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The Protective Effects of Social Support on Hypertension Among African American Adolescents Exposed to Violence

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Abstract

African Americans develop hypertension earlier in life than Whites and the racial/ethnic disparities in blood pressure level can appear as early as adolescence. Violence victimization, a prevalent environmental stressor among inner-city youth, may play a role in such disparities. In a sample of inner-city youth in the United States, the current study examines the relationship between violence victimization and hypertension while investigating the role of social support in moderating that relationship. We analyzed eight waves of data from a longitudinal study of African American youth (n = 353, 56.7% female) from mid-adolescence (9th grade, mean age = 14.9 years old) to emerging adulthood (mean age = 23.1 years old) using probit regression. Higher levels of self-reported violence victimization during ages 14–18 was associated with more reports of hypertension during ages 20–23, after adjusting for sex, socioeconomic status, substance use, and mental distress. The relationship of violence victimization with hypertension was moderated by friends' support, but not parental support. The association between victimization and hypertension was weaker and non-significant among individuals with more peer support compared to those with less support. Researchers have reported many instances of associations of early violence exposure to later risk for hypertension; however, most have focused on childhood maltreatment or intimate partner violence. We extend these findings to violence victimization in an African American sample of youth from adolescence to early adulthood, while examining social support modifiers. The disparity in African American hypertension rates relative to Whites may partly be explained by differential exposure to violence. Our findings also suggest that having supportive friends when faced with violence can be beneficial for young adulthood health outcomes.

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Declaration of Conflicting Interests

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Keywords

violence exposure; community violence; youth violence; hypertension; high blood pressure; social support

Introduction

Violence Victimization and Hypertension

Violence victimization in adolescence results in negative developmental and health outcomes across the life span (Boynton-Jarrett et al., 2008; Boynton-Jarrett et al., 2013). Specifically, researchers are paying more attention to the links between victimization early in life and hypertension status in adulthood (Ford & Browning, 2014; Fuller-Thomson et al., 2010; Scott et al., 2011; Wilson et al., 2002). Hypertension increases the mortality risks of cardiovascular diseases and other diseases (Virani et al., 2020). Strikingly, the age-adjusted mortality rates primarily attributable to hypertension have been shown to be twice as high among African American males as White males (Virani et al., 2020).

Researchers have found that male adolescents were more likely to develop hypertension after witnessing violence with a weapon compared to unexposed peers (Ford & Browning, 2014). Similarly, female adolescents were more likely to develop hypertension after direct victimization with a weapon compared to non-victimized peers (Ford & Browning, 2014). Other researchers found an association between intimate partner violence (IPV) in late adolescence and young adulthood and the increased risk of cardiovascular disease 7–14 years post-exposure (Clark et al., 2016). In addition, Renner et al. (2017) found exacerbated effects of early-life exposures to polyvictimization (i.e., exposures to multiple forms of victimization) on hypertension. Specifically, female survivors of IPV who experienced three or more forms of victimization (e.g., childhood exposure to domestic violence, being psychologically or physically abused in childhood, and lifetime sexual assault) had higher predicted 30-year cardiovascular disease when compared to female survivors who experienced two or fewer forms of victimization (Renner et al., 2017). Researchers of polyvictimization also found that multiple forms of victimization, rather than repeated victimization, may be more detrimental to mental health outcomes (Finkelhor et al., 2007; Turner et al., 2010). In line with current conceptualizations of polyvictimization (Finkelhor et al., 2007; Renner et al., 2017; Turner et al., 2010), our construct of adolescent cumulative violence victimization includes a measure of psychological, property, and physical victimization that was assessed throughout the adolescent years (ages 14 to 18). Given that both violence victimization with a weapon and IPV were associated with hypertension, it is plausible that more general forms of victimizations will also predict hypertension.

Violence Victimization During Adolescence

Few studies have examined the effects of violence victimization experienced during adolescence on risk for hypertension, as most researchers have focused on childhood maltreatment (Fuller-Thomson et al., 2010; Scott et al., 2011) or IPV (Clark et al., 2016; Renner et al., 2017). Adolescence is a critical developmental period to study the effects

of violence exposure because this is a developmental period when rates of violence and victimization peak. In fact, in a nationally representative sample of youth, approximately 37% of adolescents aged 14–17 years old witnessed violence and 32% were physically assaulted during the study period between 2013 and 2014 (Finkelhor et al., 2015). This is concerning especially among African American youth residing in urban communities, who are exposed to significantly higher levels of violence than their rural or suburban counterparts (Boynton-Jarrett et al., 2013; Browning et al., 2017).

Health Disparities of Hypertension Among African Americans

Notably, hypertension takes a heavy toll among African Americans, with much higher prevalence and cardiovascular mortality rates than among non-Hispanic Whites (Mozaffarian et al., 2016; Virani et al., 2020). African Americans develop hypertension earlier in life than Whites, and the disparities in blood pressure level can appear during adolescence when African Americans begin experiencing higher nighttime blood pressure than Whites (Wang et al., 2006). Violence exposure is also associated with elevated nighttime blood pressure (Wilson et al., 2002). Blunted nocturnal decline in blood pressure is a contributing factor to the early onset of cardiovascular disease among African Americans (Wang et al., 2006). Although African Americans were underrepresented in these studies, evidence indicates increased hypertension risk among African Americans with elevated psychological distress (Rutledge & Hogan, 2002). More specifically, African American youth living in urban, disadvantaged communities are disproportionately exposed to violence and more likely to know someone involved in a fight, or have witnessed a violent crime (Motley et al., 2017; Wilson et al., 2012). Thus, African Americans in urban areas may be at greater risk for the development of hypertension and cardiovascular complications in early adulthood than Whites.

Theoretical Backgrounds of Risk and Protective Factors of Hypertension

Research is needed to examine the link between victimization and hypertension among African American youth and the protective factors that may help youth avoid the pernicious effects of violence victimization. We draw on the Transactional Model of stress and coping (Lazarus & Folkman, 1984) and Resilience Theory (Fergus & Zimmerman, 2005) to examine the moderating effects of social support on the risks of hypertension. The Transactional Model posits that the effects of a stressor are mediated by an individual's appraisal of the stressor and coping efforts (Lazarus & Folkman, 1984) and that social support has stress-buffering effects by influencing this process of stress-coping (Wenzel et al., 2002). Violence exposure and victimization are stressful life events for African American youth that can have long-lasting detrimental effects such as post-traumatic stress disorder (PTSD; Fitzpatrick & Boldizar, 1993) and other maladaptive stress responses (e.g., mental distress, heavy alcohol use) that may lead to chronic conditions.

Social support may mitigate maladaptive stress responses or counteract the negative effects. This is consistent with the protective model in Resilience Theory (Fergus & Zimmerman, 2005) that postulates an interactive model where the promotive factor (e.g., social support) buffers, or moderates, the negative effects of violence victimization. Positive family functioning and supportive partners have been identified as protective factors for victims

of violence (DuMont et al., 2007; Gorman-Smith et al., 2004). Other researchers found initial evidence that social support plays a crucial role in helping young African American women cope with the effects of IPV victimization (Mitchell et al., 2006; Thompson et al., 2000). These studies, however, were mostly based on cross-sectional designs (Mitchell et al., 2006; Thompson et al. 2000) or small samples of non-White participants (N = 138 to 263; DuMont et al., 2007; Gorman-Smith et al., 2004; Thompson et al., 2000).

Adolescents typically regard friends as more significant sources of social support than parents; however, adolescents who have stronger relationships with their parents have stronger relationships with friends as well (Arnett, 2013). Although both forms of social support remain significant, adolescents prefer to talk with their parents about their education and career goals and prefer to talk about more personal subjects such as their feelings, social relationships, and views on sex with friends (Arnett, 2013). Such differences may transfer to distinct functioning and quality of social support from parents and friends when adolescents are faced with adversity.

Examining the association between violence victimization and hypertension longitudinally will complement prior cross-sectional analyses and lead to a better understanding of the prospective linkage over time. A focus on such temporal associations will also motivate further explanatory research on hypertension and prevention efforts. Although rare, two studies examining the longitudinal associations between violence victimization/exposure and health outcomes have found that social support may play a significant role in promoting resilience among African American youth exposed to violence (Eisman et al., 2015; Hsieh et al., 2017). To our knowledge, however, no study has examined the associations between violence victimization and social support during adolescence (14–17) and hypertension in early adult (20–23) African American males and females together. Identifying whether social support promotes resilience among at-risk youth will inform intervention strategies that can be implemented early in life. Our findings will help identify contributing factors related to disparities in increased and earlier-onset hypertension risk experienced by African Americans as well as mitigating factors that protect against long-term negative health outcomes associated with violence victimization.

Current Study

We examined the longitudinal relationship between violence victimization in adolescence and hypertension in young adulthood in African Americans. We also examined whether social support from parents and friends moderated the effects of violence victimization. Applying the protective model of resiliency theory, we hypothesize that violence victimization would be associated with increased risk of hypertension later in life and that social support would moderate this relationship such that participants reporting more parental and friend support would have a weaker association between violence victimization and hypertension risk.

Methods

Participants

We analyzed eight waves of data from a longitudinal study of youth from mid-adolescence (Wave 1, 9th grade, mean age = 14.8 years) to emerging adulthood (Wave 8, mean age = 23.0 years). The original sample (N = 850) of the study includes 9th-grade students at Wave 1 attending one of four public high schools in Flint, Michigan. To be eligible for the study, participants had to have a grade point average of 3.0 or lower at the end of eighth grade and no school diagnosis of emotional or developmental impairments. They also had to self-identify as African American (n = 681), White (n = 142), or both (n = 27; Zimmerman et al., 2002). We included only the African American participants who had complete data for study variables over the first four waves (n = 584, 68.7% of the total sample).

Assessment of Selection Bias

To examine the potential selection bias in the longitudinal data, we first created a dichotomous variable to reflect whether the participant provided data for the medical condition variables (i.e., the hypertension indicator in waves 5–8 and other chronic conditions in Wave 5). We analyzed this as the selection indicator in a probit selection model (refer to more details in the “Analytic Plan” section) to assess the potential selection bias. Of the 584 participants in the study, 29.6% of participants had missing data on the hypertension question in one or more waves and 9.9% of participants did not respond at all during waves 5–8. About 7% of participants did not respond to the chronic medical condition question in Wave 5. These participants were treated as non-selected cases (or nonrespondents in waves 5–8). This resulted in a total of 272 (46.6%) non-selected cases (coded as 0) and 312 (53.4%) selected cases (coded as 1). We therefore have 312 African American participants in the final sample of the study.

Data Collection

Four waves of data were collected annually during high school (Waves 1 to 4; 1994 to 1997) and four years after high school (Waves 5 to 8; 1999 to 2002). Trained interviewers administered questionnaire-structured interviews which lasted 50–60 minutes for each individual in each wave of data collection. The interview portion of the study included most of the psychosocial variables in the data, such as exposure to violence and violence victimization, social support and influence of family and friends, as well as diagnosed chronic conditions. After each interview, participants completed a paper and pencil questionnaire for sensitive questions on substance use and sexual behavior to ensure confidentiality. Out of the original 681 African Americans who participated at baseline, 98.7% completed at least one additional assessment after baseline. The retention rate of participants completing all seven follow-up waves of assessments was 46% (n = 315). The University of Michigan institutional review board approved all study protocols and the protection of human subjects.

Measures

Hypertension (primary outcome).—Participants were asked if they had been diagnosed with hypertension during waves 5–8. In waves 5 and 6, they were asked: "have you *ever* been diagnosed with high blood pressure/hypertension?" In waves 7 and 8, the question was: "*In the last year*, have you been diagnosed with high blood pressure/hypertension?" Participants who indicated that they had been diagnosed as having high blood pressure/hypertension in any of the waves 5–8 received a score of 1. Participants who indicated that they had not been diagnosed as having such condition in all four waves received a score of 0. Participants who reported *not* having hypertension in at least one of the four waves and had missing data in any other waves were considered as having insufficient information to determine their health conditions. These participants were coded as missing (n = 173) and were included as part of the non-selected cases in the probit selection model.

Adolescent cumulative violence victimization.—We used 3 items to assess an adolescent's experience of violence victimization during waves 1–4. The questions asked about the frequency of violence-related victimization in the past 12 months on a 5-point Likert-type scale (1 = 0 times, 5 = 4 or more times): (1) "Had someone threaten to hurt me," (2) "Had something taken from me by physical force," and (3) "Had experienced being physically assaulted or hurt by someone." We recoded each item into a dichotomous variable of 0 (*never victimized*) and 1 (*victimized at least once*). A sum of recoded scores was created for each wave (ranging from 0 to 3) and a mean of these scores across the four waves was created as an indicator of cumulative victimization experienced during adolescence in our probit models. The final score for each individual represents an average of the number of types of victimization over the four years.

Parents' and friends' support.—Five 5-point Likert scale (1= *not true*, 5 = *very true*) items from the Parental Support Scale were used to assess participants' perceived parental support in waves 1–4 (Procidano & Heller, 1983). Participants also reported friends' support during waves 1–4 using the same 5 items specified to friends (Procidano & Heller, 1983). Example items include "I rely on my mother/father/friends for emotional support" and "My mother is/father is/friends are good at helping me solve problems." In the first wave of data collection, support from parents was measured using a single set of items (i.e., parent support instead of mother/father support separately). In waves 2 to 4, mother and father support were asked separately. We computed the mean composite parental support score by combining mother and father support scores in each wave (if a parent was missing, we used just the score for the remaining parent). Cronbach's α values ranged from .88 to .95 for parent support and from .82 to .90 for friends' support across waves 1–4. We calculated a composite score by averaging across the four waves for parents' support and friends' support, respectively. Scores of parental support and friends' support were mean-centered before computing the interaction term with a mean-centered violence victimization score.

Covariates

Socioeconomic status.—We assessed the highest occupational prestige score at Wave 1 for either parent as the indicator of socioeconomic status (SES). Participants were asked to indicate their father's and mother's occupations. Prestige scores were then assigned to

occupations based on 20 major occupational classifications (Nakao & Treas, 1990). The highest occupational prestige score of the two parents was assigned to the participant. The scores of our sample range from 29.28 (private household work) to 64.38 (professional). The mean prestige score was 40.14 (*SD* = 10.48), which represents a skilled blue-collar occupation (Caldwell et al., 2004).

Substance use.—We measured past 30-day cigarette and alcohol use during waves 1 to 8. Participants were asked: "How often have you smoked cigarettes during the past 30 days?" on a 7-point frequency scale: 1 (not at all) to 7 (two packs or more per day). They were also asked: "In the last 2 weeks, how many times have you had 5 or more drinks in a row?" on a 6-point scale: 1 (none) to 6 (10+ times). We averaged the scores of each item across waves 1 to 4 and 5 to 8, respectively, and then averaged them to create a substance use score.

Depression and anxiety.—We measured mental distress during waves 1 to 8 using the Brief Symptom Inventory (Derogatis & Spencer, 1982) for depression and anxiety. The Cronbach's α ranged from .78 to .86 for depression and .79 to .88 for anxiety across waves 1–4. The ratings of anxiety and depression were averaged to create a mental distress score for each wave of waves 5 to 8. We then created a composite score by taking a mean of anxiety and depression across waves 5 to 8 for the primary regression model. We also created a mean depression score across waves 1 to 4 for the embedded selection model (refer to more details in the "Analytic Plan" section).

Perceived stress.—We used 5 items from the Perceived Stress Scale to assess the lack of control participants may feel in their personal life during waves 2 to 4 (Cohen et al., 1983). Participants were asked about feeling in control in the last month on a 5-point Likert scale (1= never, 5 = very often). Cronbach's α ranged from .74 to .83 across waves 2–4. This scale was not measured in Wave 1. We therefore created a mean score for perceived stress across waves 2 to 4.

Future orientation.—We used 2 items to assess how often the participant thought about the future during waves 1 to 4: "I think a lot about my future job" and "I think a lot about my career will be." Participants responded to a scale range from 1 (no true) to 5 (very often). Inter-item correlations range from .60 to .63 across four waves. We created a mean score for the 2 items for each wave and then computed a composite score by averaging the mean scores across waves 1 to 4.

Other chronic medical conditions.—Participants were asked at Wave 5 about whether they have ever been diagnosed with the following chronic conditions: chronic bronchitis/ emphysema, diabetes, sickle cell diseases, other diseases. Participants who said they have been diagnosed as having any of the medical conditions were coded as 1 and 0 if they have never been diagnosed at or before Wave 5.

Analytic Plan

Our analyses were conducted using probit regression models with sample selection. The primary probit model (substantive equation) was specified to include predictors during

waves 1–4 (violence victimization, parental and friends' support), the dependent variable during waves 5–8 (self-reported hypertension diagnosis), and the following covariates: sex, age and SES at Wave 1, chronic medical conditions at Wave 5, mental distress in waves 5–8 and heavy substance use in waves 5–8. Next, we included the interaction between violence victimization in waves 1–4 and social support in waves 1–4 in the model to examine the moderating effect of social support on the relationship between violence victimization and hypertension, holding all other predictors constant.

Selection Model for Assessing and Correcting for Nonresponse Bias in Longitudinal Data—Probit models with sample selection (van de Ven & van Praag, 1981), also known as Heckman selection models (Heckman, 1979), involve the simultaneous estimation of a *primary probit model* and an *embedded selection model* to address non-random missing values due to sample selection bias (Cuddeback et al., 2004; Hall, 2002). The models involve two dependent variables: the primary outcome variable (i.e., self-reported hypertension diagnosis) and the response/selection indicator (i.e., whether participants responded in waves 5–8). The primary probit model examines factors that may predict self-reported hypertension diagnosis. The simultaneously embedded selection model examines additional covariates that may influence the nonresponse, in other words, the missingness process. The Heckman selection model allows the underlying error terms for the two dependent variables (i.e., self-reported hypertension diagnosis and the probability of responding) to be correlated. A stronger correlation indicates stronger selection bias.

Selection models have been successfully applied to address nonresponse bias in longitudinal data about substance use (West & McCabe, 2017). Such corrective approaches can examine whether a systematic nonresponse bias exists and provide the adjusted coefficients in the primary model by accounting for the potential selection bias. The sample can then be considered as if it was randomly selected in the adjusted model (Hall, 2002; Heckman, 1979). The model fitting process results in estimates of the regression coefficients that are unbiased by the differential missingness. For the embedded selection model to be well-identified, we included a set of variables (i.e., perceived stress during adolescence, depressive symptoms during adolescence, future orientation, sex, SES, age) that may contribute to study attrition exclusively and are not statistically significant in the substantive equation (Cuddeback et al., 2004; Stata, 2013). We used the *heckprobit* command in Stata (Version 15.0) to fit the probit model with sample selection.

Results

Table 1 presents demographic characteristics by selected sample (n = 312) and non-selected sample (n = 272). Of the 312 respondents who (a) responded in waves 5–8 and (b) had information available to determine the indicator of interest, 51 reported being diagnosed with hypertension and 261 reported not having been diagnosed with hypertension during waves 5–8. Twenty-two males (16.5% of males) and 29 females (16.2% of females) reported being diagnosed with hypertension, indicating that both prevalence rates are higher when compared to roughly 8% among of all Americans aged 20–34 during 1999–2002 (National Center for Health Statistics, 2017). In each of the four waves of data collection during adolescence, the participants reported 0 to 3 types of victimization out of a total of three

types of victimization asked annually. About 29% of the participants reported never being victimized (a score of 0) during adolescence.

In the primary probit model examining the association between violence victimization and hypertension diagnosis, violence victimization during adolescence was associated with reports of a hypertension diagnosis during age 20–23 (Beta = .45; 95% C.I. = .20, .70; Table 2). Other covariates were not associated with the outcome. The embedded selection model (Table 2) generated an estimate of *rho* (the correlation of the residuals in the selection model) suggesting that this correlation was not different from 0 (95% C.I. = −.79, .96) confirming no evidence of systematic nonresponse bias in our final sample.

We found a two-way interaction between victimization and friends' support (Beta = −.49; 95% CI = −.95, −.02), but no interaction effect for parental support (Beta = .39; 95% CI = −.08, .87; Table 2). We plotted marginal predicted values for the probability of hypertension according to the fitted model separately for cases with high (1 *SD* above the mean; 4.05), average, and low (1 *SD* below the mean; 2.51) values of friends' support to further investigate this interaction. Figure 1 presents the corresponding sets of marginal predictions illustrating the relationships between violence victimization and risks of hypertension as moderated by friends' support. Marginal predictions confirmed that the association between victimization and hypertension was weaker and non-significant among individuals with more peer support (1 *SD* above the mean, Beta = .02, CI =−.04, .08) compared to those with less support (1 *SD* below the mean, Beta = .18, C.I. = .11, .23) (Figure 1).

Discussion

Our results support previous findings linking violence exposure in childhood to later risk for hypertension (Ford & Browning, 2014; Fuller-Thomson et al., 2010; Scott et al., 2011; Wilson et al., 2002). We extend these findings to victimization during adolescence when rates of violence increase. This study informs research on the biopsychosocial links between violence exposure and chronic diseases and contributes to our understanding of modifiable risks and protective factors of hypertension among African Americans. Our finding that violence victimization was associated with later reported diagnoses of hypertension after adjusting for relevant risk factors suggests that violence victimization is a significant health risk factor among African American adolescents. Given that African American adolescents experience higher rates of violence victimization relative to Whites, the long-term sequelae of such exposure may help explain part of the disparity in African American hypertension rates relative to Whites. Our results are consistent with prior research supporting a psychosomatic connection between social experience and physical health (Dong et al., 2004; Fuller-Thomson et al., 2010; Scott et al., 2011). Our study adds longitudinal evidence to this body of work by demonstrating that the negative effects of violence exposure are manifested in a relatively short time in developmental terms.

Future studies should use a full frequency scale for victimization to examine if an inoculation or desensitization effect occurs for African Americans exposed to violence victimization. Some studies have demonstrated curvilinear associations between violence exposure and mental distress or depression among African American youth, where youth

exposed to high levels of violence began to show lower rates of depression/distress (Gaylord-Harden et al., 2011; Ng-Mak et al., 2004). However, scholars also note alternative explanations of the curvilinear findings: instead of becoming desensitized to violence, African American youth may actually be suppressing depressive symptoms or "acting tough" for protection in response to high levels of violence exposure (Gaylord-Harden et al., 2011). Expanding the examination of the desensitization effect to physiological indicators such as hypertension may further the understanding of the observed phenomenon beyond psychological responses.

Prior cross-sectional studies suggest that social support may play a crucial role in the coping process among African American women who are victims of IPV (Mitchell et al., 2006; Thompson et al., 2000). Our results add to existing evidence of a protective model of resilience in that friend support buffers the long-term negative effects of violence victimization experienced during adolescence. This suggests that friend support is an enduring social resource. Encouraging the development and maintenance of friendships and creating opportunities for engagement with positive peers may help increase the protective effect of social support. Researchers have also found that adolescents experience variability in the availability and quality of support from friends (Boisvert & Poulin, 2016). Future research including measures for the quality (i.e., satisfaction) and quantity (i.e., social ties) of social relationships and the types of support (i.e., tangible, emotional, or informational support) may help further the understanding of the salubrious effects and inform intervention strategies for African American adolescents. We did not find evidence that parental support during adolescence offers the same buffering effects. This finding, however, is consistent with Arnett's (2013) notion that adolescents typically have less involvement with family and greater involvement with peers. Therefore, the peer influences on risk behavior, support, and nurturance may be expected to be more relevant than parental influences at the developmental stage we studied.

Limitations

Several study limitations should be acknowledged. First, missing data were not trivial, but we applied a relatively new approach to address systematic nonresponse over time (West & McCabe, 2017). We have included in our regression model the available covariates that we believe may be confounders of the relationship between hypertension and victimization. However, the presence of unmeasured confounders can never be eliminated in observational data. We acknowledge that the adjustment may be incomplete due to unmeasured confounders. The probit model of sample selection that we used estimated the potential selection bias of our final sample and indicated that missingness was random and that our findings were not biased by attrition.

Second, our self-reported hypertension status measure may introduce some measurement bias, such as an underestimation of the prevalence of hypertension in the study sample. Notably, however, researchers have found that self-reported hypertension data are highly correlated with objective data (Stein et al., 2010). Moreover, since the hypertension question asked about *lifetime* diagnosis in Waves 5 and 6, there is a likelihood that for some of our participants, hypertension occurred before or co-occurred with violence victimization when

another confounding factor (i.e., living in a chronically stressful environment or condition) is associated with both hypertension and victimization. This chance of co-occurrence could have influenced the onset and progression of hypertension in ways we were not able to measure with the current study design. Our inclusion of covariates such as SES and chronic medical conditions in the analyses may help to address such issues; however, future studies should explore the effects that victimization may have for someone already at risk of hypertension.

It is also worth noting that the estimated prevalence of hypertension is about 3.37% among African American youth aged 12–17 (Jackson et al., 2018). On the other hand, it was estimated that nearly 30% of adults aged 18–39 were unaware of their hypertension (Paulose-Ram et al., 2017). Both findings indicate a relatively small bias in overestimating hypertension onset during young adulthood in the current study. Nevertheless, it is plausible that youth who were diagnosed with hypertension may be different than those who were not aware of their symptoms as well as those who did not seek medical assessment or treatment. Future research that includes objective measures of blood pressure may further improve the validity and enhance the generalizability of the findings.

Finally, our sample consisted of African American participants in one inner-city location that is resource-limited and exhibiting high rates of violence. Thus, our results may not be generalizable to other subpopulations or community settings. Yet, our findings may be especially relevant for adolescents living in low-resource contexts where violence may also be prevalent (Like, 2011).

Conclusion

Despite some limitations, this study has several strengths that contribute to the literature. First, prospective studies looking at-risk factors, moderators, and health outcomes at later time points are rare. Second, victimization is assessed throughout adolescence, not just one wave or set of experiences. Third, the study links psychosocial experience to physical health in an at-risk sample. The findings, therefore, have significant implications for the millions of youth exposed to violence in homes, schools, and neighborhoods, but those implications may not be known or recognized for 10 years or more. Our findings highlight the importance of preventive efforts for adolescents living in communities with high rates of violence. These youth are not only under the immediate threat of violence exposure and victimization, but also under the shadow of developing long-lasting negative physiological outcomes. Social support during adolescence provides an opportunity to mitigate risks of negative long-term outcomes among adolescents exposed to violence victimization.

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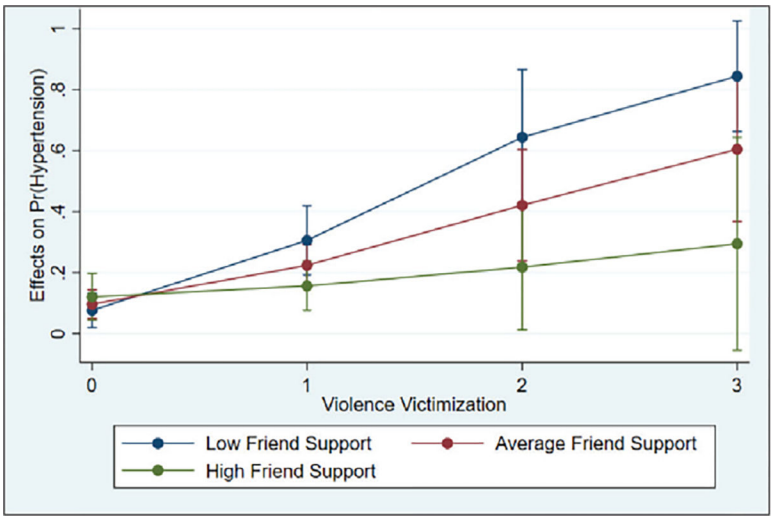

Figure 1.

Risk-protective model of friend support. The lines indicate predictive margins with 95% C.I.. For friend support, high refer to 1 standard deviation above the mean, low refers to 1 standard deviation below the mean.

Table I.

Demographics by Selected Sample and Non-selected Sample in the Final Model.

	Representing Sample in Final Model			Non-representing Sample in Final Model			
Variable	***n***	**%**		***n***	**%**		**χ^2**
Sex							14.56**
Male	133	42.63		159	42.63		
Female	179	57.37		113	57.37		
	N	Mean	SD	*n*	Mean	SD	*t*
Violent victimization (Waves 1–4)	312	0.56	0.57	272	0.61	0.57	1.17
Friends' support (Waves 1–4)	312	3.30	0.78	272	3.18	0.78	−2.03
Parent support (Waves 1–4)	312	3.81	0.78	272	3.74	0.76	−1.17
Age at Wave 1	312	14.77	0.61	272	14.88	0.61	2.05*
SES at Wave 1	312	40.04	10.58	272	39.72	10.49	−.37
Cigarette use (Waves 1–4)	312	1.50	0.90	272	1.53	0.85	.46
Cigarette use (Waves 5–8)	312	1.77	1.10	213	2.03	1.37	2.43*
5+ Drinks past 14 days (Waves 1–4)	312	0.56	0.80	272	0.62	0.91	.67
5+ Drinks past 14 days (Waves 5–8)	312	1.03	1.13	213	1.15	1.41	1.02
Depression and anxiety (Waves 1–4)	312	1.77	0.56	272	1.65	0.48	−2.72**
Depression and anxiety (Waves 5–8)	312	1.64	0.48	213	1.62	0.55	−.44

* $p < .05$,

** $p < .01$.

Table 2.

The Effects of Violent Victimization, Parental Support, and Friends' Support on Hypertension.

Main Effects[a]	**Coefficient**	**95% CI**	
Violent victimization (Waves 1–4)	.45**	.20	.70
Friends' support (Waves 1–4)	−.11	−.31	.09
Parental support (Waves 1–4)	.11	−.10	.32
Male	−.20	−.53	.13
Age at Wave 1	.02	−.22	.27
SES at Wave 1	−.01	−.03	.01
Heavy substance use (Waves 5–8)	−.03	−.11	.05
Depression and anxiety (Waves 5–8)	−.04	−.37	.29
Chronic medical conditions at Wave 5	.18	−.22	.57
Interaction Effects[b]			
Violent victimization × Friends' support	−.49*	−.95	−.02
Violent victimization × Parent support	.39	−.08	.87
Embedded Selection Model[c,d]	Coefficient	95% CI	
Perceived stress (Waves 2–4)	.03	−.16	.21
Depression (Waves 1–4)	.23	−.02	.42
Future orientation (Waves 1–4)	.33*	.13	.53
Male	−.28*	−.50	−.07
Age at Wave 1	−.10	−.27	.07
SES at Wave 1	.00	−.01	.01
Rho	.35	−.79	.96

*$p < .05$,

**$p < .01$.

[a]Model 1: Main effects only ($n = 312$, Log likelihood = −516.24). The coefficients represent main effects when interaction term was not included in the model.

[b]Model 2: Included interaction effect between violent victimization and friends support and interaction effects between violence victimization and parental support ($n = 312$, Log likelihood = −512.61).

[c]Embedded probit model of selection within Model 2.

[d]The probit model with sample selection assumes that the underlying probit models for both the selection equation and the equation of substantive interest are well-specified and that there is at least one variable (or instrument) that appears with a non-zero coefficient in the selection equation but does not appear in the equation of substantive interest. We believe that these assumptions are reasonable for our current application, based on assessments of the goodness-of-fit of the substantive probit model and the presence of future orientation as a significant predictor in the selection equation but not the substantive equation. We also assessed the robustness of our results to potential violations of these assumptions by using the vce(robust) option in Stata for the heckprob model fitting command, and we arrived at the same substantive conclusions.