeat bullying victimization, delinquent activity, and symptoms of depression/anxiety.

Table 5 presents standardized parameter estimates for two bivariate liability-threshold models examining the covariance in liability among bullying victimization, delinquency, and depression/anxiety. As constraining the shared environmental parameter (C) improved the overall model fit in each

Table 5. Bivariate Estimates for Bullying Victimization, Delinquency, and Symptoms of Depression/Anxiety.

	A	C	E	$\Delta\chi^2$	Δdf	CFI	RMSEA
Bullying victimization/delinquency							
AE	**.61*****	**.00**	**.39*****	**-**	**-**	**.93**	**.05**
	[0.43, 0.97]	**[0.00, 0.00]**	**[0.02, 0.56]**				
E	.00	.00	1.00***	113.28***	1	.63	.12
	[0.00, 0.00]	[0.00, 0.00]	[1.00, 1.00]				
Bullying victimization/depression/anxiety							
AE	**.44*****	**.00**	**.56*****	-	—	**.95**	**.05**
	[0.27, 0.64]	**[0.00, 0.00]**	**[0.36, 0.73]**				
E	.00	.00	1.00***	121.43***	1	.32	.14
	[0.00, 0.00]	[0.00, 0.00]	[1.00, 1.00]				

Note. The best fitting ACE model is boldfaced. Results are standardized parameter estimates. 95% confidence intervals are presented in brackets. CFI = comparative fit index; RMSEA = root mean square error of approximation.
*Parameter significant at $p \leq .10$. **Parameter significant at $p \leq .05$. ***Parameter significant at $p \leq .01$.

univariate model (i.e., AE models), the C parameter was constrained in all bivariate models. As can be seen, AE models (compared with E models) fit the data best based on chi-square difference statistics, CFI values, and RMSEA values. The bivariate model of bullying victimization and delinquency was an adequate fit to the data (CFI = .93, RMSEA = .05). Standardized bivariate estimates suggested that additive genetic influences accounted for 61% of the covariance between repeated bullying victimization and delinquent activity, whereas nonshared environmental influences accounted for 39% of the covariance. The best fitting AE model of bullying victimization and symptoms of depression/anxiety fit the data well (CFI = .95, RMSEA = .05). As can be seen, additive genetic factors explained 44% of the covariance between bullying victimization and symptoms of depression/anxiety. The nonshared environment (plus error) accounted for the remaining 76% of the covariance in liability.

Discussion

Bullying victimization is a widespread phenomenon commonly experienced by children and adolescents. Although bullying has often been regarded as a normal experience, a growing body of research has revealed that bullying victimization increases the risk of developing several behavioral and

emotional problems (Arseneault et al., 2010). Given the potential public health implications associated with repeated bullying victimization among youth, an emerging line of research cutting across a range of disciplines has begun to focus on identifying shared causes and correlates of bullying victimization and maladaptive behavioral development (Arseneault et al., 2006; Bouffard & Koeppel, 2014; Kim et al., 2006). Less research, however, has used behavioral genetic methods to assess the extent to which genetic and environmental factors influence this association over the life course. The current study was designed to fill this void in the literature. Overall, three key findings emerged from our analyses.

First, parameter estimates from a series of univariate liability-threshold models revealed that genetic factors accounted for a considerable amount of variance in liability for bullying victimization, delinquent involvement, and symptoms of depression/anxiety. Specifically, results suggested that additive genetic influences accounted for 70% of the liability for repeated bullying victimization before age 12, 56% of the variance in delinquent activity over the life course, and 65% of the variance in symptoms of depression/anxiety in young adulthood. Bivariate parameter estimates indicated that additive genetic factors explained 61% of the covariance between victimization and delinquency, whereas 44% of the covariance between victimization and symptoms of depression/anxiety was accounted for by additive genetic influences. Although previous behavioral genetic research has found that genetic factors play a role in bullying victimization and bullying behavior (Ball et al., 2008), evidence from the current study offers new insights into the overlapping genetic liability for personality characteristics that may function to increase a child's risk of victimization, delinquent behavior, and subsequent psychological problems. These findings offer evidence for genotype–environment correlation (*r*GE) where the same genetic factors that explain individual differences in delinquency and symptoms of depression/anxiety may also explain variation in risk of bullying victimization (Scarr & McCartney, 1983). Specifically, individuals with genetic propensities for delinquent behavior may actively self-select into antisocial peer groups that increase their exposure to bullies and frequent victimization (i.e., active genotype–environment correlation). However, genetically influenced depression/anxiety disorder may also evoke bullying efforts from peers because bullies perceive individuals with such symptoms as "easy targets" (i.e., evocative gene–environment correlation). Given that bullying victimization was measured before delinquency and symptoms of depression/anxiety in the present study, we can only speculate as to whether children exhibited early signs of both behaviors before being bullied. However, previous research has shown that victims of bullying tend to exhibit higher levels of both externalizing and

internalizing problems compared to non-victims (Barker et al., 2008; Bowes et al., 2013) both types of behaviors have been shown to demonstrate relative stability from childhood to adulthood (Hofstra, Van der Ende, & Verhulst, 2000; Loeber & Hay, 1997). Taken together, results from the present study contribute to an emerging line of research that suggests that genetic vulnerability for bullying victimization may be closely related to genetic vulnerability for delinquent involvement and negative psychological development (Benjet, Thompson, & Gotlib, 2010; Sugden et al., 2010).

Second, nonshared environmental influences (plus error) accounted for the remaining variance in early life bullying victimization, delinquency, and negative mental health. As nonshared environmental influences are experiences that are unique to each sibling rather than experiences shared between each sibling, these estimates imply that perhaps different peer groups, family environments, or neighborhood experiences affect individual liability for bullying victimization and later life maladjustment. Some research has revealed that family-level influences such as differential maternal treatment can uniquely increase the risk of externalizing behavioral problems among children via the nonshared environment (Caspi et al., 2004). However, further research is needed to identify important nonshared environmental influences that could inform prevention efforts aimed at decreasing the likelihood of bullying within school, household, or neighborhood settings.

Third, univariate parameter estimates from the best fitting models suggested that the shared environment was not a significant contributor to individual differences in bullying victimization, delinquent behavior, or negative mental health. Based on these results, liability risk of bullying victimization and other maladaptive behaviors may be due to genetically influenced traits that correlate with unique nonshared environmental experiences (e.g., *r*GE). The absence of the shared environment in the current study underscores the importance of taking into consideration individual personality differences and how these differences may increase or decrease a child's risk of being bullied across different environmental contexts.

Limitations to the current study must be addressed for future research purposes. First, the current study relied on a single measure of bully victimization that asked respondents to report on their victimization experiences nearly 6 years ago. This type of recall measure is more prone to error because respondents may confuse the timing of events, offer a biased account based on emotions related to the event, or forget altogether. As a result, future research should combine respondent self-reports with parent and teacher reports to improve measurement validity and reliability. In doing so, the effects of measurement error may also be reduced.

Second, respondents were not given a definition of bullying victimization before being asked whether they had been the victim of repeated bullying before age 12. Given this limitation, response rates for bullying victimization are based on respondents' interpretation of bullying behavior that could include both physical and verbal forms of victimization. Therefore, responses rates for being bullied most likely reflect whether the respondents remembered being subjected to bullying as they defined it.

Third, sibling data used in the current study were generated by linking siblings based on self-reports. Although the sibling links were validated by biometric model fitting and sensitivity analyses, self-reports provide a less than perfect estimate of genetic relatedness among siblings. Consequently, we encourage researchers to use the sibling data from the NLSY97 for future research purposes and offer additional validation to the sibling links.

Fourth, behavior genetic designs used in the current study were only capable of decomposing variance into additive genetic, shared environmental, and nonshared environmental latent components. As such, parameter estimates do not offer any insights into the specific genes or nonshared environmental influences that may contribute to the reported associations. An emerging line of molecular genetic research has begun to assess the extent to which a specific serotonin transporter gene (5-HTTLPR) moderates the association between bully victimization and the development of emotional problems (Benjet et al., 2010; Sugden et al., 2010). However, this line of research still remains in its infancy. Therefore, future research examining bullying victimization and maladaptive development should apply genetically informative research designs capable of identifying specific genetic risk factors for victimization and the emergence of antisocial behaviors during childhood (Beaver & Connolly, 2013).

Results from the current study suggest that genetic influences play a shared role in liability for chronic bullying victimization, delinquent involvement, and negative psychological health. Although further research on the precise mechanisms that explain this causal pathway between repeated bullying victimization, externalizing behaviors, and internalizing problems is needed, evidence from our study suggests the need for an individualized approach in anti-bullying efforts. Such an approach may help treat individual differences in vulnerability for victimization by addressing signs of internalizing and externalizing problem behaviors early in life before they are subjected to peer victimization that could exacerbate behavioral problems. Early intervention efforts may help reduce the chances of chronic victimization in unique environmental contexts over the life course as well including school classrooms during childhood, peer groups in adolescence, and professional work environments during adulthood. As such, evidence from contemporary

research on bullying victimization (Boivin et al., 2013; Bowes et al., 2013) and the current study highlight the need to intervene early in life with approaches that focus on both the individual and surrounding peer contexts. Doing so can proactively break the cycle of victimization that has been linked to the development of negative behavioral and psychological health. Moreover, it is important to recall that genetic effects on behavioral development operate in a probabilistic and not a deterministic fashion. Thus, biosocial evidence from the current study implies that early bullying intervention efforts may help reduce the likelihood that youth with genetically influenced traits will experience further maladjustment later in life, not that children should be blamed for their own victimization experiences. Indeed, further understanding how genetic *and* environmental factors work together to explain variation in exposure to bullying victimization and the development of psychopathology will only help create a stronger knowledge base that can create more targeted intervention/prevention programs aimed at reducing the risk of repeated bullying victimization.

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Notes

1. The range of factors controlled for in their analysis included age, race, mean household income, marriage status, chronic condition status, delinquency, and substance use.
2. During the 1997 survey wave, National Longitudinal Survey of Youth 1997 (NLSY97) respondents were also asked to identify non-biological household members. Given the scope of the current study, we focused only on identifying biologically related siblings.
3. There are two major assumptions of behavioral genetic analyses: assortative mating and the equal environments assumption (for more, see Plomin, DeFries, Knopik, & Neiderhiser, 2013). Assortative mating refers to non-random mating between two individuals with similar behavioral characteristics that, if violated, could bias heritability estimates. The equal environments assumption of behavioral genetics assumes that phenotypic similarly between siblings is the result of equal environmental experiences within the same family. If identical twins experience more similar environments compared to fraternal twins, the equal environments assumption would be violated and consequently inflate heritability

estimates. However, previous research has found that the equal environments assumption appears to not be violated for most traits (Derks, Dolan, & Boomsma, 2006) and if violated, results in a minimal effect on hertiability estimates (Barnes et al., 2014).

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