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Age-related differences in the structure of genetic and environmental contributions to types of peer victimization

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Abstract

Background: The goal of the present investigation was to clarify and compare the structure of genetic and environmental influences on different types (e.g., physical, verbal) of peer victimization experienced by youth in pre-/early adolescence and mid-/late adolescence.

Method: Physical, verbal, social, and property-related peer victimization experiences were assessed in two twin samples (306 pairs, ages 9–14 and 294 pairs, ages 15–20). Cholesky decompositions of individual differences in victimization were conducted, and independent pathway (IP) and common pathway (CP) twin models were tested in each sample.

Results: In the younger sample, a Cholesky decomposition best described the structure of genetic and environmental contributors to peer victimization, with no evidence that common additive genetic or environmental factors influence different types of peer victimization. In the older sample, common environmental factors influenced peer victimization types via a general latent liability for peer victimization (i.e., a CP model).

Conclusions: Whereas the pre-/early adolescent sample demonstrated no evidence of a shared genetic and environmental structure for different types of peer victimization, the mid-/late adolescent sample demonstrates the emergence of an environmentally-driven latent liability for peer victimization across peer victimization types.

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Compliance with Ethical Standards

All study materials and procedures were approved by the VCU Institutional Review Board. Written informed consent was obtained from adults (parents or guardians of minor children and participants 18 years of age or older) and assent was obtained from minor children whose parents or guardians provided written consent. The authors of this paper have no potential conflicts of interest to disclose.

Keywords

Peer victimization; common pathway; behavior genetics; genetics; environment

Introduction

Short- and long-term negative psychological effects of peer victimization in childhood and adolescence have been well-documented and include anxiety, depression, social withdrawal, suicidal ideation, and substance use (e.g., Arseneault et al., 2010; Hawker & Boulton, 2000; Reijntjes et al., 2010; van Geel et al., 2014). Given the harmful effects of peer victimization, efforts have been made to understand the etiology of the phenomenon, focusing most often on characteristics of the perpetrator, home environments, and school environments that contribute to peer victimization perpetration (Card & Hodges, 2008; Cook et al., 2010; Hong & Espelage, 2012; Kljakovic & Hunt, 2016). Such research has informed prevention programs that aim to reduce bullying and peer victimization perpetration in school contexts.

Not all individuals have the same likelihood of being victimized by their peers. Previous research (Champion et al., 2003; Cook et al., 2010; Hong & Espelage, 2012; Hunt & Kljakovic, 2016) has examined victim characteristics (e.g., personality and other individual characteristics as well as aspects of the family environment) that increase the likelihood of victimization in childhood and adolescence. Additionally, previous efforts to identify genetic liability to experiencing peer victimization have found widely different estimates of additive genetic contributions ranging from 0% (Brendgen et al., 2008) to 45% (Brendgen et al., 2015; also see Brendgen et al., 2014; Shakoor et al., 2015). Variability in the heritability estimates is likely due to a number of factors, such as the use of different assessment methods (e.g., self-report, teacher report), measuring different forms of peer victimization (e.g., physical, verbal, social), and the examination of heritability in different age groups. For example, Brendgen and colleagues (2008) used a peer nomination procedure to identify victims of physical and verbal victimization (combined) in a sample of 6-year-olds, whereas Brendgen et al., 2015 used self-report to assess frequency of relational, physical, and verbal overt victimization (combined) in 10-year-olds. Theoretical and empirical work on developmental trajectories in heritability of behavioral phenotypes suggests that heritability estimates increase with age concomitant with decreased influence of passive genotype-environment correlations, such as parental environmental influence (Bergen et al., 2007; Tully et al., 2010). Stability of the environmental and genetic influences on peer victimization across age groups, however, remains unclear from previous investigations.

Although most investigations (e.g., Brendgen et al., 2008; Brendgen et al., 2015) rely on a single sum score across different types of peer victimization (e.g., physical, verbal, social), it is possible that genetic and environmental factors contributing to peer victimization differ according to the type of victimization experienced. Risk factors for physical violence, for example, may be different from risk factors for relational victimization. These different types of peer victimization must be distinctly assessed to clarify genetic and environmental influences. Additionally, different types of peer victimization have been differentially associated with psychopathology, strengthening the case for assessing them separately. For

example, Casper and Card's (2016) meta-analysis found associations between overt victimization and overt aggression on the one hand, and between relational victimization and internalizing problems on the other.

The present study extends previous work on the genetic epidemiology of peer victimization by examining additive genetic, shared environmental, and unique environmental contributions to different types of peer victimization within a developmental context. We examined the influence of genetic and environmental effects on four different types of peer victimization: physical victimization, verbal victimization, social manipulation, and attacks on property. Additionally, leveraging cross-sectional samples from two related twin studies representing different age groups (pre-/early adolescents aged 9–14 and mid-/late adolescents 15–20) allows for cross-age comparisons regarding the genetic and environmental etiology of peer victimization.

Behavior genetic studies of peer victimization have the potential to inform and enrich intervention efforts. For example, if additive genetics are found to be a significant contributing factor to experiencing one or more types of peer victimization, then universal interventions to prevent bullying and peer victimization perpetration may be coupled with targeted behavioral interventions to improve social skills in individuals who demonstrate genetically-mediated traits or behaviors (e.g., neuroticism, behavioral inhibition, poor emotion regulation) that put them at risk for peer victimization. The relevance of such intervention strategies may vary by age group to be responsive to age-related differences in the genetic and environmental structure of peer victimization types.

Hypotheses

In keeping with recent research on peer victimization trajectories which suggests continuous reductions in peer victimization across school years (Ladd et al., 2017), we expected to observe declines in peer victimization by age, with higher mean levels of peer victimization in the younger sample as compared to the older sample. Furthermore, because previous studies have found evidence for an additive genetic contribution to peer victimization and bullying victimization (a related, though not identical, phenotype), we hypothesized that at least one of the four peer victimization types assessed (physical, verbal, social, property) would have a significant additive genetic component. Two hypothesis-driven multivariate models describing the structure of genetic and environmental contributions to types of peer victimization were tested in each age group and compared to the Cholesky decomposition, which is used to estimate unstructured genetic and environmental covariance matrices. The independent pathway (IP) model hypothesizes that while both additive genetic and environmental factors may influence peer victimization of every type, the structure of the genetic factor is substantively different (i.e., with significant and substantively different factor loadings) from the environmental factor. In contrast, the common pathway (CP) model hypothesizes that genetic and environmental factors contribute to a single latent phenotype reflecting general risk for peer victimization, which can be decomposed into additive genetic, shared environmental, and unique environmental variance. The Cholesky decomposition would underscore the importance of treating each type of peer victimization independently, the IP model would highlight the differential importance of focusing on

genetic versus environmental factors that contribute to victimization type when designing intervention efforts, and the CP model would suggest that risk for victimization is general (i.e., not specific to type) and that genetic and environmental risk factors would respond to the same types of (phenotypic-level) interventions.

Method

Participants

The sample for the current investigation consisted of twin pairs from two separate but related studies: The Twin Study of Negative Valence Emotional Constructs (hereafter referred to as the Juvenile Anxiety Study [JAS]) and the Genetic Contributions of Negative Valence Systems to Internalizing Pathways (hereafter referred to as the Adolescent/Young Adult Twin Study [AYATS]). Both JAS and AYATS recruited (independent samples of) predominantly Caucasian twin pairs through the Mid-Atlantic Twin Registry (MATR), a database housed at Virginia Commonwealth University (VCU) of twins and their family members who have expressed interest in research participation (Lilley & Silberg, 2013). An unselected sample was recruited from MATR according to registry protocols to ensure generalizability of genetic and environmental estimates and to reproduce an epidemiological distribution of psychopathology. JAS study participants ranged in age from 9–14 years old ($M=11.42$, $SD=1.52$, 53% female), and AYATS participants ranged in age from 15–20 years old ($M=17.17$, $SD=1.34$, 58% female). The inclusion of AYATS participants up to age 20 in our sample is consistent with developmentally-informed definitions of adolescence (Sawyer, Azzopardi, Wickremarathne, & Patton, 2018). Exclusion criteria for JAS were: severe or unstable medical or neurological illness, past seizures of unknown etiology, intellectual disabilities, autism spectrum disorders, substance abuse, recent suicidal or homicidal ideation, psychotic episodes, current use of psychotropics (except stimulants for ADHD), and use of non-psychotropic medications with similar effects (e.g., beta-adrenergic blockers). Additional exclusion criteria for AYATS were: spatial learning disorder and pregnancy. In all cases, exclusionary criteria were established to avoid interference with data produced during physiological tests as part of study protocols and to decrease the risk of exacerbating existing medical conditions during laboratory tasks. Complete description of study aims, physiological measures, and laboratory tasks has been detailed elsewhere (Carney et al., 2016; Cecilione et al., 2018).

During the first assessment for each of these studies, participants were asked to complete a self-report questionnaire in the REDCap electronic data capture system hosted at VCU (Harris et al., 2009) that included the Multidimensional Peer Victimization Scale (Mynard & Joseph, 2000). The MPVS was added to the JAS questionnaire not long after the start of the study and was completed by a total of 617 individuals. Technical difficulties with electronic data capture prevented some AYATS participants from completing all items of the MPVS; these individuals ($n=168$) were excluded from the present analysis. The average age of MPVS completers in the JAS study was younger than the average age of non-completers; $t(435.04)=-6.62$, $p<.001$. The average age of MPVS completers in the AYATS study was older than the average age of non-completers; $t(525.79)=2.54$, $p=.01$. There were no differences in the proportion of males and females between MPVS completers and non-

completers in either JAS or AYATS. The final sample for the present investigation was 306 JAS twin pairs (96 MZ pairs, 210 DZ pairs) and 294 AYATS twin pairs (113 MZ pairs, 180 DZ pairs).

Measures

The Multidimensional Peer Victimization Scale (Mynard & Joseph, 2000) is a 16-item questionnaire that asks “how often during the last school year has another pupil done these things to you?” Response options are 0=“Not at all”, 1=“Once”, and 2=“More than once”. Four subscales (consisting of four items each for a possible score of 0–8 for each factor) are present in the MPVS to assess multiple types of victimization: physical victimization (e.g., “Punched me”), verbal victimization (e.g., “Called me names”), social manipulation (e.g., “Tried to get me into trouble with my friends”), and attacks on property (e.g., “Tried to break something of mine”). A strength of the MPVS is that it distinguishes two modes of overt victimization (physical and verbal) in addition to relational victimization (i.e., social manipulation) and property victimization, a category that is understudied in the literature (Crothers & Levinson, 2004; Betts et al., 2015). A confirmatory factor analysis was conducted for each sample (JAS and AYATS) separately and the four-factor structure affirmed (Eastman et al., 2018). Additionally, all subscales demonstrated good internal consistency (Eastman et al., 2018). To address the right-skewness of the distribution of each of the MPVS subscales, subscale scores were converted to ordered categories with thresholds chosen to ensure adequate number of twin pairs for analysis across the entire range of each subscale. Respondents in JAS and AYATS scoring 4 or above on the physical victimization scale were placed in a single category such that the continuous physical victimization subscale was converted to ordered categories of 0,1,2,3, and 4+. Verbal victimization and social manipulation subscales were less skewed in both samples, and all categories (0–8) were retained. For property victimization, JAS scores were converted to 6 categories (0,1,2,4,5–6,7–8) and AYATS scores were converted to ordered categories of 0,1,2,3, and 4+.

Analysis

This study employed a classic twin design, which leverages the differences between MZ and DZ twin types to estimate the relative contribution of genetic and environmental influences to individual differences the outcome phenotype(s). Biometrical structural equation modeling (SEM) was used to partition the variance and covariance for the peer victimization scales into additive genetic (A), shared environmental (C), and unique environmental (E) factors (Neale and Cardon, 1992). Additive genetic effects (A) are the cumulative effect of individual alleles at genetic loci influencing a behavior. Due to MZ twins sharing 100% of their genes and DZ sharing on average 50% of their segregating genome, the genetic factor contributes twice as much to the correlation between MZ twins compared to the correlation between DZ twins. Shared environmental effects encompass influences that make family members more similar to each other compared to pairs of random individuals and, therefore, contribute to the MZ and DZ twin correlations equally. Unique environment (E) describes environmental influences that contribute to the differences seen between co-twins, which also includes measurement error, and as such is uncorrelated within the twin pair.

Because sex differences in peer victimization by type have been previously identified in the JAS and AYATS samples (Eastman et al., 2018), sex was added as a covariate in all models to control for these effects. Additionally, because twins within a pair were assessed at the same time point, age has the potential to inflate shared environmental (C) estimates. Twin age at time of assessment was controlled for in all models within each sample to reduce such effects.

Standard assumptions of twin modeling (i.e., equality of variances, covariances, and thresholds within twin pairs and within and across zygosity types) were tested in each sample (JAS and AYATS) separately. As shown by the results of the log likelihood tests in Table I, standard twin-modeling assumptions held in each study sample; adding standard constraints did not significantly worsen fit between nested models at any step in JAS or AYATS. We note, however, that the model constraining thresholds to be equal across twin order and zygosity as well as covariances across twin order and within MZs and subsequent nested models for JAS had significantly worse fit when compared directly against the saturated model. This is likely due to the accumulation of minor disturbances in fit and does not represent a failure to meet standard twin modeling assumptions. Comparing proximal nested models with stepwise constraints shows adequate conformation to these assumptions. Accordingly, the thresholds and within-person covariance matrices of the victimization types were equal regardless of zygosity and twin order. The thresholds could not be equated across the JAS and AYATS samples without significant deterioration of model, indicating a different prevalence in peer victimization at different ages (data not shown). The within-person covariances also could not be equated across studies, implying that there is a different process of victimization for each age group; therefore, each study was analyzed separately.

We then fit a Cholesky decomposition, an IP model, and a CP model to determine which of these models best described the data in each sample (JAS and AYATS) separately. The Cholesky decomposition estimates the additive genetic, shared environment, and unique environment variance-covariance matrix for the four peer victimization factors (physical victimization, verbal victimization, social manipulation, and attacks on property). The IP model estimates a common latent factor for each variance component (A_c , C_c , and E_c), thus imposing a more hypothesis-driven structure relative to the Cholesky model. Residual additive genetic and environmental factors specific to each victimization type (A_s , C_s , and E_s), are also estimated. Accordingly, the IP model hypothesizes that the factor structure differs for each victimization type at the additive genetic, common environmental and unique environmental sources of variation. The CP model estimates a single latent factor accounting for the covariance between the victimization types, which can be decomposed into the three sources of variance (A_c , C_c , and E_c). The CP model hypothesizes that the factor structure for each source of variation is equal (i.e., the CP model estimates the same factor loadings for A_c , C_c and E_c). The CP model also estimates the residual additive genetic and environmental factors specific to each victimization type (A_s , C_s , and E_s), not accounted for by the relationship with the latent factor.

The CP model is nested within the IP model, which is nested in the Cholesky decomposition. Therefore, a likelihood ratio X^2 test can be used to compare model fit between the two hypotheses-driven models (the IP and CP models) and the Cholesky

decomposition. Degrees of freedom (df) for the test are the difference between the number of estimated parameters in the full model and the nested model. Model fit was also assessed by comparing the Akaike Information Criterion (AIC) between the two models, where lower values represent better fit (Aikake, 1987; Williams & Holahan, 1994). After identifying the best fitting model in JAS and AYATS, significance of each path was tested through likelihood ratio X^2 tests where a model constraining the path of interest to zero was compared against the model in which the path was estimated. Resulting p-values below .10 were halved, reflecting the mixture of X^2 distributions of the test statistic for testing significance of a single ACE model component (Wu & Neale, 2013). An exception were parameters in the E matrix necessary for model identification. For these parameters 99.9% confidence intervals were calculated to detect significant difference from zero.

All modeling was conducted using the OpenMx package (Neale et al., 2016) in the R statistical computing environment (R Core Team, 2016).

Results

Table II provides the phenotypic correlations, means, and standard deviations for JAS and AYATS. In both samples, types of peer victimization were highly correlated. Consistent with previous research, mean levels of peer victimization were lower in AYATS (the older sample) as compared to JAS (the younger sample) for all peer victimization types. In both samples, verbal victimization was the most common and physical victimization the least common type of peer victimization.

Table III compares the goodness-of-fit statistics for the Cholesky decomposition, IP and CP models for JAS and AYATS, respectively. For JAS, fitting the data to the IP and CP models resulted in a significantly worse fit when compared to the Cholesky decomposition ($p=.003$ and $.025$, respectively), leading to the conclusion that the Cholesky decomposition best fit the data. Thus, for JAS, separate additive genetic and environmental factors directly influence peer victimization types in the absence of higher order factors. For AYATS, neither the IP nor CP models showed significantly worse fit when compared to the Cholesky decomposition; however, the AIC showed better fit (i.e., a lower value) for the CP as compared to the IP. Thus, the CP model was the most parsimonious model and best represented the genetic and environmental influences on peer victimization in the older sample. In AYATS, common factors influence peer victimization types via a general latent liability for peer victimization.

Schematic depictions of the best fitting models for each study (Cholesky decomposition for JAS and CP for AYATS) are displayed in Figures 1a-1c and 2. As shown in Figure 1a, depicting the additive genetic components of the Cholesky decomposition for JAS, one set of additive genetic factors (A_1) loads significantly onto physical victimization and social victimization. No other additive genetic pathways are significant. The common environmental factors (C_1 - C_4) shown in Figure 1b do not load significantly on any victimization types. Three of the four environmental factors (E_1 - E_3) shown in Figure 1c load significantly onto the peer victimization types, with environmental etiologies shared between physical, verbal, social, and property victimization; between verbal and social victimization;

and between social and property victimization. There is no significant unique environmental factor specific to property victimization.

Figure 2, the CP model for AYATS, shows non-significant common additive genetic (A_c) and shared environmental (C_c) and significant unique environmental (E_c) factors loading onto the latent factor representing general risk for peer victimization which, in turn, loads significantly on each of the peer victimization types. Unique environmental factors not accounted for by the relationship with the latent peer victimization factor (E_s) load significantly onto each peer victimization type. As shown, there are significant shared environmental factors (C_s) unique to property victimization. Although paths from A_c and C_c to the latent peer victimization factor were not significant when tested individually, a model with both paths dropped simultaneously was a significantly worse fit for the data ($p < .01$) than the full CP model, suggesting familial aggregation of peer victimization.

We estimated the proportion of variance attributed to additive genetic factors (heritability) for each peer victimization type from the Cholesky decompositions. In the JAS sample, this was 42% for physical victimization, 32% for verbal victimization, 33% for social manipulation, and 23% for attacks on property. In the AYATS sample, the proportion of variance attributed to additive genetic factors was 4% for physical victimization, 13% for verbal victimization, 26% for social manipulation, and 4% for attacks on property. Constraining the A and C matrices across studies resulted in no worse fit when compared to a model combining the Cholesky decompositions from both studies, suggesting no significant change in genetic and shared environmental variance components across age group; however, the unique environmental component (E matrix) was significantly greater in the older sample (AYATS) as compared to the younger sample (JAS).

Discussion

As hypothesized, we found evidence that additive genetic factors contribute to risk for peer victimization in pre-/early adolescence (i.e., in the JAS sample). Specifically, we found an additive genetic factor that jointly and significantly influences physical and verbal victimization. By contrast, among the older age group (i.e., the AYATS sample), however, we found an environmentally-driven latent factor representing risk for overall peer victimization.

The present study is consistent with previous literature, which implicates genetic factors in risk for peer victimization (e.g., Brendgen et al., 2014). For example, Shakoor et al. (2015) report 35% additive genetic contributions to overall peer victimization in a sample of 12-year-olds in England and Wales. However, previous research is limited by assessment of overall peer victimization despite evidence distinguishing between types of peer victimization (e.g., Bradshaw et al., 2014) including differential influence on psychopathology (e.g., Casper & Card, 2016). In contrast, the present study examines the genetic and environmental contributions to different types of peer victimization and clarifies the structure underlying components of peer victimization based on common and specific contributions of genetic and environmental factors.

Prior research has examined peer victimization throughout childhood and adolescence including the contribution of genetic and environmental influences at different developmental periods (e.g., Brendgen et al., 2008, 2015). However, prior research has been unable to directly compare the genetic and environmental contributions to peer victimization in younger ages to those in older ages. By studying two genetic epidemiological samples that ranged in age from 9 to 20, the present study provides the first examination of age-related differences in the structure of genetic and environmental contributions to types of peer victimization. This study demonstrates that unique environmental factors have a significantly greater impact on peer victimization in mid-/late adolescence as compared to pre-/early adolescence. It further demonstrates that there are age-related differences in the *structure* of genetic and environmental contributions to peer victimization.

Among youth in pre-/early adolescence (ages 9–14 years), the Cholesky decomposition best described genetic and environmental influences on peer victimization, with the same set of additive genetic factors significantly influencing physical and verbal victimization. The Cholesky decomposition reflects the fact that a diverse set of peer victimization experiences are spread across a large set of respondents in the absence of a common genetic and environmental structure or a higher order latent liability for victimization. This pattern is consistent with previous work by Bradshaw and colleagues (2013), who found more diversity in patterns of victimization among middle school students (4 classes: verbal and physical [14.3%]; verbal and relational [25.9%]; high verbal, physical, & relational [10.3%], and low victimization/normative [49.5%]) as compared to high school students (3 classes: high verbal, physical, and relational [8.8%]; verbal and rumors [29.2%], and low victimization/normative [62%]).

As depicted in Figure 1a, the Cholesky decomposition also shows significant additive genetic contributions to physical victimization and social victimization, with the same set of genes contributing to both types of victimization. These genetic pathways to victimization may represent genetically-mediated personality or behavioral traits, such as emotion regulation difficulties, that confer risk for peer victimization in environments where perpetration can occur. In contrast, genetic and environmental contributions in mid-/late adolescence (ages 15–20 years) were best summarized by the CP model, wherein predominantly unique environmental factors influenced a latent phenotype representing general risk for peer victimization. Peer victimization becomes less common as youth develop through adolescence (Ladd et al., 2017; Nansel et al., 2001), and we have found that direct genetic effects on specific types of victimization in younger ages (as demonstrated by the Cholesky decomposition) are replaced by a latent general victimization liability in adolescence (as demonstrated by the CP model). The genetic factors that put one at risk for specific types of peer victimization (i.e., physical and social victimization) at younger ages give way to environmental risks for multiple types of peer victimization (i.e., physical, verbal, social, and property) in later adolescence.

In the younger sample, shared environmental factors, which could include socio-economic status and other family-level factors, did not contribute to variance in any peer victimization type, whereas shared environmental factors specific to property victimization were significant in the older sample. In both age groups, however, unique environmental factors

were strong contributors to victimization experiences. Although E_s also includes measurement error, this finding suggests that efforts to prevent peer victimization should continue to focus on malleable environmental factors (e.g., school and classroom climate, peer and social networks) that minimize the risk of peer victimization perpetration. Given broad consensus on the damaging psychological effects of peer victimization (e.g. Arseneault, et al., 2010; Hawker & Boulton, 2000), primary prevention of peer victimization utilizing environmental points of leverage should remain an important focus for universal interventions. Such interventions focus not just on those with “high risk” for peer victimization but have the potential to reduce likelihood of victimization for all children and adolescents, regardless of genetic liability. However, universal interventions that aim to prevent perpetration of peer aggression can be coupled with targeted behavioral interventions that minimize victimization risk. These interventions could be particularly beneficial among subgroups of younger adolescents who may demonstrate genetically-mediated social and behavioral difficulties. Evidence of genetic liability for peer victimization does not suggest that victims are responsible for acts perpetrated against them nor does it suggest that some adolescents will inevitably be targeted by their peers. Rather, the findings reported here suggest that improving social skills (Fox & Boulton, 2005), promoting prosocial behavior (Griese et al., 2016) and modifying even genetically-predisposed behavior patterns could benefit both sides of the perpetrator-victim dyad.

Limitations and Future Directions

These findings should be considered in light of several potential limitations. Firstly, the present study assessed peer victimization subjectively based on self-report, while other methods of assessment (e.g., teacher report or peer nomination) may provide different victimization estimates (e.g., Branson & Cornell, 2009; Cornell & Brockenborough, 2004). However, subjective experiences of peer victimization are particularly informative regarding clinical sequelae (Graham & Juvonen, 1998; Gromann et al., 2013).

Secondly, the two samples used in the present study were largely limited to participants of Caucasian ancestry to optimize power for future molecular genetic analysis of the sample. Future research is needed to examine the structure of genetic and environmental contributions to peer victimization, and age-related changes in this structure, for individuals of other ancestries.

Another limitation of the samples used is that each spans school levels and transitions that represent important unmeasured contexts for peer victimization. Specifically, the child sample includes elementary school students through middle school/junior high school/first year of high school, and the adolescent sample includes high school and college-aged individuals (all of whom reported on peer victimization that occurred over the past school year). Thus, the extent to which differences in the structure of genetic and environmental contributions to peer victimization are due to developmental changes vs. contextual ones cannot be disentangled in this study (Wang et al., 2016).

Lastly, the sample sizes available for this investigation precluded testing the effect of sex differences on genetic and environmental variance components. Furthermore, our sample sizes limit our ability to significantly identify more modest etiological influences. For

example, although we detected familial aggregation (i.e., significant combined influence of A_c and C_c on the latent peer victimization factor) in the AYATS sample, we may not have been adequately powered to detect significance of the additive genetic factor (A_c) alone.

The present study identifies the structure of genetic and environmental contributions to peer victimization, which helps to clarify the etiology of peer victimization in different age groups and which may be used to inform universal and targeted peer victimization prevention efforts on both sides of the perpetrator-victim dyad. Future investigations could examine shared genetic covariance between peer victimization types and psychopathology as well as between peer victimization types and personality traits (e.g., neuroticism) or behaviors that may confer risk for victimization.

Conclusion

The present investigation leveraged two genetic epidemiological studies of younger and older adolescents to clarify the structure of genetic and environmental contributions to types of peer victimization in different age groups. We estimated heritability of four different types of peer victimization, then further indicated distinct pathways of genetic and environmental contributions to types of peer victimization among youth in pre-/early adolescence whereas, among youth in mid-late adolescence, predominantly environmental contributions were directed through a latent factor describing liability for overall peer victimization. Exposure to peer victimization would be best minimized through universal environmentally-based interventions to reduce peer aggression perpetration in both age groups, coupled with targeted behavioral interventions among younger adolescents to reduce victimization risk.

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Abbreviations:

IP	(independent pathway)
CP	(common pathway)

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Figure 1. a. Standardized A path estimates for the JAS Cholesky decomposition b. Standardized C path estimates for the JAS Cholesky decomposition c. Standardized E path estimates for the JAS Cholesky decomposition

* $p < .05$

** $p < .01$

*** $p < .001$

Figure 2. Standardized path estimates for the AYATS common pathway model

* $p < .05$

** $p < .01$

*** $p < .001$

Table I.**Assumptions-testing results in JAS and AYATS**

Model	JAS				AYATS			
	EP	df	-2LL	p-value	EP	df	-2LL	p-value
Saturated	168	2128	6984.982	N/A	160	2284	6047.159	N/A
Thresholds equated across twin order	116	2180	7048.733	.127	112	2332	6101.149	.256
Thresholds equated across twin order and zygosity	90	2206	7081.798	.160	88	2356	6122.455	.621
Thresholds equated across twin order and zygosity; covariances equated across twin order within MZs	84	2212	7091.377	.144	82	2362	6128.581	.409
Thresholds equated across twin order and zygosity; covariances equated across twin order within MZs and within DZs	78	2218	7097.923	.365	76	2368	6136.621	.235
Thresholds equated across twin order and zygosity; covariances equated across twin order within MZs and across zygosity	72	2224	7107.349	.151	70	2374	6147.505	.092

EP=number of estimated parameters; *df*=degrees of freedom; -2LL=-2 times the log likelihood; MZ=monozygotic twins; DZ=dizygotic twins. *Note:* *p*-values represent the results of log-likelihood test of model fit between the described model and the one on the previous line.

Table II.

Correlations between peer victimization types, means, and standard deviations in JAS (below the diagonal) and AYATS (above the diagonal)

Phenotype	Physical	Verbal	Social	Property	Mean	SD
Physical	1.00	.40*	.32*	.43*	.46	1.31
Verbal	.43*	1.00	.60*	.55*	3.00	2.81
Social	.38*	.56*	1.00	.58*	1.55	2.24
Property	.51*	.51*	.52*	1.00	1.17	1.64
Mean	1.24	2.65	2.12	2.29	--	--
SD	2.06	2.47	2.25	2.23	--	--

SD=standard deviation

*
p<.001

Table III.

Goodness of fit statistics for Cholesky decomposition, independent pathway, and common pathway models in JAS and AYATS

Model	JAS					AYATS				
	EP	AIC	df	-2LL	p-value	EP	AIC	df	-2LL	p-value
Cholesky decomposition	64	2644.289	2236	7116.289	N/A	62	1401.137	2386	6173.137	N/A
Independent pathway	58	2651.634	2238	7127.634	.003	56	1400.594	2388	6176.594	.178
Common pathway	53	2645.867	2244	7133.867	.025	51	1393.706	2394	6181.706	.380

EP=number of estimated parameters; AIC=Akaike Information Criterion; df=degrees of freedom; -2LL=-2 times the log likelihood.

Note: p-values represent the result of log likelihood test of model fit between the described model and the Cholesky decomposition. Best fitting model for each study is in bold.