Published in final edited form as:

J Child Psychol Psychiatry. 2019 January ; 60(1): 72–81. doi:10.1111/jcpp.12966.

Violence exposure in an urban city: A GxE interaction with aggressive and impulsive behaviors

Rashelle J. Musci¹, Amie F. Bettencourt^{1,2}, Danielle Sisto¹, Brion Maher¹, Katherine Masyn³, and Nicholas S. Ialongo¹

¹Department of Mental Health, Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD

²Department of Psychiatry, Johns Hopkins School of Medicine, Baltimore, MD

³Division of Epidemiology & Biostatistics, Georgia State University School of Public Health, Atlanta, GA, USA.

Abstract

Background: Previous research has demonstrated a reciprocal relationship between community violence exposure and disruptive behavior problems among youth. No study to date, however, has explored the potential interaction between violence exposure in early adolescence and genetics.

Methods: We explore the gene x environment interaction's impact on teacher-rated aggressive and impulsive behaviors. Violence exposure during the middle school years was assessed using self-report. Genetic data collection occurred in emerging adulthood. A polygenic score was created using findings from a conduct disorder symptomatology genome wide association study (GWAS).

Results: Three longitudinal classes of teacher reported aggressive and impulsive behavior were identified. We found a significant relationship between violence exposure and class membership. There was a significant GxE interaction, such that those with below average levels of the polygenic score and who were exposed to violence were more likely to be in the moderately high aggressive and impulsive class as compared to the no to low class.

Conclusion: These findings highlight the influence of genetic risk together with violence exposure on adolescent problem behavior. Although youth may have little control over the environments in which they live, interventions can and should focus on helping all youth.

Keywords

Aggression; genetics; molecular; violence

Correspondence Rashelle J. Musci, 624 N. Broadway, Baltimore, MD 21205, USA. Rmusci1@jhu.edu.

Conflict of interest statement: No conflicts declared.

Introduction

According to national surveys, approximately one quarter of children and adolescents reported witnessing violence in the past year.^{1,2} These rates are on par with or higher than rates of other key adverse experiences, including child maltreatment and bullying.^{1,2} In addition, this type of exposure has been shown to disproportionately impact boys, youth growing up in urban environments, and children and adolescents of color,^{3–5} with some research suggesting that as much as 45% to 95% of African American youth have witnessed some violence in their community ranging from simple assault to murder.^{4–6} A specific focus on severe violent events is important as some research suggests that the more severe the violent event the more severe the maladjustment following exposure.⁷ However, our understanding of the specific role of exposure to severe violent events on outcomes is obscured as most studies combine a series of events ranging in severity into a single measure.⁷ Therefore, we focus our attention here on witnessing severe forms of violence exposure.

Past research has indicated that violence exposure contributes to a number of negative outcomes, including externalizing behavior problems.^{8–10,11} In light of such evidence, there has been an increased emphasis on understanding the factors that moderate the relationship between violence exposure and negative outcomes.¹² Research has suggested that exposure to community violence, including witnessing violence in the community, increases risk for aggressive and impulsive behaviors.¹³ Additionally, previous research¹⁴ has demonstrated a reciprocal relationship between community violence exposure and disruptive behavior problems among youth such that experiencing one increases the chances a youth will experience the other. We also know that genetics play a role in aggressive and impulsive behaviors.^{15–17} However, no study has explored the moderating role genetics may play in the relationship between witnessing violence and behavior problems utilizing polygenic approaches. Use of polygenic approaches, as opposed to candidate gene approaches, may be beneficial particularly when the phenotype of interest is a complex behavior such as aggressive and disruptive behavior.¹⁸ Polygenic approaches may help quantify a person's genetic burden for a particular behavior rather than elucidate specific biological pathways through which genetics influences behavior.¹⁹ A number of studies have found significant associations between polygenic risk scores and aggressive/impulsive behavior related phenotypes. For example, within the Eagle Consortium, several loci were found to be associated with aggressive behavior in children.¹⁷ Similar results have been found with hyperactive/impulsive behavior.²⁰

Previous work has explored exposure to different types of environmental stressors (e.g., child maltreatment, substance-abusing peers, neighborhood disadvantage) and the relation to behavior problems, along with the moderating role of known genetic loci. For example, Weder and colleagues (2008) found that aggressive behavior varied based on level of childhood trauma moderated by the MAOA genotype.²¹ Similar findings have been identified for impulsive acts of violence,²² delinquency,²³ and anti-social behavior.²⁴ Other research has focused on gene by environment interactions with polygenic approaches. For example, Salvatore and colleagues found that externalizing behavior problems varied based on degree of exposure to substance abusing peers and level of parental monitoring

moderated by a polygenic risk score. A meta-analysis of the field from 2015 suggests heterogeneous findings, likely a result of varied samples, polygenic scores and environmental influences.²⁵ There is, however, a dearth of research related to understanding the factors that influence the strength of the association between witnessing community violence during late childhood and early adolescence specifically and experiencing behavior problems in later adolescence.

In order to address these gaps in the literature, we utilized parallel process longitudinal latent profile analysis to capture heterogeneity in aggressive and impulsive behaviors over a unique period in the life course. We chose to include a measure of impulsivity in particular as difficulty controlling emotional and behavioral impulses is a core aspect of the genetic factor shared across externalizing behavior disorders.^{26,27} Further, to gain understanding about the antecedents of these complex behaviors, we explore a gene by environment interaction. Specifically, does witnessing community violence moderate the relationship between a conduct disorder polygenic score and longitudinal profiles of aggressive and impulsive behavior.

Method

Ethical information

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study. The Institutional Review Board at the Johns Hopkins Bloomberg School of Public Health approved the study. This work used data from the Center for Prevention and Early Intervention at the Johns Hopkins Bloomberg School of Public Health.

Participants

Data were drawn from a longitudinal study conducted by the Johns Hopkins Prevention Intervention Research Center (JHU PIRC). The analytic study sample consisted of 404 participants who were part of a school based preventive intervention trial and were representative of students entering first grade in nine Baltimore City public elementary schools in 1993. Students in the original sample were recruited for participation in two school-based, universal, preventive interventions targeting early learning and aggressive/disruptive behavior.²⁸ Three first-grade classrooms in each of the nine elementary schools were randomly assigned to one of the two intervention conditions or a control condition. In the analytic sample, 85.2% were African American, and 55.6% were male. Approximately 60.8% of the sample qualified for the federally subsidized school meals program (FARMS)—a proxy for socioeconomic status (SES)—in first grade.²⁹

Procedure

The Classroom Centered Intervention (CC).—The CC intervention consisted of three components: (1) curricular enhancements; (2) improved classroom behavior management

practices; and (3) supplementary strategies for children not performing adequately. Teacher behavior management practices were enhanced by the Good Behavior Game (GBG).³⁰

The Family–School Partnership Intervention (FSP).—Like the CC intervention, the FSP intervention included 3 components: (1) training for teachers/school mental health professionals and other relevant school staff in parent-school communication and partnership building³¹; (2) weekly home-school learning and communication activities, and (3) a series of 9 workshops for parents led by the 1st grade teacher and the school psychologist or social worker aimed at improving parent support for child academic achievement and positive discipline.

Both interventions included aggressive and shy behaviors and academic achievement as proximal targets. Interventions were provided over the first-grade year, following a pretest assessment in the early fall. Because intervention was not the focus of this analysis, intervention groups were collapsed to create a binary intervention variable. Written informed consent was obtained for each participant and the study was approved by the university’s institutional review board for the protection of human subjects in research.

Aggression and Impulsivity.—The Teacher Observation of Classroom Adaptation-Revised (TOCA-R) was used to assess students aggressive and impulsive behaviors at baseline (i.e., fall of grade 1 prior to randomization).³² The teachers also completed the Teacher Report of Classroom Behavior Checklist (TRCBC), an adolescent adaptation of the TOCA-R, in the spring of grades 6 through 12. Both measures included items such as “harms or hurts others physically” and “starts fights with classmates” to assess aggression, whereas impulsivity was measured using items such as “waits for turn” and “blurts out answer”. Teachers rated the student’s behavior on a six-point Likert scale from “almost never” to “always”. Previous research on the TOCA-R has demonstrated a high level of predictive validity. See Petras et al. for additional information on the reliability and validity of these measures.³³ The coefficient alphas for the TRCBC subscales in high school ranged from 0.79 and 0.88 for aggressive behavior and 0.65 to 0.78 for impulsivity.

Violence Exposure.—From 6th through 8th grade, participants reported on their violence exposure using items from the Children’s Report of Exposure to Violence (CREV) with an adapted response scale.³⁴ This self-report measure assessed the frequency of exposure to community violence in the past year. Students were asked to endorse whether or not they witnessed violence in a given year. The items of interest for the present analysis were witnessing someone being shot, stabbed, or killed. For the purpose of this study, a categorical variable was created such that endorsement of these items at any of the three time-points was coded as violence exposure.

Parental Monitoring.—The Structured Interview of Parent Management Skills and Practices–Youth Version (SIPMSP) was designed to assess the major constructs included in Patterson et al.’s model of the development of antisocial behavior and substance use.³⁵ The subscale used in this study is parental monitoring–supervision. The scale includes seven items measuring such things as how often the youth tell their parents when they will be back or leave a note about their whereabouts when they go out. Items are rated on a 5-point Likert

scale, ranging from 1 (all of the time) to 5 (never). The average score of the seven items was calculated to indicate the overall parental monitoring level. Higher scores indicate less parent monitoring.

DNA and Genotyping.—Blood or buccal samples were obtained in young adulthood. DNA was extracted and genotyped using Affymetrix 6.0 microarrays according to manufacturer's instructions.³⁶ Genome-wide genotypes were imputed to the 1000 Genomes Phase 3 reference panel using IMPUTE2 with pre-phasing performed in SHAPEIT.^{37,38} Best guess genotype files were generated in PLINK format.

Polygenic Score Generation.—We utilized a polygenic score based on a genome-wide assay to create a score associated with conduct disorder symptomatology. Recently, Dick and colleagues conducted a genome wide association study (GWAS) for conduct disorder symptomatology.³⁹ This analysis included participants from the Study of Addiction: Genes and Environment (SAGE), the cases of which met diagnostic criteria for alcohol dependence. Participants (N=3963) retrospectively reported on conduct disorder symptoms. The scores were made using the --score command in PLINK2⁴⁰ with mean imputation for missing genotypes and alleles weighted by the test statistic from the original GWAS. Top SNPs ($p < .01$) were included in a polygenic score and centered for inclusion in the analysis.

Population Stratification.—When exploring genetic associations, it is important to identify and control for population stratification, or genetic differences between subpopulations so that any significant associations found are not confounded by ancestry. Previous studies have emphasized the importance of this control, particularly in admixed populations (e.g., African American).⁴¹ In PLINK 1.9, we used principal components analysis (PCA), a dimension reduction technique based on the variance-standardized relationship matrix. This process generates linearly uncorrelated variables that can be used as covariates in regression analysis to help account for ancestry differences within the population.⁴²

Analytic Strategy.—In order to explore trajectories of aggression and impulsivity, a longitudinal latent profile analysis was conducted using Mplus (Mplus v.8.1.5 software).⁴³ This method of analysis allows for the joint modeling of teacher reported aggressive and impulsive behaviors over time. That is, classes are created based upon different patterns of indicator responses over time or trajectories. For the purposes of this analysis, the indicators were teacher reported impulsive and aggressive behaviors collected annually from 9th through 12th grade.

Analysis began with class enumeration whereby models were fit with an increasing number of classes. Each model was then compared using standard fit statistics such as the Bayesian information criterion (BIC) and the Lo-Mendell-Rubin Likelihood Ratio Test (LMR).⁴⁴ To better isolate the effects of violence exposure and genetics on membership in classes characterized by specific trajectories of aggressive and impulsive behaviors in high school, we controlled for known confounding variables in our analyses, including gender, socioeconomic status as measured by whether the child qualified for the free and reduced price school meals program (FARMS), intervention status, baseline (first grade) aggression

and impulsivity, population stratification, and parental monitoring. In order to explore a gene by environment interaction, we included an interaction term predicting latent class membership. Gene-environment correlation was found to be non-significant ($r=0.43$, $p=0.344$) and thus gene by environment interaction was the sole focus of the analysis.

Missing Data.—Missing data in longitudinal studies is common.⁴⁵ In the current analysis, the percent of annual missing data ranged from 22.6% to 27.6%. Current research standards indicate data can be treated as missing at random (MAR) when the missing data are unrelated to the outcome of interest. Chi-square tests revealed no significant differences in terms of ethnicity, $X^2(2)=1.805$, $p=.189$; gender, $X^2(2)=3.195$, $p=.081$; FARMS, $X^2(2)=0.068$, $p=.794$ and intervention condition, $X^2(3)=.003$, $p=.998$, between those who provided both phenotypic and genetic data and those who did not. Further, independent samples t-tests indicated no baseline differences in aggression and impulsivity between those who provided both phenotypic and genetic data and those who did not (aggression: $t=0.869$, $p=0.385$; impulsivity: $t=0.963$, $p=0.338$). In order to handle missing data, analysis was completed in Mplus using full information maximum likelihood estimation.⁴³ This type of estimation adjusts the parameter estimates using all information available. In order for these models to successfully converge, Mplus requires that the proportion of available data for each study variable and between each pair be at least 0.10.⁴³ Coverage for all variables and comparisons was at least 0.70 in the current study.

Results

Descriptive statistics can be seen in Table 1, including the proportion of individuals who reported violence exposure. The polygenic risk score was centered for ease of interpretation; however, unstandardized statistics are displayed in Table 1.

Class Enumeration.

The class enumeration process began with fitting a single class model and then comparing fit statistics with the addition of each class. Class fit statistics can be seen in Table 2. Fit statistics indicated a three-class model fit the data best, with the LMR-LRT becoming non-significant at a four-class model. Similarly, the BIC revealed that the last substantial drop occurred between the two- and three-class models, indicating that a three-class model fit the data best. Three longitudinal latent classes were identified (see Figure 1). Class one, which we designated the little to no problem behaviors class, contained a majority of the sample (77%), and displayed very low to no aggression and impulsivity. Class two, with 17% of the sample, displayed low levels of problem behaviors. Class three, with 6% of the sample, displayed moderately high levels of these behaviors, with higher levels of impulsivity compared to aggression. After class enumeration was complete, control variables were added, these included intervention status, gender, baseline aggression and impulsivity, parental monitoring, and FARMS.

Covariates of class membership.

There was a significant association between intervention status and class membership, suggesting that those who received either of the interventions were more likely to be in the

normative class as compared to the moderate class (est.=1.221, $p=.001$). There was also a significant association between gender and class membership wherein females were more likely than males to be in the normative class as compared to the moderate class (est.=−1.24, $p=.002$). Genetic ancestry, parental monitoring during the violence exposure years, FARMS, and first grade aggression and first grade impulsivity were not significant predictors of class membership.

We found a significant relationship between violence exposure and class membership (est.=1.528, $p=.012$), whereby those in the moderately high problem behaviors class were significantly more likely to be exposed to violence as compared to the low to no problem behaviors class. Additionally, there was a significant gene by environment interaction in the pairwise comparison of class 1 (little to no problem behaviors versus class 2 (low problem behaviors) (est.=−2.208, $p=.02$) as well as the pairwise comparison of class 2 (low problem behaviors) versus class 3 (moderately high problem behaviors) (est.=2.22, $p=.038$). Figure 2 provides a graphical probe of the interaction. This figure shows that those individuals who are at the mean for the polygenic risk score are more likely to be in the low to no behavior problem class regardless of violence exposure. Further, those who are lower than average on the polygenic risk score and exposed to violence are more likely to be in the moderately high class as compared to those not exposed to violence.

Discussion

Witnessing community violence is a significant problem during adolescence, with national estimates indicating that approximately 27.0% of youth ages 10–13 have witnessed an assault in their community, and 4.1% have witnessed a shooting.^{2,46} Rates of witnessing violence in the present study are sandwiched between these two estimates with 12.5% of middle school youth reporting witnessing someone being shot, stabbed, or killed in Baltimore City. The notable rate of witnessing severe community violence in Baltimore City is particularly concerning given the myriad negative psychological and behavioral consequences associated with such exposure.^{9,11,47}

Prior research has largely considered relations between witnessing community violence and aggression and impulsivity separately. Studies have shown that witnessing community violence contributes to increased aggressive behavior,⁴⁸ and these relations have been explained through a social learning perspective⁴⁹ (Bandura, 1986) such that witnessing violence models for youth that behaving aggressively is an acceptable way to get what you want or may even suggest that it is a necessary way to protect yourself from future harm.^{50,51} Research has also shown that witnessing violence may disrupt children's development of self-regulatory capacities resulting in greater difficulties with impulse control and emotional and behavioral dysregulation.^{12,47,52} Such self-regulation difficulties are a hallmark of multiple mental health problems.⁵³ The present study adds to this literature by examining the association between witnessing community violence and parallel patterns of aggression and impulsivity across adolescence. Consistent with prior research examining these behaviors separately, witnessing community violence significantly predicted membership in the class characterized by moderately high levels of both aggression and impulsivity. This finding may suggest that witnessing community violence simultaneously

disrupts children's self-regulatory capacities and reinforces to them that aggressive behavior is allowed resulting in increases in externalizing behavior problems. The increase in such externalizing behavior problems is particularly concerning as this pattern of behavior may be indicative of more serious problems such as a key subtype of conduct disorder marked by significant emotional and behavioral dysregulation.⁵⁴ Moreover, research has shown that such aggressive and impulsive behaviors can actually increase risk for further exposure to violence,^{48,55–57} which may in turn have more serious long-term consequences.

The present study extends prior work further by examining relations between witnessing severe community violence and externalizing behaviors and the moderating role of a polygenic risk score for conduct disorder symptomatology. There are several possible explanations for these findings. First, our results may indicate that in spite of some relative level of genetic “protection” against conduct problems, witnessing violence severely disrupts the prosocial development of the youth in the moderately high class resulting in the emergence of externalizing behavior problems. Alternatively, the findings may indicate that the indicator of genetic risk for conduct disorder symptomatology is more strongly linked to other conduct disorder symptoms (e.g., callous-unemotional traits) beyond aggression and impulsivity. In fact, some prior research has found stronger genetic influences for the conduct disorder pathway marked by significant callous-unemotional traits as compared to the conduct disorder pathway marked by significant emotional and behavioral dysregulation.^{54,58} Finally, it is also possible that the symptoms of aggression and impulsivity are indicative of a different mental health problem, such as Post-traumatic Stress Disorder, rather than Conduct Disorder (e.g., Armour, Contractor, Shea, Elhai, & Pietrzak, 2016; Netto et al., 2016) which might explain why those at low genetic risk for conduct disorder symptomatology manifested the highest level of impulsivity and aggression.^{59,60}

Previous research exploring potential gene by environment interactions in relation to violence has focused primarily on candidate genes and other important environmental stressors such as child maltreatment and involvement with deviant peers. While these environmental factors are clearly important and some studies have considered multiple adversities at once (e.g., Salvatore et al., 2015) community violence exposure remains understudied and a key environmental exposure that could impact a number of important developmental outcomes in the long-term. Future work should explore the relative impacts on development of witnessing community violence and other types of adversities together with genetics. Further, gene-environment interaction studies with candidate genes have found that violence exposure interacted with genotype to predict a number of behavioral outcomes.^{21,22,24} The mechanisms through which genetic factors can modify the effects of violence is not well-known (Holmes et al., 2005). It could be, as Holmes and colleagues (2005) point out based upon animal research, that while environmental factors can affect the development of emotional behavior, the duration and timing of sensitive periods of development are determined by genetic factors that vary across individuals. An elegant body of preclinical work on epigenetics shows complex interactions between genes and stressful experiences, with environmental influences causing changes in gene expression as well as stress-related behavioral outcomes (Szyf, 2012).

This study has important implications for prevention. Results revealed that youth with low genetic risk were more likely to be in the moderately high aggressive and impulsive class following witnessing severe community violence highlighting the impact of traumatic experiences on aggressive and impulsive behaviors. These data underscore the need for prevention efforts to include a community-wide strategy which implements interventions at multiple levels of the social ecology as well as a concerted effort to reduce community violence. Aside from systematic changes made to support urban areas to reduce violence, research has suggested that community social control and collective efficacy can aid in the reduction of violence.⁶¹ Therefore, we suggest a multifaceted approach, with societal and government changes coupled with community-based support. In concert with the efforts to reduce community violence, resources should be available to adolescents to improve coping skills and provide mental health services when needed.

A clear implication of this research is that preventing or limiting youth's exposure to violence may be the most effective way to reduce or prevent the negative consequences of such exposure. This may be accomplished by providing opportunities for families to move to less disadvantaged, safer neighborhoods where exposure to community violence is less likely (e.g., Moving to Opportunities Program).⁶² Alternatively, helping youth to establish strong bonds to prosocial institutions, including families, schools, prosocial peers, and community organizations may also prevent affiliation with deviant peers and involvement in dangerous situations that increase their risk for violence exposure.⁶³ For example, mentoring programs such as the Baltimore City Youth Bureaus and Big Brothers/Big Sisters programs that target high-risk youth and focus on increasing their bonds with prosocial adults and organizations have shown promise in reducing aggressive behaviors.⁶⁴

In spite of its many strengths, this study is not without limitations. In particular, we examined the association between violence exposure and externalizing behaviors within a predominantly African American sample living in an urban environment. In light of the unique nature of this sample, it may be difficult to generalize the findings to more diverse samples of youth. In addition, the issue of population stratification, particularly in studies that use polygenic approaches, is an active area of research. Currently, research suggests that in admixed populations use of a multi-ethnic sample for the GWAS discovery improves prediction in subsequent polygenic risk analyses.⁶⁵ However, more research must be done to understand and handle sources of bias that can be introduced due to population stratification. Further, this study did not examine the full array of behaviors consistent with a diagnosis of conduct disorder, which may have contributed to the finding that those at low genetic risk were exhibiting the most aggressive and impulsive behavior difficulties. Future studies should include additional measures of conduct disorder symptomatology (e.g., callous unemotional traits, serious rule violation, etc.) to determine whether violence exposure leads to genetic risk for conduct disorder being realized.

In addition, this study focused on severe exposure to violence, namely witnessing someone being shot, stabbed, or killed, and therefore results cannot be generalized to other types of violence exposure. Future research should attempt to replicate these findings using more diverse samples of youth from different contexts and examine a more diverse array of community violence exposures, including victimization experiences. Further, adolescents are

exposed to a larger number of experiences at home, in school, and in the community. Future research could benefit from taking a holistic view of violence exposure by including measures of domestic or home-based violence, school-based exposures like bullying, and other less severe community or neighborhood violence exposure. Additionally, more adolescents are spending time consuming and interacting with digital media, therefore future studies must not ignore cyber violence exposure.^{5,66,67}

Acknowledgements

This research was supported by grants to NI from the National Institute of Mental Health (MH57005), and the National Institute on Drug Abuse (DA11796) as well as a grant from the National Institute on Drug Abuse to Brion Maher (DA036525). This work used data from the Center for Prevention and Early Intervention at the Johns Hopkins Bloomberg School of Public Health. The authors are grateful for the collaboration of the Baltimore City Public Schools, teachers, parents, and students who participated in the study. They also appreciate the work of COGA and SAGE groups. The authors have declared that they have no competing or potential conflicts of interest.

References

1. Finkelhor D, Truner H, Ormrod R, Hamby SL. Violence, Abuse, and Crime Exposure in a National Sample of Children and Youth. *Pediatrics*. 2009;124(5):1411–1423. doi:10.1542/peds.2009-0467. [PubMed: 19805459]
2. Finkelhor D, Turner HA, Shattuck A, Hamby SL. Violence, Abuse, and Crime Exposure in a National Sample of Children and Youth: An Update. *JAMA Pediatr* 2013;167(7):614–621. doi: 10.1542/peds.2009-0467. [PubMed: 23700186]
3. Buka SL, Stichick TL, Birdthistle I, & Earls FJ. Exposure to Violence: Prevalence, Risks,. *Am J Orthopsychiatry*. 2001;71(May 2000).
4. Gaylord-harden NK, Cunningham JA, Zelencik B. Effects of Exposure to Community Violence on Internalizing Symptoms: Does Desensitization to Violence Occur in African American Youth? *J Abnorm Child Psychol* 2011;711–719. doi:10.1007/s10802-011-9510-x. [PubMed: 21505848]
5. Hanson RF, Self-brown S, Fricker-elhai AE, Kilpatrick DG, Saunders BE, Resnick HS. The Relations Between Family Environment and Violence Exposure Among Youth: Findings From the National Survey of Adolescents. *Child Maltreat* 2006;11(1):3–15. doi:10.1177/1077559505279295. [PubMed: 16382087]
6. Margloin G & Gordis EB. The effects of family and community violence on children. *Annu Rev Psychol* 2000;51.
7. Kennedy TM, Ceballo R. Who, what, when, and where? Toward a dimensional conceptualization of community violence exposure. *Rev Gen Psychol* 2014;18(2):69–81. doi:10.1037/gpr0000005.
8. Farver JAM, Eppe S, Fernandez A, Schwartz D. Community Violence, Family Conflict, and Preschoolers' Socioemotional Functioning. *Dev Psychol* 2005;41(1):160–170. doi: 10.1037/0012-1649.41.1.160. [PubMed: 15656746]
9. Fowler PJ, Tompsett CJ, Braciszewski JM, Jacques-tiura AJ, Baltes BB. Community violence: A meta-analysis on the effect of exposure and mental health outcomes of children and adolescents. *Dev Psychopathol* 2009;21:227–259. doi:10.1017/S0954579409000145. [PubMed: 19144232]
10. Kersten L, Vriends N, Steppan M, et al. Community Violence Exposure and Conduct Problems in Children and Adolescents with Conduct Disorder and Healthy Controls. *Front Behav Neurosci* 2017;11(November):1–14. doi:10.3389/fnbeh.2017.00219. [PubMed: 28174525]
11. McDonald CC & Richmond TR. The relationship between community violence exposure and mental health symptoms in urban adolescents. *J Psychiatr Ment Health Nurs* 2008;15:833–849. [PubMed: 19012675]
12. Low S, Espelage D. Conduits From Community Violence Exposure to Peer Aggression and Victimization: Contributions of Parental Monitoring,. *J Couns Psychol* 2014;61(2):221–231. doi: 10.1037/a0035207. [PubMed: 24635595]

13. McCabe KM, Lucchini SE, Hough RL, Yeh M, Hazen A. The Relation Between Violence Exposure and Conduct Problems Among Adolescents: A Prospective Study. *Am J Orthopsychiatry*. 2005;75(4):575–584. doi:10.1037/0002-9432.75.4.575. [PubMed: 16262515]
14. Lambert SF, Bradshaw CP, Cammack NL, Nicholas S. Examining the Developmental Process of Risk for Exposure to Community Violence among Urban Youth. *J Prev Interv Community*. 2011;39(2). doi:10.1080/10852352.2011.556558.Examining.
15. Gizer IR, Ehlers CL, Vieten C, Feiler HS, Gilder DA, Wilhelmsen KC. Genome-wide linkage scan of antisocial behavior, depression, and impulsive substance use in the UCSF family alcoholism study. *Psychiatr Genet* 2012;22:235–244. doi:10.1097/YPG.0b013e328353fb77. [PubMed: 22517380]
16. Vassos E, Collier DA, Fazel S. Systematic meta-analyses and field synopsis of genetic association studies of violence and aggression. *Mol Psychiatry*. 2014;19(October 2012):471–477. doi: 10.1038/mp.2013.31. [PubMed: 23546171]
17. Pappa I, St Pourcain B, Benke K, et al. A genome-wide approach to children's aggressive behavior: The EAGLE consortium. *Am J Med Genet Part B Neuropsychiatr Genet* 2015;(June):562–572. doi:10.1002/ajmg.b.32333.
18. Salvatore JE, Dick DM. Genetic influences on conduct disorder. *Neurosci Biobehav Rev* 2016. doi: 10.1016/j.neubiorev.2016.06.034.
19. Glahn DC, McIntosh AM. Commentary Using Polygenic Risk Scores to Establish Endophenotypes: Considerations and Current Constraints. *Biol Psychiatry Cogn Neurosci Neuroimaging*. 2017;2(2):113–114. doi:10.1016/j.bpsc.2017.01.008. [PubMed: 29560914]
20. Ebejer JL, Duffy DL, Van Der Werf J, et al. Genome-wide association study of inattention and hyperactivity-impulsivity measured as quantitative traits. *Twin Res Hum Genet* 2013;16(2):560–574. doi:10.1017/thg.2013.12. [PubMed: 23527680]
21. Weder N, Yang BZ, Douglas-palumberi H, et al. MAOAGenotype, Maltreatment, and Aggressive Behavior: The Changing Impact of Genotype at Varying Levels of Trauma. *Biol Psychiatry*. 2009;65(5):417–424. doi:10.1016/j.biopsych.2008.09.013. [PubMed: 18996506]
22. Tikkanen R, Ducci F, Goldman D, et al. MAOA Alters the Effects of Heavy Drinking and Childhood Physical Abuse on Risk for Severe Impulsive Acts of Violence Among Alcoholic Violent Offenders. *Alcohol Clin Exp Res* 2010;34(5):853–860. doi:10.1111/j.1530-0277.2010.01157.x. [PubMed: 20201935]
23. Liu H, Li Y, Guo G. Gene by social-environment interaction for youth delinquency and violence: Thirty-nine aggression-related genes. *Soc Forces*. 2015;93(3):881–903. doi:10.1093/sf/sou086. [PubMed: 25755300]
24. Frazzetto G, Lorenzo G Di, Carola V, et al. Early Trauma and Increased Risk for Physical Aggression during Adulthood: The Moderating Role of MAOA Genotype. *PLoS One*. 2007;(5). doi:10.1371/journal.pone.0000486.
25. Weeland J, Overbeek G, de Castro BO, Matthys W. Underlying Mechanisms of Gene–Environment Interactions in Externalizing Behavior: A Systematic Review and Search for Theoretical Mechanisms. *Clin Child Fam Psychol Rev* 2015;18(4):413–442. doi:10.1007/s10567-015-0196-4. [PubMed: 26537239]
26. Kendler KS, Prescott CA, Myers J, & Neale MC. The Structure of Genetic and Environmental Risk Factors for Common Psychiatric and Substance Use Disorders in Men and Women. *Arch Gen Psychiatry*. 2003;60.
27. Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *J Abnorm Psychol* 2002;111(3):411–424. doi:10.1037/0021-843X.111.3.411. [PubMed: 12150417]
28. Ialongo NS Werthamer L Kellam SG Brown CH Wang S Lin Y Proximal Impact of Two First-Grade Preventive Interventions on the Early Risk Behaviors for Later Substance Abuse, Depression, and. *Am J Community Psychol* 1999;27(5).
29. Ensminger ME, Forrest CB, Riley AW, Green BF, Ryan SA. The Validity of Measures of Socioeconomic Status of Adolescents. *J Adolesc Res* 2000;15(3):392–419.

30. Barrish HH, Saunders M, Wolf MM. GOOD BEHAVIOR GAME: EFFECTS OF INDIVIDUAL CONTINGENCIES FOR GROUP CONSEQUENCES ON DISRUPTIVE BEHAVIOR IN A CLASSROOM. *J Appl Behav Anal* 1969;2(2):119–124. [PubMed: 16795208]
31. Canter L, Canter M. *Parents on Your Side: A Comprehensive Parent Involvement Program for Teachers*. Santa Monica, CA: Lee Canter & Associates; 1991.
32. Werthamer-larsson L, Kellam S, Wheeler L. Effect of First-Grade Classroom Environment on Shy Behavior, Aggressive Behavior, and Concentration Problems. *Am J Community Psychol* 1991;19(4):585–602. [PubMed: 1755437]
33. Petras H, Masyn K, Ialongo N. The Developmental Impact of Two First Grade Preventive Interventions on Aggressive / Disruptive Behavior in Childhood and Adolescence: An Application of Latent Transition Growth Mixture Modeling. *Prev Sci* 2011;12:300–313. doi:10.1007/s11121-011-0216-7. [PubMed: 21519860]
34. Cooley-Quille MR, Turner SM, Beidel DC. Emotional Impact of Children's Exposure to Community Violence: A Preliminary Study. *J Am Acadamy Child Adolesc Psychiatry*. 1995;34(10).
35. Granic I, Patterson GR. Toward a comprehensive model of antisocial development: a dynamic systems approach. *Psychol Rev* 2006;113(1):101–131. doi:10.1037/0033-295X.113.1.101. [PubMed: 16478303]
36. Persico AM, Bird G, Gabbay FH, Uhl GR. D 2 Dopamine Receptor Gene TaqI A1 and B 1 Restriction Fragment Length Polymorphisms: Enhanced Frequencies in Psychostimulant-Preferring Polysubstance Abusers. *Soc Biol Psychiatry*. 1996;40(776–784).
37. Consortium T 1000 GP. A map of human genome variation from population-scale sequencing. *Nature*. 2011;467. doi:10.1038/nature09534.
38. Howie BN, Donnelly P, Marchini J. A Flexible and Accurate Genotype Imputation Method for the Next Generation of Genome-Wide Association Studies. *PLoS Genet* 2009;5(6). doi:10.1371/journal.pgen.1000529.
39. Dick DM, Aliev F, Krueger RF, et al. Genome-wide association study of conduct disorder symptomatology. *Mol Psychiatry*. 2011;16:800–808. doi:10.1038/mp.2010.73. [PubMed: 20585324]
40. Chang CC, Chow CC, Tellier LCAM, Vattikuti S, Purcell SM, Lee JJ. Second-generation PLINK: rising to the challenge of larger and richer datasets. *Gigascience*. 2015;1–16. doi:10.1186/s13742-015-0047-8. [PubMed: 25838885]
41. Montana G, Pritchard JK. Statistical Tests for Admixture Mapping with Case-Control and Cases-Only Data. *Am J Hum Genet* 2004;75:771–789.
42. Pritchard JK, Rosenberg NA. Use of Unlinked Genetic Markers to Detect Population Stratification in Association Studies. *Am J Hum Genet* 1999;65:220–228. [PubMed: 10364535]
43. Muthén LK, Muthén BO. *Mplus User's Guide*.
44. Nylund KL, Asparouhov T, Muthén BO. Deciding on the Number of Classes in Latent Class Analysis and Growth Deciding on the Number of Classes in Latent Class Analysis and Growth Mixture Modeling: A Monte Carlo Simulation Study. *Struct Equ Model*. 2007;14(4):535–569. doi:10.1080/10705510701575396.
45. Schafer JL, Graham JW. Missing Data: Our View of the State of the Art. *Psychol Methods*. 2002;7(2):147–177. doi:10.1037/1082-989X.7.2.147. [PubMed: 12090408]
46. Finkelhor D, Shattuck A, Turner H a, Hamby SL. Trends in Children's Exposure to Violence, 2003 to 2011. *JAMA Pediatr* 2014;(2003):1–7. doi:10.1001/jamapediatrics.2013.5296.
47. Overstreet S. Exposure to Community Violence: Defining the Problem and Understanding the Consequences. *J Child Fam Stud* 2000;9(1):7–25. doi:Article.
48. Farrell AD, Mehari KR, Kramer-kuhn A, Goncy EA. The Impact of Victimization and Witnessing Violence on Physical Aggression Among High-Risk Adolescents. *Child Dev* 2014;85(4):1694–1710. doi:10.1111/cdev.12215. [PubMed: 24410717]
49. Bandura A. *Social Foundations of Thought and Action: A Social Cognitive Theory*. Englewood Cliffs: Prentice-Hall, Inc; 1986.
50. Farrell AD, Erwin EH, Bettencourt A, et al. Individual Factors Influencing Effective Nonviolent Behavior and Fighting in Peer Situations: A Qualitative Study with Urban African American

Adolescents. *J Clin Child Adolesc Psychol* 2008;37(2):397–411. doi:10.1080/15374410801955821. [PubMed: 18470776]

51. Guerra NG, Huesmann LR, Spindler A. Community Violence Exposure, Social Cognition, and Aggression Among Urban Elementary School Children. 2003;74(5):1561–1576.
52. Monahan KC, King KM, Shulman EP, Cauffman E. The effects of violence exposure on the development of impulse control and future orientation across adolescence and early adulthood: Time-specific and generalized effects in a sample of juvenile offenders. *Dev Psychopathol* 2015;27:1267–1283. doi:10.1017/S0954579414001394. [PubMed: 26439073]
53. Beauchaine TP. Future Directions in Emotion Dysregulation and Youth Psychopathology. *J Clin Child Adolesc Psychol* 2015;44(5):875–896. doi:10.1080/15374416.2015.1038827. [PubMed: 26016727]
54. Frick PJ. *Conduct Disorders and Severe Antisocial Behavior* Springer Science & Business Media; 2012.
55. Lambert SF, Ialongo NS, Boyd RC, Cooley MR. Risk Factors for Community Violence Exposure in Adolescence. *Am J Community Psychol* 2005;36(September). doi:10.1007/s10464-005-6231-8.
56. Nylund-gibson SFLK, Ialongo NCNS. Patterns of Community Violence Exposure During Adolescence. *Am J Community Psychol* 2010;46:289–302. doi:10.1007/s10464-010-9344-7. [PubMed: 20878229]
57. Mrug S, Windle M. Bidirectional Influences of Violence Exposure and Adjustment in Early Adolescence: Externalizing Behaviors and School Connectedness. *J Abnorm Child Psychology*. 2009;37:611–623. doi:10.1007/s10802-009-9304-6.
58. Viding E, Blair RJR, Moffitt TE, Plomin R. Evidence for substantial genetic risk for psychopathy in 7-year-olds. *J Child Psychol Psychiatry*. 2005;16(6):592–597. doi:10.1111/j.1469-7610.2004.00393.x.
59. Armour C, Contractor A, Shea T, Elhai JD, & Pietrzak RH. Factor Structure of the PTSD Checklist for DSM-5: Relationships among symptom clusters, anger, and impulsivity. *J Nerv Ment Dis* 2016;204(2). doi:10.1097/NMD.0000000000000430.
60. Netto LR, Pereira JL, Nogueira JF, et al. Impulsivity is relevant for trauma exposure and PTSD symptoms in a non-clinical population. *Psychiatry Res* 2016;239:204–211. doi:10.1016/j.psychres.2016.03.027. [PubMed: 27016879]
61. Sabol WJ, Coulton CJ, Korbin JE. Building Community Capacity for Violence Prevention. *J Interpers Violence*. 2004;19(3 SPEC. ISS.):322–340. doi:10.1177/0886260503261155. [PubMed: 15005995]
62. Kling JR Ludwig J Katz LF. NEIGHBORHOOD EFFECTS ON CRIME FOR FEMALE AND MALE YOUTH: EVIDENCE FROM A RANDOMIZED HOUSING VOUCHER EXPERIMENT. *Q J Econ* 2005.
63. Herrenkohl TI Hill KG Chung I Guo J Abbott RD Hawkins JD. Protective factors against serious violent behavior in adolescence: A prospective study of aggressive children. *Soc Work Res* 2003;27(3).
64. Fagan AA, Catalano RF. What Works in Youth Violence Prevention: A Review of the Literature. *Res Soc Work Pract* 2012;23(2):141–156. doi:10.1177/1049731512465899.
65. Márquez-luna C, Loh P, Asian S, Diabetes T. Multi-ethnic polygenic risk scores improve risk prediction in diverse populations. *Genet Epidemiol* 2016;41(8):811–823.
66. Fanti KA, Demetriou AG, Hawa VV A longitudinal study of cyberbullying: Examining risk and protective factors. *Eur J Dev Psychol* 2012;9(2):168–181.
67. Upton D, Sung J, Ranney M, et al. Computers in Human Behavior Social media as a vector for youth violence: A review of the literature. *Comput Human Behav* 2014.

Key Points

- Exposure to community violence is all too common among adolescents with national estimates indicating that more than a third of middle school-age youth have witnessed such violence in the past year
- Witnessing community violence is associated with internalizing and externalizing behavior problems. What is not well understood, however, is the role genetics play in the relationship between witnessing community violence and experiencing mental health problems
- This study found that witnessing community violence in middle school significantly increases the likelihood that youth at low genetic risk for conduct problems will experience high rates of those problems by high school
- Findings have implications for efforts to reduce youth's exposure to violence to begin with and to provide appropriate supports and services to those who have already been exposed to prevent mental health problems

Figure 1.
Joint problem behavior trajectories resulting from the three-class parallel process longitudinal latent class analysis.

Figure 2.

A figure of class probabilities based on violence exposure and polygenic risk score distributions.

Table 1:

Descriptive Statistics

Gender	55.6% male		
Race	85.2% Black		
FARMS	60.8% Free/reduced		
Design status	64.4% Intervention		
Violence Exposure	12.5% exposed		
	<i>Mean (SD)</i>	<i>Minimum</i>	<i>Maximum</i>
Aggression 1 st	1.6511 (.88)	1.0	6.00
Aggression 9 th	1.6783 (.72)	1.0	4.70
Aggression 10 th	1.5759 (.59)	1.0	4.50
Aggression 11 th	1.5006 (.54)	1.0	3.70
Aggression 12 th	1.4394 (.50)	1.0	4.40
Impulsivity 1 st	2.2973 (1.2)	1.0	6.00
Impulsivity 9 th	2.1888 (1.0)	1.0	6.00
Impulsivity 10 th	2.0305 (.97)	1.0	6.00
Impulsivity 11 th	1.8610 (.83)	1.0	6.00
Impulsivity 12 th	1.7241 (.81)	1.0	6.00

Note. Violence Exposure defined as witnessing someone being shot Or stabbed between 6th grade and 8th grade

Table 2:

Fit Statistics

Fit Statistics					
Number of Classes	Loglikelihood	# parameters	BIC	LMR <i>p</i> - value	Entropy
1	−4222.973	16	8549.204	NA	NA
2	−3655.248	25	7471.836	0.00	.908
3	−3530.334	34	7280.091	.1416	.884
4	−3430.363	43	7138.232	.1807	.822
5	−3360.723	52	7057.035	.6054	.796

Note: BIC=Bayesian Information Criteria; LMR=Lo-Mendell-Rubin adjusted LRT test.