# ADDITIVE GENETIC AND ENVIRONMENTAL CONTRIBUTIONS TO THE LONGITUDINAL ASSOCIATION BETWEEN VIOLENT VICTIMIZATION AND DEPRESSION

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## Abstract

The present study analyzed unique and common additive genetic and environmental influences on self-reported violent victimization and major depression across 8 years of life. Young adults ($M_{age}$ = 20.14, SD = 3.94), including 473 full-sibling pairs and 209 half-sibling pairs (N = 1,364) from the Child and Young Adult Sample of the National Longitudinal Survey of Youth were analyzed to examine the association between violent victimization and depression from late adolescence to young adulthood, estimate how much additive genetic and environmental factors account for variation in liability for single and repeat violent victimization and major depression, and compare the magnitude of genetic and environmental influences on the covariance in liability between single and repeated victimization and major depression. Cross–lagged models revealed that while victimization was associated with an increased risk for depression during late adolescence, major depression was more strongly and consistently associated with increased risk for future victimization across young adulthood. Biometric models revealed that 20% of the association between single victimization and major depression was accounted for by common additive genetic influences, while 30% of the association between repeat victimization and major depression was accounted for by common additive genetic influences. Results and implications are discussed.


**Keywords:** Major Depression, Victimization, Repeat Victimization, Behavioral Genetics

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In 2015, it was estimated that over 2.7 million U.S. residents age 12 and older experienced a violent victimization (Truman & Morgan, 2016). Based on annual health care expenditures and lost productivity estimates, the economic toll of intimate partner violence against women alone (i.e., rape, physical assault, and murder) is more than 8 billion dollars (Max, Rice, Finkelstein, Bardwell, & Leadbetter, 2004). It is clear that violent victimization is a serious public health concern. At the same time, national reports show that anxiety and depressive disorders are the most common mental health illnesses in the U.S. and affect close to 40 million U.S. adults age 18 and older every year (Kessler, Chiu, Demler, & Walters, 2005). Research suggests that the economic burden of individuals with major depressive disorder is between 173 and 210.5 billion dollars (Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015). Taken together, there is little doubt that violent victimization and major depression take a large toll on individual victims, their families, and society at large. However, research examining the underlying mechanisms that explain the link between violent victimization and major depression across the life course is scarce.

A substantial body of literature has documented a positive association between victimization and major depressive disorder (e.g. Barchia & Bussey, 2010; Hawker & Boulton, 2000; Kaltiala-Heino, Rimpelä, Marttunen, Rimpelä, & Rantanen, 1999; Ranta, Kaltiala-Heino, Pelkonen, & Marttunen, 2009; Slee, 1995). Both victimization by peers (i.e. bullying; Connolly & Beaver, 2016; Hawker & Boulton, 2000; Kaltiala-Heino et al., 1999) and violent victimization (Boney-McCoy & Finkelhor, 1995; Kilpatrick et al., 2003) have been associated with increased symptoms of anxiety and depression. Based on this growing body of evidence, the standard

assumption regarding the phenotypic association between victimization and depression is that the relationship is largely causal, with victimization directly impacting the subsequent development of major depression (Barchia & Bussey, 2010; Boney-McCoy & Finkelhor, 1995; Hawker & Boulton, 2000; Kilpatrick et al., 2003). Indeed, it seems reasonable to suggest that being victimized, especially violently, could possibly result in the emergence of depressive symptoms in some individuals. However, other studies show that not everyone develops major depression following a victimization (e.g., DeMaris & Kaukinen, 2005; Vaske, Makarios, Boisvert, Beaver, & Wright, 2009), and very few individuals experience more than one violent victimization in their lifetime (Hope & Trickett, 2008).

As such, an important point to consider when evaluating individual differences in the longitudinal association between violent victimization and major depression concerns the possibility of genetic influences (Barnes et al., 2014). It is now well established that variation across virtually all human phenotypes is at least partly the result of genetic differences in the population (Polderman et al., 2015). Neither victimization nor depression are exempted from this pattern of results, as bodies of research have revealed that both traits are partly heritable. The moderate heritability of depression has been firmly established by a long line of research (Johnson, McGue, Gaist, Vaupel, & Christensen, 2002; Kendler, Gatz, Gardner, & Pedersen, 2006; Sullivan, Neale, & Kendler, 2000). Although somewhat more so in men, depression is heritable in both sexes and is heritable across the lifespan (Kendler, Gatz, Gardner, & Pedersen, 2006). Furthermore, depression has been found to be heritable in both community and clinical populations (Sullivan, Neale, & Kendler, 2000).

More recently, an emerging line of research has found that being victimized is also partly heritable (Beaver, Boutwell, Barnes, & Cooper, 2009; Boutwell et al., 2017; Shakoor et al.,

2015). DiLalla and John (2014) found a moderate heritability for "receiving aggression" in 5-year-olds. Shakoor and colleagues (2015) report a modest heritability estimate for bullying victimization in late childhood, and importantly for the current study, report a shared genetic risk for both victimization and paranoid symptoms. Violent victimization has also been found to be heritable in adults, with an even higher heritability estimate for repeat victimization (Beaver, Boutwell, Barnes, & Cooper, 2009).

Given the evidence pertaining to the heritability of both traits, it remains possible that at least some of the phenotypic covariance for the traits might be partly accounted for by common genetic influences (Barnes, Boutwell, Beaver, Gibson, & Wright, 2014). Put another way, a genetic correlation may exist between violent victimization and depression. At least some support for this hypothesis can be found in prior research revealing a shared genetic vulnerability for victimization and other psychopathological outcomes (e.g., paranoid symptoms; Shakoor et al., 2015). Importantly, it seems that psychopathology, in general, is often comorbid with other deleterious phenotypes (Caspi et al., 2014) owing to a shared genetic architecture (Pettersson, Larsson, & Lichtenstein, 2016). Despite a rapidly accumulating body of evidence in this area of psychopathological research, less focus has been directed at examining the longitudinal association between major depressive symptomology and the experience of violent criminal victimization. As a result, the current study seeks to address this gap in the current literature by examining the relationship between violent victimization and major depression over time in a longitudinal study of sibling pairs.

## CURRENT STUDY

The current study analyzes longitudinal sibling data to examine the direction of the association between violent victimization and major depression as well as differences in additive

genetic and environmental influences on the association between single and repeat victimization and major depression over time. Based on previous research, we hypothesize that violent victimization will be more strongly associated with increases in risk for major depression than major depression with increases in risk for violent victimization. Support for this hypothesis would align with the state dependence hypothesis of victimization, which holds that psychopathology, such as major depressive symptomology, develops primarily because of traumatic social life experiences. Evidence in opposition to this hypothesis whereby major depression is more strongly associated with subsequent violent victimization would offer support for the population heterogeneity hypothesis of victimization, which argues that stable dispositional characteristics, such as major depression, makes some individuals more vulnerable or at risk of experiencing a violent victimization. With respect to the magnitude of common additive genetic and environmental influences on violent victimization and major depression, we hypothesize that variation in liability for repeat victimization will account for more genetic variance in major depression than single victimization since selective gene-environment processes are more likely play a role.

## METHODS

### Data

Data analyzed in the current study were drawn from the Child and Young Adult sample of the National Longitudinal Survey of Youth (CNLSY). The CNLSY consists of a sample of youth born to a nationally representative sample of females from the National Longitudinal Survey of Youth 1979 (NLSY79). All biological children born to women from the NLSY79 have been assessed biennially from 1986 to 2012 (Chase-Lansdale, Mott, Brooks-Gunn, & Phillips, 1991). When children reached the age of 15, they were administered the Youth Adult

(YA) self-report survey at each survey wave. The YA survey allowed children to respond to several different age-appropriate sensitive questions in a private self-reporting format. Thus, respondents were asked to report on topics including, delinquent behavior, family relationships, personal attitudes, and violent victimizations. Retention rates for children from the CNLSY from 1986 to 2012 have been above 70%.

Since the 2012 survey wave, nearly 5,000 females from the NLSY79 have given birth to over 11,500 children. Kinship links have been established by Rodgers and colleagues (2016) using explicit indicators of sibling status (e.g., twin sibling, full-sibling, half-sibling, or adopted sibling) based on questionnaires first administered during the 2006 survey wave. Since 2006, 16,083 sibling pairs (100% of possible pairs) from the CNLSY have been identified using the kinship links (Hadd & Rodgers, 2017). Based on the richness of these data, several researchers have used the kinship links to conduct behavioral genetic and sibling comparison analyses on a wide range of topics including antisocial behavior (Van Hulle et al., 2009), delinquency (Connolly & Beaver, 2014; Harden, Quinn, & Tucker-Drob, 2012), intelligence (Rodgers, Rowe, May, 1994), maternal smoking (D'Onofrio et al., 2008), maternal nutrition (Connolly & Beaver, 2015), and violent victimization (Boutwell et al., 2017) (see Rodgers et al., 2016, for background and summary). Researchers interested in taking advantage of the CNLSY kinship pairs can find documentation and code for the links at http://liveoak.github.io/NlsyLinks/.

The current study takes advantage of the kinship links and uses data on respondents who had at least one full- or half-sibling with complete data on violent victimization and major depression. Only one sibling pair per household was included in the sample. The analytic sample was restricted to full- and half-sibling pairs because there were too few identical twin

pairs ($n$ = 7) to include in the analysis. The final sample therefore included $n$ = 473 full-sibling pairs and $n$ = 209 half-sibling pairs ($N$ = 1,364 siblings).

**Measures**

*Major Depression*. Major depression was assessed by the seven-item Center for Epidemiologic Studies Depression Scale (CESD-D) short form (CES-D-SF). Respondents were asked during the 2004, 2006, 2008, 2010, and 2012 survey wave to report how often in the past week they had suffered from each of the following symptoms: (1) poor appetite, (2) trouble focusing, (3) feeling depressed, (4) everything taking extra effort, (5) restless sleep, (6) saddened, and (7) unable to get "going". Response categories ranged from 0 (*rarely, none of the time, 1 day*) to 4 (*most, all the time, 5-7 days*). Responses were summed to create scales of depression at each survey wave. Scales demonstrated adequate internal consistency across time with Cronbach's alphas ranging from .68 to .71 and all items loading highly on a single common factor at each survey wave. Previous research analyzing data from the NLSY79 has shown that the CES-D-SF demonstrates better psychometric properties compared to the CES-D with higher internal consistency and better unidimensionality (Levine, 2013). Moreover, evidence from prior psychometric research indicates that a CES-D-SF cut-off score of 8 or more has acceptable specificity with the standard CES-D cutoff score of 16 or more (Levine, 2013), which would satisfy the DSM-V criteria for major depressive disorder (American Psychiatric Association, 2013). As such, to assess risk for major depression at each survey wave, CESD-D-SF scales were dichotomized such that 0 = score of 7 or less and 1 = score of 8 or more. A cumulative scale of major depression across survey was also created by adding together all dichotomized measures to examine the frequency of major depression over time. Table 1 presents the percentage of respondents classified as suffering from major depression at each survey wave.

***Insert Table 1 Around Here***

*Violent Victimization.* Violent victimization was first assessed during the 2004 survey wave and then again during the 2006, 2008, 2010, and 2012 survey waves by asking respondents if they had been the victim of a violent crime (e.g., physical or sexual assault, robbery or arson) since the date of their last interview. To reduce social desirability bias and encourage accurate reporting, respondents were asked to report whether they had been the victim of violent crime in the YA survey, which is administered in a private self-reporting format. Response categories were 0 = no and 1 = yes. To assess differences in major depression over time between single and repeat victims, two additional measures were created. The first measure was created to identify respondents who had only experienced one victimization from 2004 to 2012. As such, the single victimization measure was coded such that 0 = no victimization and 1 = one victimization. The second measure created was designed to identify respondents who had experienced more than one violent victimization from 2004 to 2012. Therefore, the repeat victimization measure was coded such that 0 = no victimization or 1 victimization and 1 = two or more victimizations. Table 1 reports the percentage of respondents who experienced a single victimization and repeat victimizations. Overall, 8.04% of the sample reported one violent victimization, while 2.31% reported two or more violent victimizations.

*Statistical Controls.* Age, race, and sex were controlled for in all analyses. Age was measured by the age of respondents (in years) during the 2004 survey wave. Race was measured by a dichotomous variable where 0 = Non-Black and Non-Hispanic and 1 = Black or Hispanic. Sex was measured by a dichotomous variable where 0 = female and 1 = male. Table 1 shows that the average age of respondents was 20 years old, 46% were Black or Hispanic, and 51% were male.

## PLAN OF ANALYSIS

The plan of analysis was carried out in three interrelated steps. First, an autoregressive cross-lagged model was fit to the data to examine whether victimization at earlier waves predicted increases in probability of major depression at later waves while controlling for stability in both measures and statistical controls. Autoregressive cross-lagged models offer a unique advantage over other longitudinal modeling approaches, such as latent growth curve modeling and group-based trajectory modeling, in that they allow for the estimation of the directionality of change between two variables over time, while maintaining temporal order (Selig & Little, 2012). While latent growth curve modeling and group-based trajectory modeling provide evidence of correlated within-individual and group change over time, neither approach can provide estimates of the direction of the association (i.e., whether victimization is associated with risk for major depression or major depression is associated with risk for victimization). All autoregressive cross-lagged models were estimated in M*plus* 7.4 (Muthén & Muthén, 2012) using a weighted least squares variance estimator (WLSMV) which is appropriate for latent variables with binary or ordinal properties. Model fit was assessed using the comparative fit index (CFI), Tucker-Lewis index (TLI), and root mean square error of approximation (RMSEA). As recommended (Hu & Bentler, 1999), the following cut-off pointes were used to assess acceptable model fit: CFI $\geq$ .90, TLI $\geq$ .90, and RMSEA $\leq$ .10. Standard errors were also adjusted for non-independence since several respondents coming from the same household were nested within the analytic sample.

Second, between-sibling correlations were calculated to examine the concordance between siblings for major depression and violent victimization. Between-sibling correlations are traditionally first calculated before biometric model fitting approaches are used to investigate

whether there is evidence of genetic influence on a measure. Evidence for genetic influence would appear if siblings who share more genetic material have stronger between-sibling resemblance or concordance on a measure compared to siblings who share less genetic material. With regards to the present study, if full-sibling pairs (who share, on average, 50% of their genetic material with one another) demonstrate stronger concordance for major depression and violent victimization compared to half-siblings (who share, 25% of their genetic material with one another) than this can be interpreted as evidence of genetics partly explaining variation in risk for major depression and violent victimization. Since the cumulative measure of major depression analyzed in this step of the analysis was a categorical variable, and both measures of single and repeat victimization were binary variables, different types of correlations were calculated to assess between-sibling concordance for all three variables. Spearman's correlation coefficients were calculated for the cumulative measure of major depression and used to assess differences in concordance between full- and half-sibling pairs, while tetrachoric correlation coefficients were calculated for single and repeat victimization.

Third, univariate liability-threshold models were estimated to partition the total observed variance in liability for major depression and violent victimization into unstandardized (which were subsequently converted into standardized) estimates of variance attributable to additive genetic (symbolized as A), shared environmental (symbolized as C), and non-shared environmental (symbolized as E) influences (Neale & Cardon, 1992; Prescott, 2004). The magnitude of A represents the amount of variance in liability accounted for by additive genetic influences, while the magnitude of C represents the amount of variance in liability accounted for by shared environmental experiences that make siblings like one another for major depression and risk for violent victimization. The magnitude of E represents the amount of variance in

liability accounted for by environmental experiences that are not shared between siblings, but are unique to each sibling and create differences in risk for major depression and violent victimization between them. Measurement error is also included in the E estimate (Plomin, DeFries, Knopik, & Neiderhiser, 2013). After evaluating parameter estimates from the best fitting univariate ACE models, bivariate liability-threshold models were estimated to determine if genetic and environmental influences on major depression were shared with single and repeat victimization (see Figure 1). The variance in liability for major depression was therefore decomposed into additive genetic and environmental components common to single and repeat victimization and unique of single and repeat victimization. The proportion of total variance in liability for major depression accounted for by genetic influences shared with single and repeat victimization was calculated using the following algebraic equation:

$$a^2_{shared} = \frac{a_{12^2}}{a_{12^2} + e_{12^2} + a_{2^2} + c_{2^2} + e_{2^2}}$$

***Insert Figure 1 About Here***

Model fit for each univariate and bivariate liability-threshold model was evaluated using the following model fit indices: the Satorra-Bentler scaled-difference chi-square test ($\Delta\chi^2$) where a nonsignificant change in chi-square indicates that the model with fewer estimated parameters fits the data equally well and is preferred for the sake of parsimony (Satorra & Bentler, 2001); the comparative fit index (CFI) where CFI $\geq$ .90 indicates an adequate fit, while CFI $\geq$ .95 indicates a good fit; the root mean square error of approximation (RMSEA) where RMSEA $\leq$ .10 indicates an adequate fit and RMSEA $\leq$ .05 indicates a good fit (Hu & Bentler, 1999).

Because we restricted our analysis to only one full- or half-sibling pair per household with complete data on major depression and victimization from 2004 to 2012, the sibling pairs analyzed in the current study are a subset of the 16,083 sibling pairs nested within the CNLSY.

Preliminary analyses were conducted to examine whether this resulted in a biased sample. Equal proportions of male and female siblings were included in the analysis, but a higher percentage of Black (30% vs. 27%) and Hispanic (23% vs. 17%) siblings were included in the analysis compared to excluded siblings. There was no substantive difference in age between siblings included and excluded from the current analysis (20.14 vs. 20.87).

## RESULTS

*Phenotypic Analysis*. Before estimating a cross-lagged model, phenotypic associations between major depression and violent victimization at each survey wave were examined. Since all measures were binary, tetrachoric correlations were used to examine associations between all variables. Table 2 summarizes the phenotypic associations between major depression and victimization from 2004 to 2012. As can be seen, there was moderate stability in risk for major depression (*rho* = .46 to *rho* = .50, $p < .001$) and violent victimization (*rho* = .28 to *rho* = .51, $p < .001$) across waves. Major depression was positively and significantly associated with violent victimization at each survey wave (*rho* = .16 to *rho* = .28, $p < .001$).

***Insert Table 2 About Here***

*Cross-Lagged Analysis*. After establishing that major depression and violent victimization were associated with one another from 2004 to 2012, we estimated a bidirectional cross-lagged model to control for stability in both measures and assess the directionality of the association over time. The specified model also controlled for age, race, and sex. Overall, the model provided a close fit to the data (CFI = .95, TLI = .91, RMSEA = .04). Standardized coefficients from the specified model are presented in Figure 2. As shown, standardized path coefficients revealed that while violent victimization in 2004 was associated with increased risk for major depression in 2006 ($\beta = .09$, $p < .01$), major depression was more strongly, and

consistently, associated with subsequent increases in risk for violent victimization over time. Specifically, major depression was associated with increases in victimization from Wave 1 to Wave 2 ($\beta = .13$, $p < .001$), Wave 2 to Wave 3 ($\beta = .15$, $p < .001$), Wave 3 to Wave 4 ($\beta = .18$, $p < .001$), and Wave 4 to Wave 5 ($\beta = .16$, $p < .001$).

***Insert Figure 2 About Here***

*Behavioral Genetic Analysis.* To better understand the underlying mechanisms involved in the association between major depression and victimization over time, behavioral genetic analyses were conducted. Table 3 summarizes the cross-sibling correlations for the cumulative measure of major depression, single violent victimization, and repeat violent victimization. Full-siblings demonstrated stronger concordance for major depressive episodes ($r = .31$, $p < .01$), single victimization (*rho* = .34, $p < .01$), and repeat victimization (*rho* = .43, $p < .01$) compared to half-siblings. Cross-sibling correlations for full-siblings were not more than double the size of correlations for half-siblings, thus suggesting that dominance genetic effects (D) did not warrant further investigation. The results therefore suggested that additive genetic influences partly accounted for variance in liability for major depression and victimization.

***Insert Table 3 About Here***

Univariate ACE models were then estimated to identify the magnitude of additive genetic and environmental influences on all major depression and victimization. All estimated univariate models and standardized parameter estimates are summarized in Table 4, with the best-fitting model bolded. As shown, the best-fitting model for major depression was an ACE model (CFI = .89, RMSEA = .06) where 38% of the variance in liability was attributable to additive genetic influences, 16% of the variance in liability was attributable to shared environmental influences, and 46% of the variance in liability was attributable to nonshared

environmental influences. The best-fitting model for single violent victimization was an AE model ($\Delta\chi^2$ = 1.18, $p$ = .41, CFI = .92, RMSEA =.06) where 36% of the variance in liability for experiencing a single victimization was attributable to additive genetic influences, while 64% of the variance in liability was attributable to nonshared environmental influences. An AE model also fit the data more closely than a full ACE model for repeat victimization ($\Delta\chi^2$ = .07, $p$ = .26, CFI = .88, RMSEA =.06). Standardized parameter estimates from the best-fitting AE model revealed that 51% of the variance in liability for repeat violent victimization over time was attributable to additive genetic influences, while 49% of the variance in liability was attributable to nonshared environmental influences.

***Insert Table 4 About Here***

Bivariate liability-threshold models were then used to examine genetic and environmental influences on major depression shared with single and repeat victimization. Unstandardized parameter estimates from the best-fitting bivariate models are summarized in Table 5. As can be seen, after calculating the proportion of covariance between major depression and victimization with the unstandardized parameter estimates, the results revealed that 20% of the variance in liability for major depression was shared with single victimization, while 30% of the variance in liability for major depression was shared with repeat victimization. Additional standardized estimates for additive genetic and environmental influences of major depression common to, and unique of, single and repeat violent victimization are presented in Figures 3 and 4. Based on the presented estimates, there was a small, albeit significant, nonshared environmental overlap between major depression and both single and repeat victimization over time (1%, 95% CI: .01-.03). The results also show that genetic factors unique to major depression (22%) played a more

important role in the association between major depression and single victimization, compared to major depression and repeat victimization (10%).

***Insert Table 5 About Here***

***Insert Figure 3 About Here***

***Insert Figure 4 About Here***

## DISCUSSION

The current study sought to further elucidate the strength and direction of the relationship between depression and violent victimization over time. In particular, we hypothesized that violent victimization at a given time point would be more strongly associated with an increased risk for future depression. Contrary to this hypothesis, bidirectional cross-lagged analyses revealed that while victimization reported at Wave 1 was positively associated with depression at Wave 2 during late adolescence, depression was more strongly associated with a higher risk of victimization from late adolescence to young adulthood. These results lend support to a self-selection based interpretation of the data, which suggests that individual traits and behaviors may predispose some individuals to the risk of victimization as compared to others in a population (Beaver et al., 2016). These findings also offer a cautionary note about inferring the directionality of a correlation between two factors measured at a single time point as has been done in much of the prior research on this topic (e.g., Kaltiala-Heino et al., 1999; Kilpatrick et al., 2003).

In the second phase of the analysis, we assessed the magnitude of common genetic and environmental influences on single victimization and major depression as well as repeat victimization and major depression. We hypothesized that there would be a genetic overlap between victimization and depression and that there would be a larger shared genetic component

between repeat victimization and depression than between single victimization and depression. In line with our hypothesis, bivariate liability-threshold models revealed that the genetic covariance in liability between single victimization and frequency of major depression was 20% while the covariation between repeat victimization and frequency of major depression was 30%. The finding of a stronger genetic overlap between repeat victimization and major depression is also consistent with the selection hypothesis of victimization as selection is likely to play a larger role in repeat victimization, whereas single victimization is likely a more random occurrence.

In previous decades, scholars have articulated selection processes by which genetic propensities can contribute to increased risk of exposure to deleterious environments, such as what we observed in the current study (Scarr & McCartney, 1983). In this case, active or evocative gene-environment correlation (*r*GE) may be used to explain why some individuals who suffer from major depressive episodes are more likely to experience repeat violent victimization compared to others (Scarr & McCartney, 1983). For example, active gene-environment correlation takes place when an individual actively selects into an environment based in part on their genotype, while evocative gene-environment correlation takes place when an individual evokes specific behavioral or verbal responses from immediate members of their environment in part because of their genotype. Based on this logic, individuals with a higher level of genetic risk for developing major depressive disorder may engage in risky behaviors, such as heavy alcohol or illicit drug use, that actively place them into unsafe environments with antisocial individuals (active *r*GE) who victimize them because they are perceived as easy and vulnerable targets (evocative *r*GE). Additional research using other samples should help to further elucidate the specifics of how these selection processes work.

The current study is not without limitations, chief among them being the dichotomous coding of the relevant variables. First, prior researchers in the area of psychopathology have rightly pointed out the potential problems with the use of arbitrary cut-points separating psychopathology from otherwise "normal" variation on some trait (Kotov et al., 2017). The same limitations apply to our measure of victimization. Though we were able to assess repeated violent victimization over time (broadly defined), this part of our analysis relies on dichotomous indicators of victimization that were asked repeatedly across several waves of data collection. The use of a binary item restricts variation in undesirable ways, and does not allow us to examine victimization across a range of possible experiences (i.e., physical assaults versus sexual assaults). To the extent that our findings replicate with better measures of victimization (and continuously measured psychopathological items) remains an open empirical question.

Pending independent replication, the results of this study suggest a need for more research into the association between violent criminal victimization and psychopathology that can better assess the direction of relationships. Future research will also benefit from examining what specific kinds of psychopathology, as well as which specific symptoms, are most associated with victimization and evaluate how these traits might be increasing an individual's risk for violent victimization. Additionally, while genetically sensitive designs are not always feasible, the current study re-emphasizes the tenuousness of causally interpreting the associations found between two or more phenotypes without controlling for unobservable genetic confounds (Barnes et al., 2014).

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**Table 1. Descriptive Statistics**

| | Mean/Percent | SD | Min | Max | n |
|---|---|---|---|---|---|
| Major Depression$_{2004}$ | 18.97% | - | 0 | 1 | 1532 |
| Major Depression$_{2006}$ | 19.60% | - | 0 | 1 | 1910 |
| Major Depression$_{2008}$ | 17.95% | - | 0 | 1 | 2026 |
| Major Depression$_{2010}$ | 15.64% | - | 0 | 1 | 1984 |
| Major Depression$_{2012}$ | 14.60% | - | 0 | 1 | 1080 |
| Major Depression$_{2004\text{-}2012}$ | .85 | 1.20 | 0 | 5 | 1330 |
| Violent Victimization$_{2004}$ | 8.56% | - | 0 | 1 | 1106 |
| Violent Victimization$_{2006}$ | 12.50% | - | 0 | 1 | 1850 |
| Violent Victimization$_{2008}$ | 6.50% | - | 0 | 1 | 2042 |
| Violent Victimization$_{2010}$ | 4.00% | - | 0 | 1 | 1600 |
| Violent Victimization$_{2012}$ | 4.17% | - | 0 | 1 | 1048 |
| Single Violent Victimization | 8.04% | - | 0 | 1 | 1364 |
| Repeat Violent Victimization | 2.31% | - | 0 | 1 | 1364 |
| Age | 20.14 | 3.94 | 15 | 30 | 1364 |
| Black or Hispanic | 46% | - | 0 | 1 | 1364 |
| Male | 51% | - | 0 | 1 | 1364 |

**Table 2. Polychoric and Tetrachoric Phenotypic Correlations for Major Depression and Violent Victimization**

| | 1. | 2. | 3. | 4. | 5. | 6. | 7. | 8. | 9. | 10. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. Major Depression$_{2004}$ | - | | | | | | | | | |
| 2. Major Depression$_{2006}$ | .48** | - | | | | | | | | |
| 3. Major Depression$_{2008}$ | .45** | .48** | - | | | | | | | |
| 4. Major Depression$_{2010}$ | .43** | .47** | .50** | - | | | | | | |
| 5. Major Depression$_{2012}$ | .39** | .40** | .47** | .46** | - | | | | | |
| 6. Violent Victimization$_{2004}$ | .22** | .14** | .13** | .09 | .25** | - | | | | |
| 7. Violent Victimization$_{2006}$ | .20** | .21** | .27** | .16** | .21** | .51** | - | | | |
| 8. Violent Victimization$_{2008}$ | .10* | .17** | .16** | .21** | .22** | .29** | .28** | - | | |
| 9. Violent Victimization$_{2010}$ | .09 | .15** | .20** | .26** | .17* | .31** | .24** | .37** | - | |
| 10. Violent Victimization$_{2012}$ | .09 | .19** | .21** | .19** | .28** | .24* | .30** | .30** | .42** | - |

*Notes:* ** $p < .001$; * $p < .01$

**Table 3. Cross-Sibling Correlations**

| | Full-Siblings | | Half-Siblings | |
|---|---|---|---|---|
| | r/rho (*n*) | 95% CI | r/rho (*n*) | 95% CI |
| Major Depression$_{2004\text{-}2012}$ | .31* (473) | - | .19* (209) | - |
| Single Violent Victimization | .34* (473) | [.20-.48] | .20* (209) | [.07-.31] |
| Repeat Violent Victimization | .43* (473) | [.24-.59] | .23* (209) | [.08-.37] |

*Notes:* $n$ = number of sibling pairs. * $p < .01$.

**Table 4. Parameter Estimates from Univariate ACE Models**

| | A | C | E | $\Delta\chi^2$ | $\Delta$ df | *p* | CFI | RMSEA |
|---|---|---|---|---|---|---|---|---|
| Major Depression | | | | | | | | |
| **ACE** | **.38**** | **.16*** | **.46**** | - | - | - | **.89** | **.06** |
| | **[.28-.51]** | **[.04-.26]** | **[.39-.62]** | | | | | |
| AE | .49** | .00 | .51** | 25.71* | 4 | <.01 | .80 | .07 |
| | [.29-.62] | [.00-.00] | [.38-.71] | | | | | |
| CE | .00 | .24** | .76** | 49.03* | 4 | <.01 | .77 | .08 |
| | [.00-.00] | [.14-.39] | [.61-.86] | | | | | |
| E | .00 | .00 | 1.00*** | 104.55** | 5 | <.001 | .60 | .09 |
| | [.00-.00] | [.00-.00] | [1.00-1.00] | | | | | |
| Single Violent Victimization | | | | | | | | |
| ACE | .32** | .08 | .60** | - | - | - | .90 | .07 |
| | [.19-.44] | [-.01-.11] | [.51-.86] | | | | | |
| **AE** | **.36**** | **.00** | **.64**** | **1.18** | **1** | **.41** | **.92** | **.06** |
| | **[.25-.50]** | **[.00-.00]** | **[.50-.75]** | | | | | |
| CE | .00 | .10* | .90** | 37.81** | 1 | <.001 | .74 | .08 |
| | [.00-.00] | [.01-.18] | [.82-.99] | | | | | |
| E | .00 | .00 | 1.00*** | 92.61** | 2 | <.001 | .69 | .09 |
| | [.00-.00] | [.00-.00] | [1.00-1.00] | | | | | |
| Repeat Violent Victimization | | | | | | | | |
| ACE | .49** | .01 | .50** | - | - | - | .86 | .07 |
| | [.36-.69] | [-.02-.04] | [.32-.62] | | | | | |
| **AE** | **.51**** | **.00** | **.49**** | **.07** | **1** | **.26** | **.88** | **.06** |
| | **[.38-.74]** | **[.00-.00]** | **[.26-.62]** | | | | | |
| CE | .00 | .09* | .91** | 47.40** | 1 | <.001 | .65 | .08 |
| | [.00-.00] | [.01-.13] | [.87-.99] | | | | | |
| E | .00 | .00 | 1.00*** | 110.61** | 2 | <.001 | .60 | .10 |
| | [.00-.00] | [.00-.00] | [1.00-1.00] | | | | | |

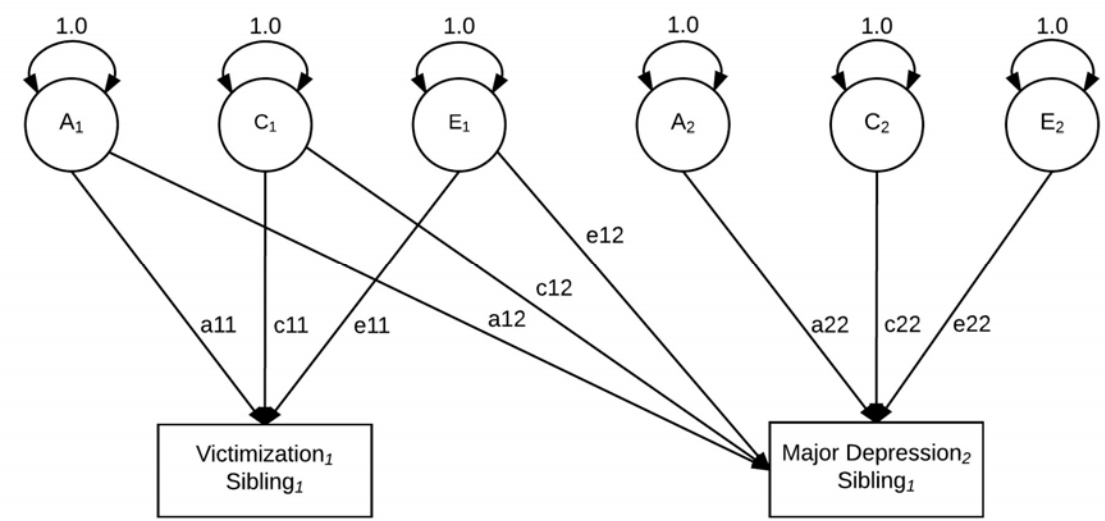
*Notes:* Standardized ACE parameter estimates presented. 95% confidence intervals presented in brackets. CFI = comparative fit index, RMSEA = root mean square error of approximation. ** $p < .001$; * $p < .01$

**Table 5. Parameter Estimates from Bivariate Models**

| | $A_1$ | $E_1$ | $b_A$ | $b_E$ | $A_2$ | $C_2$ | $E_2$ | $a^2_{shared}$ | 95% CI |
|---|---|---|---|---|---|---|---|---|---|
| Major Depression / Single Violent Victimization | .58 (.05) | .81 (.04) | .45 (.08) | .06 (.05) | .46 (.20) | .40 (.14) | .65 (.05) | .20* | [.07-.32] |
| Major Depression / Repeat Violent Victimization | .70 (.05) | .71 (.03) | .54 (.06) | .11 (.05) | .40 (.20) | .33 (.17) | .64 (.05) | .30** | [.19-.41] |

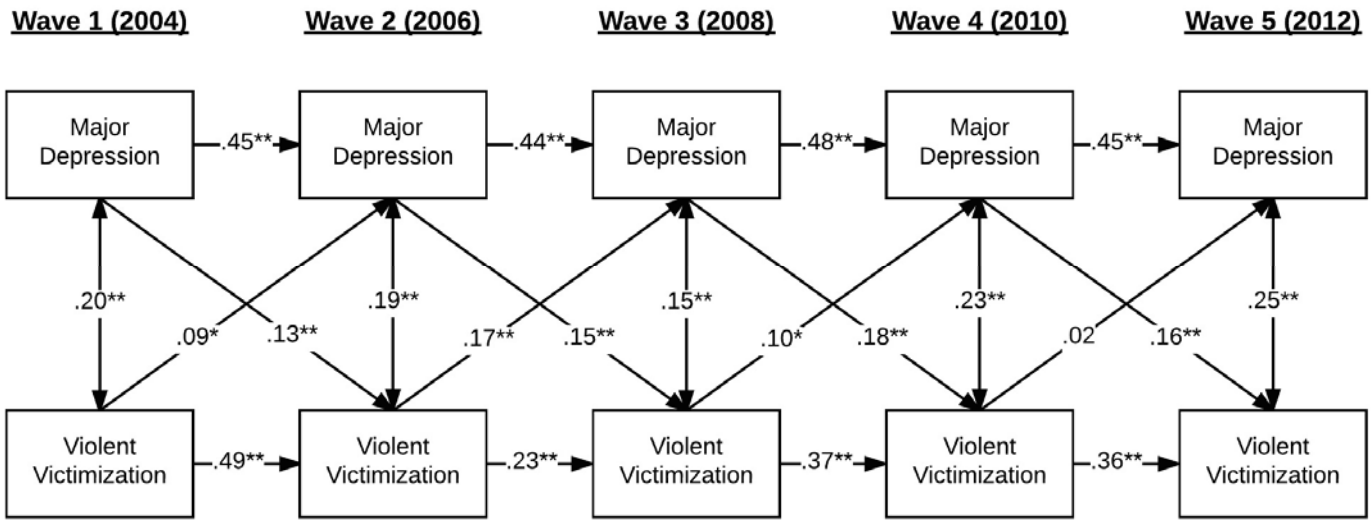
*Notes:* $a^2_{shared}$ = proportion of covariance between major depression and victimization explained by shared additive genetic influences. CI = confidence intervals.
** $p < .001$; * $p < .01$

**Figure 1. Path Diagram for Bivariate Model**




*Notes:* Bivariate model where major depression is regressed on latent additive genetic and environmental variance components of victimization (single or repeat). Path coefficients a11, c11, e11, a22, c22, e22 are additive genetic (a), shared environmental (c), and nonshared environmental (e) effects that are unique to each phenotype. Path coefficients a12, c12, and e12 are additive genetic, shared environmental, and nonshared environmental effects that are common between phenotypes. Path diagram is shown for one sibling pair.

**Figure 2. Cross-Lagged Model for Major Depression and Violent Victimization**




*Notes:* Standardized path coefficients presented. Model estimated with controls for age, race, and sex. Model fit statistics: $\chi^2 = 467.84(173)$, $p < .001$; CFI = .95; TLI = .91; RMSEA = .04. $^{**}p < .001$; $^{*}p < .01$

**Figure 3. Latent Genetic and Environmental Overlap between Major Depression and Single Violent Victimization**

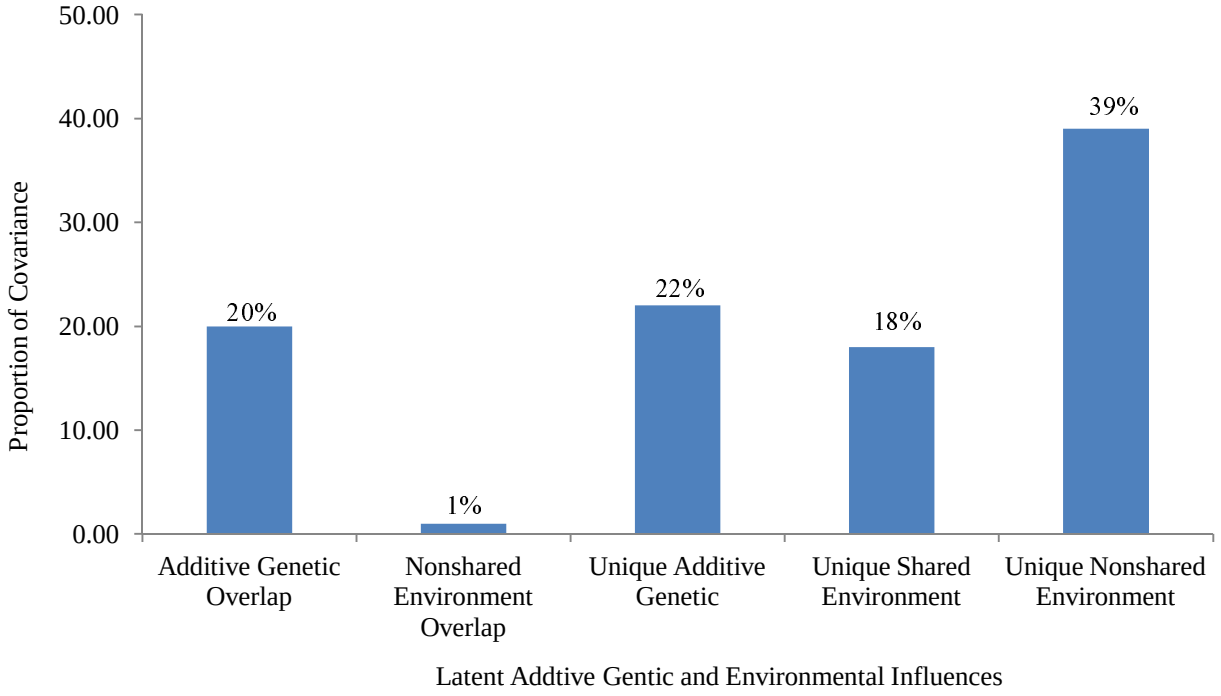


**Figure 4. Latent Genetic and Environmental Overlap between Major Depression and Repeated Violent Victimization**