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Relationships between Past Trauma Exposures and PTSD among Survivors of Intimate Partner Violence

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This study examined child abuse and community violence exposure as potential risk factors in the development of posttraumatic stress disorder (PTSD) symptoms following exposure to intimate partner violence (IPV). In a community sample of 51 primarily low-income women who had experienced IPV, childhood exposure to child abuse made a unique contribution to PTSD symptom severity from subsequent IPV. Community violence also accounted for variance in PTSD symptom severity, but in the opposite direction, with individuals exposed to community violence reporting lower levels of PTSD symptoms from IPV. These findings suggest the need for further research to identify which factors related to community violence exposure might inoculate individuals against the development of PTSD following IPV exposure.

KEYWORDS child abuse, community violence, domestic violence, PTSD, trauma type

According to the National Comorbidity Study (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), 60.7% of men and 51.2% of women reported experiencing at least one traumatic event in their lifetime, with 56% of men and 48.6% of women experiencing multiple traumas. The experience of intimate

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partner violence (IPV), defined as physical or sexual assault by a current or former spouse or romantic partner, is a particularly pervasive traumatic experience. According to findings from the National Violence against Women Survey (Tjaden & Thoennes, 2000), 22% of women reported having been victims of IPV.

The experience of IPV can result in distressing mental health outcomes, including posttraumatic stress disorder (PTSD). Rates of PTSD for individuals exposed to IPV range from 31% to 84% (Jones, Hughes, & Unterstaller, 2000). Because of the high degree of variation in the development of PTSD symptoms resulting from IPV, researchers have investigated the particular risk and protective factors that make an individual more vulnerable or resilient to PTSD. Meta-analyses have revealed a number of risk factors including gender, psychiatric history, biological factors, perceptions of life threat, peritraumatic emotional response and dissociation, and social support (Brewin, Andrews, & Valentine; 2000; Ozer, Best, Lipsey, & Weiss, 2003).

Among risk factors, the relationship between prior trauma exposure and the development of PTSD has received particular attention. Researchers have conceptualized the impact of prior trauma from two perspectives: vulnerability or inoculation. The vulnerability perspective suggests that repeated exposure to traumatic events will deplete coping resources and thus increase one’s vulnerability to subsequent traumas (Follette, Polusny, Bechtle, & Naugle, 1996; McCauley et al., 1997). In contrast, the inoculation perspective (Meichenbaum & Cameron, 1983) suggests that multiple stressful events increase familiarity with trauma leading to a perception of trauma as expectable or manageable, and thus enabling more successful adaptation if other traumatic experiences are encountered.

In the PTSD literature, a great deal of research has provided evidence for the vulnerability perspective; meta-analyses have found prior trauma exposure to be a risk factor for the development of PTSD following a subsequent trauma (Brewin et al., 2000; Ozer et al., 2003). Similar results were found in studies focusing specifically on women exposed to interpersonal violence. Hedtke et al. (2008) found the odds of meeting criteria for PTSD increased for women who had experienced multiple types of interpersonal violence (physical assault, sexual assault, or witnessing violence) over their lifetimes. In a study of undergraduates, women reporting histories of both childhood sexual abuse and adult interpersonal violence had higher levels of PTSD symptom severity than women reporting only adult exposure to physical or sexual violence (Messman-Moore, Long, & Siegfried, 2000). One exception to the vulnerability perspective comes from a study of women with adult exposure to sexual assault in which similar rates of PTSD were found for women with and without histories of childhood sexual abuse (Cloitre, Scarvalone, & Difede, 1997).

Although there is less research supporting the inoculation perspective (Meichenbaum & Cameron, 1983), which proposes an enhanced ability to
adapt in the face of traumatic experience, the inoculation model might provide a theoretical foundation for the multitude of research findings of posttraumatic growth (see Linley & Joseph, 2004, for a review). By definition, posttraumatic growth occurs when, following an adverse event, one perceives enhancements in relationships, improvements in self-concept, and changes in life outlook. For example, people who have experienced posttraumatic growth report feeling more altruistic, resilient, accepting, and appreciative following trauma exposure (Joseph & Linley, 2005).

Joseph and Linley (2005) suggested that the meaning attributed to a traumatic event is central to the cognitive and emotional processing of trauma, and therefore a key mechanism in the development of PTSD. In their analysis of posttraumatic growth, Joseph and Linley posited that following trauma, one of three processes occurs: (a) the experience is assimilated and there is a return to a pretrauma outlook on the world; (b) the experience is accommodated in a negative direction through psychopathology; or (c) the experience is accommodated in a positive direction through growth. These three potential outcomes, which represent an individual’s efforts to make meaning in ways that either maintain or shift the cognitive disruption created by a traumatic event, help to explain why some individuals with prior trauma experiences are more vulnerable to the negative effects of future traumatic events, whereas others are more resilient (i.e., inoculated).

When an individual assimilates, he or she attempts to negate or deny the traumatic event and returns to the pretrauma notion that “traumatic experiences do not happen to me,” an outlook that could make the person more vulnerable to unfavorable outcomes following future traumas (Joseph & Linley, 2005). However, an alternative response would be to assume misfortunes sometimes happen and that life should therefore be appreciated and valued. This response could potentially inoculate against the effects of later trauma as the individual’s worldview shifts to accommodate the possibility of future trauma, thereby improving one’s ability to effectively cope with future adverse events (Joseph & Linley, 2005).

Considering the inoculation and vulnerability perspectives simultaneously raises the possibility that certain traumatic experiences might be more likely to produce an adaptive or inoculating response, whereas others might be more likely to produce a maladaptive response. Maladaptive responses such as the development of PTSD symptomology, for example, might be predictable given certain types of prior exposure. There is some evidence to suggest prior trauma exposures that are similar to the current traumatic event are more predictive of a negative outcome than dissimilar experiences (Solomon & Ginzburg, 1998). Prior trauma exposure can also predict future exposure: Women who experience IPV tend to have previous experiences with interpersonal violence, often in the form of childhood abuse and witnessing of violence between caregivers (Heyman & Slep, 2002). Indeed, exposure to childhood sexual abuse and a higher number of prior traumas
have been found to increase women’s susceptibility to developing PTSD as a result of adult IPV (Astin, Ogland-Hand, Coleman, & Foy, 1995). Griffing et al. (2006) also found that childhood abuse experiences predicted PTSD symptomology following IPV in adult women and suggested that IPV experiences reactivate a sense of powerlessness and heightened state of arousal created by a history of childhood abuse. Thus, similarities in experiences might place women at greater risk for maladaptive coping and subsequent PTSD development.

Whereas the literature has provided strong evidence regarding the relationships between exposure to childhood abuse and interpersonal trauma and the development of PTSD to subsequent traumas (Astin et al., 1995; Griffing et al., 2006), the relationship between community violence exposure and development of PTSD is not as clear. Community violence is defined by Morrison (2000) as encompassing “crime-related and random acts of violence outside the home [that] does not include domestic violence or child abuse” (p. 299), and has been associated with increased rates of IPV in a nonshelter sample of low-income women (Raghavan, Mennerich, Sexton, & James, 2006). Victims of community violence and crime can develop a host of long-lasting symptoms including depression, anxiety, somatization, hostility, fear, and PTSD (Birmes et al., 2001; Kessler et al., 1995; Kilpatrick & Resnick, 1993; Norris & Kaniasty, 1994). However, there is significant variation in the degree to which community violence exposure has predicted negative outcomes. For example, in one study, whereas indirect exposure to community violence (e.g., witnessing or learning about an incident of community violence) was related to PTSD symptom severity, direct exposure was not (Walling, Eriksson, Putman, & Foy, 2011). In a separate study analyzing relative contributions of different types of trauma exposure in adolescents, violence taking place in the home was predictive of internalizing and externalizing outcomes for adolescents, whereas community violence exposure was only related to aggressive fantasies (Mrug, Loosier, & Windle, 2008). Notably, Bogat et al. (2005) found community violence failed to make a significant contribution to trauma and anxiety symptoms in a sample of women exposed to domestic violence. Applying an inoculation perspective, individuals who experience community violence might become desensitized to violence over time, making them less vulnerable to negative mental health outcomes (Ng-Mak, Salzinger, Feldman, & Stueve, 2004; White, Bruce, Farrell, & Kliewer, 1998).

This study examines two types of prior trauma exposure, child abuse and community violence, as predictors of PTSD symptom severity resulting from IPV. Given the vulnerability model and prior research documenting child abuse as a risk factor for PTSD to other traumas (Astin et al., 1995; Griffing et al., 2006; Ozer et al., 2003), we hypothesized child abuse history would predict greater PTSD symptom severity. The examination of community violence from a vulnerability or inoculation perspective is
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exploratory, as this particular type of trauma exposure has not been examined as a risk factor for the development of PTSD from IPV. However, like child abuse, community violence could be perceived as threatening, and thus similarly activating of a PTSD response. Alternatively, community violence exposure might inoculate an individual to the effects of subsequent traumas. We explored the potential relationships among PTSD symptomology, community violence, and child abuse history in a nonclinical sample of women from urban communities.

METHOD

Participants

The sample for this study included women who participated in the Family Violence Research Project at Alliant International University, a project examining the effects of IPV on mothers’ psychological health, parenting, mother–child relationships, and children’s emotional, behavioral, and neuropsychological functioning. Fifty-two women who were over the age of 18 and had experienced IPV, but were not currently in a violent partner relationship, participated in the study. For the purposes of this study, IPV was defined as physical or sexual abuse within an intimate relationship. One participant was excluded from analyses because she endorsed verbal abuse, but not physical or sexual abuse in her intimate relationship, resulting in a final sample of 51. Participants were required to have been out of the abusive relationship for at least six months. Participants were recruited through flyers placed throughout San Francisco and Oakland (at YMCAs, cafes, bus stations, community agencies, churches, colleges, housing projects) and through ads placed on Craigslist, a local online advertising Web site.

Measures

Participants met with clinicians individually and were administered the following measures.

BACKGROUND QUESTIONNAIRE

The background questionnaire, which was orally administered, included questions regarding a range of demographic variables, such as age, marital status, ethnicity, employment status, level of education, household income, and history of mental health interventions. There were several dichotomously scored items on the questionnaire about the participant’s trauma history including direct experiencing of childhood physical abuse, sexual abuse, and neglect. To assess for childhood exposure to abuse, participants were asked “Were you the victim of abuse, either physical or sexual, as a child?”
with follow-up questions to determine if the abuse qualified as a trauma meeting Criterion A1 of the PTSD diagnosis (that the abuse involved a threat of serious injury or a threat to physical integrity).

**Clinician-Administered PTSD Scale**

PTSD symptom severity was determined through the use of the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995), a structured clinical interview that assesses for the presence of the 17 symptoms for PTSD outlined in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 2000). CAPS interviews specifically assessed PTSD symptoms related to IPV (e.g., “In the past month, have you had any unpleasant dreams about the intimate partner violence?”). During these interviews a symptom was only coded if the interviewer could ascertain that the onset or exacerbation of the symptom corresponded with the time period of the IPV event being assessed. If a participant met full or subthreshold diagnostic criteria for PTSD as a result of IPV, no further assessment of PTSD symptoms to other traumas was performed. Subthreshold diagnostic criteria for PTSD was defined as meeting diagnostic criteria for Criterion B and either Criterion C or Criterion D (Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994). If a participant did not meet diagnostic criteria for PTSD because of IPV and had other traumas in her history, the CAPS was readministered, focusing on that event.

Various studies have found the CAPS to have good internal consistency (α = .94) and good test–retest reliability (.90–.98; Weathers, Blake, & Litz, 1991; Weathers, Keane, & Davidson, 2001), and is considered the highest standard in PTSD assessment (Zayfert, Becker, Unger, & Shearer, 2002). Severity of PTSD symptoms was measured by summing the frequency and intensity ratings for each PTSD symptom assessed, resulting in a total CAPS score. Higher CAPS scores are indicative of greater PTSD symptom severity. Cronbach’s alpha for the three symptom clusters of the CAPS was .85.

**Life Events Checklist**

Embedded within the CAPS is the Life Events Checklist, which lists 16 categories of potentially traumatic events. Questions assessing community violence exposure were the foci of this study’s analyses. Community violence exposure, a dichotomous variable, was coded if participants had ever experienced or witnessed physical assault or assault with a weapon in their neighborhood where the perpetrator was someone other than a family member or intimate partner. Experiencing sudden loss or serious injury to a family member because of assault or homicide by a nonfamily member was also considered community violence exposure.
CONFLICT TACTICS SCALE–2

The Revised Conflict Tactics Scale (CTS–2; Straus, 1979; Straus, Hamby, Boney-McCoy, & Sugarman, 1996) was administered verbally to determine the occurrence and frequency of IPV perpetrator behaviors toward participants during the last year of their most recent IPV relationship. The measure includes five subscales: Negotiation, Psychological Aggression, Physical Assault, Sexual Coercion, and Injury. For each question, participants were asked to indicate the frequency of occurrence of particular conflict tactics on a Likert scale ranging from 0 (never) to 7 (more than 20 times). IPV severity was determined by totaling midpoint scores for the Physical Assault subscale. Internal consistency for the CTS–2 in this study was very high ($\alpha = .89$).

Procedure

The study protocol and consent form were approved by Alliant International University’s Institutional Review Board. Participants first completed a phone screen to determine if they met inclusion criteria for the study. If participants were deemed eligible, they met with a clinical interviewer, either a clinical psychologist or a graduate student in clinical psychology, at Alliant International University in San Francisco. All interviewers received extensive training and ongoing case consultation with the second author, a clinical psychologist and experienced trauma interviewer. Written informed consent was obtained and all measures were administered orally. Participants were paid $50 for their time and for travel associated with study participation.

RESULTS

Table 1 provides complete demographic and background information for study participants. The average age of the participants was 36 years old and 88.5% of the women had completed high school, with a mean education level of 13.1 years. Eighty-eight percent of participants were women of color, primarily African American (68.6%). Sixty-five percent of the sample was living below the poverty line at the time of the study, calculated using the 2007 Federal Health and Human Services Guidelines. Poverty line was used to measure socioeconomic status (SES) based on research that has found poverty to measure economic need and strain in a way that income data alone do not (Cattaneo & DeLoveh, 2008).

Participants reported on IPV experiences that took place between six months and twelve years prior to the time of the interview. Twenty-three percent of the sample experienced IPV within one year of participating in
TABLE 1 Means, Standard Deviations, and Frequencies for Demographic Characteristics, Intimate Partner Violence (IPV) Severity, Posttraumatic Stress Disorder (PTSD) Symptom Severity, and Child Abuse and Community Violence Exposure

<table>
<thead>
<tr>
<th>Variables</th>
<th>M</th>
<th>SD</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>36.03</td>
<td>6.47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Years of completed education</td>
<td>13.01</td>
<td>1.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income (n = 50)</td>
<td>$23,842.34</td>
<td>$20,274.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Below poverty line (n = 49)</td>
<td>32</td>
<td>65.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>17</td>
<td>33.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnic background</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>35</td>
<td>68.6%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian American</td>
<td>1</td>
<td>2.0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>7</td>
<td>13.7%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian or White</td>
<td>6</td>
<td>11.8%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiracial</td>
<td>2</td>
<td>3.9%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior use of mental health services (n = 50)</td>
<td>37</td>
<td>72%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IPV severity (CTS–2 total, n = 50)</td>
<td>27.78</td>
<td>31.62</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD symptom severity (CAPS total)</td>
<td>33.54</td>
<td>24.59</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child abuse exposure</td>
<td>25</td>
<td>49%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Community violence exposure</td>
<td>29</td>
<td>56.9%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. CTS–2 = Conflict Tactics Scale–2 severity score; CAPS = Clinician-Administered PTSD Scale symptom total.

*aOne woman declined to share this information. bTwo women declined to share this information.

the study, 49% reported experiencing IPV from one to five years prior to study participation, and 27% of respondents experienced IPV more than five years prior to assessment. The most common forms of IPV included being grabbed, pushed, or slapped by a partner. The experience of severe acts of violence was strikingly prevalent in this sample, with 90% of women reporting experiences qualified as “severe” on the Conflict Tactics Scale, including being punched or hit with an object, slammed against a wall, and beaten up by a partner.

The majority of participants had high levels of violence exposure other than direct victimization exposure to IPV: 57% endorsed being exposed to community violence and 49% had experienced physical or sexual abuse as a child. Of the individuals reporting exposure to community violence, 31% of participants had been assaulted, 41% had witnessed an assault, and 31% reported that an intimate partner or family member had been murdered or seriously physically injured.

Seventeen percent of the sample met full diagnostic criteria for IPV-related PTSD. Twenty-two percent of participants endorsed subthreshold diagnostic criteria for IPV-related PTSD. To ensure that PTSD symptoms were related to IPV, PTSD symptoms were only coded if the symptom was specifically focused on IPV (e.g., intrusive thoughts related to an IPV event) or if the onset of the symptom was chronologically linked to the participant’s IPV experience.
Associations between Demographic, Independent, and Dependent Variables

First, analyses were performed to determine if any demographic variables or IPV severity were related to the independent and dependent variables in this study, and if so, would be included in subsequent analyses as covariates. Mann–Whitney $U$ analyses revealed that women exposed to childhood abuse and community violence did not differ significantly in severity of IPV compared to women who did not experience those traumas (child abuse: $U = 301.50, p = .838$; community violence: $U = 263.00, p = .379$). There were also no significant differences in time since last IPV exposure (child abuse: $U = 240.50, p = .24$; community violence: $U = 257.50, p = .46$). In addition, IPV severity ($\rho = .129, p = .373$) was not significantly correlated with PTSD symptom severity. Women exposed to community violence were significantly younger ($M = 34.5, SD = 5.1$) than women without these experiences ($M = 38, SD = 7.5$), $t(49) = .032, p = .048$; however, age was not significantly correlated with PTSD symptom severity ($\rho = .137, p = .336$). No other aspects of trauma exposure or PTSD symptom severity were related to age, education, or SES.

There were no significant differences between White women and women of color on PTSD symptom severity, SES, or severity of IPV experience. Although there were not enough participants per group to run analyses of racial group differences on categorical variables such as trauma exposure, it is notable that 16.7% of White women compared to 62.2% of women of color in the sample had experienced community violence and 33.3% of White women and 51% of women of color had experienced child abuse.

A bivariate correlation was conducted between PTSD symptom severity and number of traumatic events endorsed ($\rho = .203, p = .158$); the lack of a significant correlation suggests that PTSD symptom severity could not be explained by the overall number of traumatic events an individual had experienced. In addition, Mann–Whitney $U$ analyses were used to confirm that PTSD symptom severity did not differ based on other types of trauma exposure, such as childhood witnessing of family violence ($U = 211.00, p = .161$) and exposure to multiple IPV relationships ($U = 270.50, p = .312$).

Trauma Exposure and PTSD Symptom Severity

Assumptions required to utilize a multiple regression model were analyzed and sufficiently met. Results of the multiple regression, with exposure to child abuse and community violence as predictors and PTSD symptom severity as the criterion variable, are shown in Table 2. The model was significant, $F(2, 49) = 4.591, p = .015$, with the types of trauma exposure accounting for 12.6% of variance in PTSD symptom severity. Both child abuse
TABLE 2  Multiple Regression Predicting CAPS Score From Trauma Exposure Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>( R )</th>
<th>( R^2_{\text{adj}} )</th>
<th>( F )</th>
<th>( \beta )</th>
<th>( p )</th>
<th>( sr^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predictors</td>
<td>.401</td>
<td>.126</td>
<td>4.591*</td>
<td>.277</td>
<td>.041</td>
<td>.077</td>
</tr>
<tr>
<td>Child abuse</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exposure to community violence</td>
<td>-.284</td>
<td>.037</td>
<td>.081</td>
<td>-.284</td>
<td>.037</td>
<td>.081</td>
</tr>
</tbody>
</table>

Note. \( n = 51 \).
*\( p < .05 \).

and community violence exposure made a significant contribution to the prediction of PTSD symptom severity. Exposure to child abuse predicted higher levels of PTSD symptoms from IPV, \( \beta = .277, p = .041 \), and accounted for 7.7% of the variance. Community violence exposure also made a significant contribution to the model, in the opposite direction. Community violence exposure (\( \beta = -.284, p = .037 \)), was negatively related to severity of PTSD symptoms, and uniquely accounted for 8.1% of the variance in CAPS severity. Thus, women exposed to community violence had lower PTSD symptom severity to IPV.

DISCUSSION

This study aimed to clarify the contribution of two types of previous trauma exposure to PTSD symptom severity to subsequent IPV exposure. Prior research has suggested differential effects of trauma exposures on psychological outcomes; violence taking place in the home is more consistently related to negative psychological outcomes, whereas the contributions of community violence are more variable (Bogat et al., 2005; Mrug et al., 2008). In our study, individuals with child abuse histories reported higher levels of PTSD symptom severity related to adult IPV exposure, whereas experiences of community violence were related to lower levels of PTSD symptoms. The finding of a relationship between childhood abuse and increased levels of PTSD symptoms to subsequent IPV exposure has been documented in previous research (Astin et al., 1995), and is consistent with the vulnerability model.

The finding that community violence was negatively correlated with IPV-related PTSD symptom severity is unique and is consistent with an inoculation model. It suggests that individuals exposed to community violence might develop adaptive responses to trauma, which could in turn protect against PTSD symptom development. Indeed, previous research has indicated that individuals living in a high-violence community might adjust and become desensitized to violence over time, reporting lower levels of anxiety and internalizing symptoms (Ng-Mak et al., 2004; White et al., 1998).
The posttraumatic growth literature provides additional support for positive adaptation following exposure to trauma (Joseph & Linley, 2005).

It is possible that communities with significant violence might not have the ability to provide safety to their members, and in turn, might require individuals living in these environments to adapt and develop more resilience to the harms experienced within the community (Harvey, 1996; Harvey & Tummala-Nara, 2007). Another potential explanation for the unique finding regarding community violence exposure pertains to the particular nature of shared traumas. In contrast to traumas experienced by individuals, there are specific coping mechanisms, such as social support, rituals, and social connection, that are elicited when a trauma is shared by other individuals (Paez, Basabe, Ubillos, & Gonzalez-Castro, 2007). Overall, these types of factors have been found to increase an individual’s sense of self-efficacy in regard to successfully recovering from trauma (Schwarzer & Knoll, 2007). Although much of this research has focused on shared traumatic events such as bombings or natural disasters, more research is needed to explore specific protective factors that might serve to buffer the deleterious effects of community trauma.

Several limitations of the study should be noted. Results should be interpreted with caution given the small sample size of this study and the limitations posed by the trauma history measures. The small sample size could account for some of the surprising nonfindings in this study that are inconsistent with previous research, such as the lack of significant relationships among IPV severity, number of traumatic events endorsed, and PTSD symptom severity (Coker, Weston, Creson, Justice, & Blakeney, 2005; Scott, 2007). The retrospective nature of the reports provided, as well as the lack of additional information regarding characteristics of trauma exposure, also reflect weaknesses of this study. An inoculation theory posits there might be characteristics of the trauma exposure that support adaptive responses, but further inquiry into which factors related to trauma exposure contribute to these outcomes is needed. For example, there might be characteristics shared by the individuals exposed to community violence, as well as the communities they inhabit, such as a sense of community cohesion or access to social support, that were not captured with these data. Shared individual or community characteristics could suggest an alternative explanation for the finding that community violence exposure provided protection from the development of PTSD after experiences of IPV. Although factors associated with community violence exposure might have adaptive functions that protect an individual from developing PTSD to other traumas, there could be other negative sequelae associated with community violence exposure, such as insomnia, depression, or other types of anxiety disorders. Qualitative research exploring perceptions of and responses to IPV and community violence would also help to clarify the relationship between community violence and PTSD.
Despite its limitations, this study has potential implications for trauma research and practice. First, these findings suggest the constellation of traumas an individual has experienced could play a role in PTSD symptom severity. Clinicians should gather a full childhood trauma history for patients presenting with current trauma exposure, as these patients might be at greater risk of reactivating that prior trauma, leading to poorer psychological outcomes. In addition, the inclusion of community violence assessment in both trauma research and clinical practice is particularly needed, and could offer important information regarding the unique and overlapping effects of community violence and IPV.

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