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Cognitive–Behavioral Therapy for PTSD and Depression Symptoms Reduces Risk for Future Intimate Partner Violence Among Interpersonal Trauma Survivors

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Objective: Women who develop symptoms of posttraumatic stress disorder (PTSD) and depression subsequent to interpersonal trauma are at heightened risk for future intimate partner violence (IPV) victimization. Cognitive–behavioral therapy (CBT) is effective in reducing PTSD and depression symptoms, yet limited research has investigated the effectiveness of CBT in reducing risk for future IPV among interpersonal trauma survivors. ***Method:*** This study examined the effect of CBT for PTSD and depressive symptoms on the risk of future IPV victimization in a sample of women survivors of interpersonal violence. The current sample included 150 women diagnosed with PTSD secondary to an array of interpersonal traumatic events; they were participating in a randomized clinical trial of different forms of cognitive processing therapy for the treatment of PTSD. Participants were assessed at 9 time points as part of the larger trial: pretreatment, 6 times during treatment, posttreatment, and 6-month follow-up. ***Results:*** As hypothesized, reductions in PTSD and in depressive symptoms during treatment were associated with a decreased likelihood of IPV victimization at a 6-month follow-up even after controlling for recent IPV (i.e., IPV from a current partner within the year prior to beginning the study) and prior interpersonal traumas. ***Conclusions:*** These findings highlight the importance of identifying and treating PTSD and depressive symptoms among interpersonal trauma survivors as a method for reducing risk for future IPV.

Keywords: cognitive–behavioral therapy, interpersonal victimization, intimate partner violence, posttraumatic stress disorder, depression

Intimate partner violence (IPV) remains one of the most serious and complex public health problems faced by women in the United States (Centers for Disease Control and Prevention, 2009). National surveys indicate that approximately 25% of American women will experience IPV in their lifetime (Breiding, Black, & Ryan, 2008; Tjaden & Thoennes, 2000), and the mental health consequences of IPV place a large burden on health care systems (Rivara et al., 2007). Prevalence estimates for one of the most common consequences, posttraumatic stress disorder (PTSD), range from 31% to 84.4% among IPV survivors (Golding, 1999). Depression is also a common problem following IPV, with a weighted mean prevalence estimate of 48% among IPV survivors (Golding, 1999). Unfortunately, both PTSD and depression are often chronic in this population and can persist many years after the abuse has ended (Campbell & Soeken, 1999; Zlotnick, Johnson, & Kohn, 2006).

Given that IPV is so pervasive in the United States and that the consequences are so costly for victims, families, and society, it is imperative to develop and evaluate methods aimed at reducing its occurrence. Toward this end, numerous studies have focused on

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prevention and treatment of the male perpetrators of IPV, but such interventions have had limited success in reducing IPV (Babcock, Green, & Robie, 2004; Cornelius & Ressegui, 2007). Due to concerns related to "blaming the victim," there has been relatively limited empirical attention to characteristics of victims of IPV and to psychotherapy interventions aimed at enhancing women's safety (Cattaneo & Goodman, 2005). Yet, previous interpersonal violence in childhood and/or adulthood is a risk factor for IPV (Black, Heyman, & Slep, 2001; Coid et al., 2001; Cougle, Resnick, & Kilpatrick, 2009; Desai, Arias, Thompson, & Basile, 2002; Whitfield, Anda, Dube, & Felitti, 2003). Therefore, although victim-blaming concerns are legitimate, it is also important to identify factors that clinicians and survivors of interpersonal violence can influence in order to reduce women's risk for future IPV (Dutton, 2009; Noll, 2005).

The link between previous interpersonal violence and subsequent IPV may be due, in part, to symptoms of PTSD and depression resulting from previous interpersonal traumas (see Classen, Palesh, & Aggarwal, 2005; Messman-Moore & Long, 2003). Although much of the research to date has focused on sexual revictimization as an outcome as opposed to IPV per se, several studies have highlighted the role of PTSD in subsequent interpersonal victimization (e.g., Cloitre, Scarvalone, & Difede, 1997; Messman-Moore, Brown, & Koelsch, 2005; Risser, Hetzel-Riggin, Thomsen, & McCanne, 2006). Only a few studies have examined the prospective association between PTSD and risk for IPV victimization, and these studies have yielded somewhat discrepant findings. One longitudinal study of community women found that PTSD symptoms from a variety of traumas predicted future interpersonal victimization from a nonintimate partner but did not predict future IPV (Cougle et al., 2009). In contrast, in their longitudinal study of IPV survivors, Krause, Kaltman, Goodman, and Dutton (2006) found that PTSD symptoms significantly increased the likelihood of IPV victimization at a 1-year follow-up even after controlling for childhood abuse, length of involvement in the relationship, and baseline IPV. Similarly, Bell, Cattaneo, Goodman, and Dutton (2008) found that more severe PTSD symptoms increased women's risk for future psychological abuse at an 18-month follow-up. Finally, another longitudinal study using data from the Chicago Women's Health Study found that PTSD symptom severity predicted future IPV above and beyond the effects of previous interpersonal violence experiences, help-seeking behaviors, social support, and depression symptoms (Perez & Johnson, 2008). Thus, three of four published studies have revealed PTSD symptoms to be predictive of future IPV. The discrepant findings observed in Cougle et al.'s study may be attributable to differences in the measurement of PTSD and IPV in their study. For example, Cougle et al. coded individual PTSD symptoms as dichotomous variables (present or absent), whereas the other studies measured PTSD symptoms as continuous variables.

We theorize, consistent with the majority of empirical research, that PTSD symptoms may increase risk for IPV among interpersonal violence survivors via emotional numbing symptoms. The emotional numbing symptoms of PTSD, which involve a general suppression or analgesia of emotional responsiveness, may impede survivors' ability to detect and/or respond to actual risk (Chu, 1992; Messman-Moore & Long, 2003). Consistent with this theoretical conceptualization, Krause et al. (2006) found that emotional numbing was the only PTSD symptom cluster that significantly predicted the odds of IPV revictimization among IPV survivors.

Depression symptoms have also been implicated as a potential mental health risk factor for general revictimization. In separate studies of college women, Gidycz and colleagues (Gidycz, Coble, Latham, & Layman, 1993; Gidycz, Hanson, & Layman, 1995) found that symptoms of depression (and anxiety) preceded revictimization among college students. Similarly, Acierno, Resnick, Kilpatrick, Saunders, and Best (1999) found that depression increased women's risk of future physical assault (and PTSD) following IPV exposure. Finally, Cougle et al. (2009) found that depression, but not PTSD symptoms, predicted future IPV victimization among a large community sample of women. Depression symptoms may increase risk for IPV via a reduced cognitive and affective capacity by which to detect potential abusers, detect physical IPV triggers, and/or make decisions to avoid risk. Feelings of guilt, worthlessness, helplessness, or hopeless may also impede the termination of abusive relationships or potentially abusive relationships. Additionally, the low motivation and energy levels characteristic of depression may also interfere with one's ability to escape from dangerous situations or potentially violent relationships (Breslau, Davis, Andreski, Peterson, & Schultz, 1997; Cougle et al., 2009).

Although symptoms of PTSD and depression appear to be important mental health risk factors for subsequent interpersonal violence, there is scant research examining the impact of interventions aimed at reducing psychological distress on the risk of future victimization among at-risk populations. Of note, interpersonal violence prevention programs that focus on psychoeducation have been shown to be efficacious for women who have not already experienced interpersonal victimization but have demonstrated inconsistent results with previous survivors of interpersonal violence (Breitenbecher & Gidycz, 1998; Breitenbecher & Scarce, 2001; Hanson & Gidycz, 1993; Marx, Calhoun, Wilson, & Meyerson, 2001). Given that many women develop symptoms of PTSD and depression subsequent to experience of interpersonal violence (Acierno et al., 1999; Golding, 1999) and that PTSD and depression symptoms increase risk for future victimization, interventions aimed at reducing these symptoms may reduce the likelihood of future IPV. Fortunately, there is strong support for cognitive–behavioral therapy (CBT) in producing reductions both in PTSD and in depression symptoms among survivors of interpersonal trauma (Iverson, Lester, & Resick, 2011). However, when we reviewed the published treatment outcome literature, we could not locate a single study that examined whether reductions in PTSD and/or depression symptoms among interpersonal trauma survivors are associated with a decreased likelihood of future IPV victimization.

In order to examine the association between decreases in symptoms of PTSD and depression during CBT and risk of future IPV victimization, we utilized data from a randomized clinical trial of cognitive processing therapy (CPT) for women who were interpersonal trauma survivors with PTSD (Resick et al., 2008). CPT is a type of CBT. It is an ideal treatment with which to examine such effects because it has been shown to result in significant decreases both in PTSD and in depressive symptoms in three well-controlled trials for female interpersonal violence survivors (Chard, 2005; Resick et al., 2008; Resick, Nishith, Weaver, Astin, & Feuer, 2002). Therefore, on the basis of research and theory described

previously, we hypothesized that women who benefited significantly from treatment, as indexed by reductions in PTSD and depressive symptoms, would report less IPV victimization 6 months after treatment.

Method

Participants

This study included 150 women with PTSD secondary to an index event of sexual or physical assault in childhood and/or adulthood, age 18 and over, who participated in a larger dismantling study of CPT for PTSD (for a full description, see Resick et al., 2008). In brief, women were randomized to receive full CPT ($n = 53$), cognitive portion only (CPT-C; $n = 47$), or the written account only (WA; $n = 50$). All women met criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed; *DSM–IV*; American Psychiatric Association, 1994) for PTSD as measured by the Clinician-Administered PTSD Scale (Blake et al., 1995).

In terms of sample characteristics, women were an average of 35.4 years of age ($SD = 12.4$). Over half of the women (62%) were Caucasian, 34% were African American, and 4% identified themselves as belonging to other racial groups. On average, participants had completed 13.8 years of education ($SD = 2.8$). Approximately half (53.7%) had an annual income of less than $20,000 per year. Approximately 20% of the sample was married or cohabiting. Most of the women in this sample had experienced multiple forms of interpersonal violence victimization prior to entering the study. Approximately 84% of the sample reported adult physical assault, 80.7% reported adult sexual victimization, 78% reported child sexual abuse (60% penetrative sexual abuse), and 77% reported childhood physical abuse. The average length of time since the index event was 14 years; this is because a significant portion of participants (45.3%) reported child sexual or physical abuse as their index event. Overall, 19.3% of the intent-to-treat (ITT) sample reported IPV as their index event.

Of the 150 women in the ITT sample, 24 did not return for their first session of therapy, 126 completed one or more therapy sessions (i.e., treatment starters), and 86 completed all 12 hr of therapy (i.e., treatment completers). Women in the ITT sample completed an average of 8.0 therapy hours ($SD = 5.12$; range: 0–12 hr). Women who began treatment (i.e., treatment starters) completed an average of 9.5 hr of therapy ($SD = 5.12$, range: 1–12 hr) over the course of 7.3 weeks ($SD = 1.30$).

Measures

Standardized trauma interview. The standardized trauma interview was adapted from Resick, Jordan, Girelli, Hutter, and Marhoefer-Dvorak's (1988) treatment study. It includes both investigator-generated and standardized questionnaires to assess demographic characteristics as well as physical and sexual victimizations occurring in childhood and adulthood (full descriptions of questionnaires and psychometric properties are provided in Resick et al., 2008). Because the amount of previous interpersonal trauma may impact women's risk for future IPV (McKinney, Caetano, Ramisetty-Mikler, & Nelson, 2009), items from this interview were used to quantify the cumulative amount of interpersonal trauma exposure to be included as a covariate in the analyses. A series of four questions assessed the frequency of rape, attempted rape, other sexual assault, and physical assault that occurred in childhood (before 16th birthday) and in adulthood (after 16th birthday). Each item was rated on a Likert-type scale ranging from 0 (*never*) to 6 (*more than 20 times*). The eight responses to these items were summed to form a lifetime prior interpersonal trauma score.

Conflict Tactics Scale (CTS), Physical Aggression subscale (Straus, 1979). IPV was assessed with a modified version of the original CTS as part of the Standardized Trauma Interview, which has demonstrated reliability and validity (Straus, 1990). We administered the 8-item Physical Assault subscale plus 3 additional items to assess IPV. Respondents reported on the frequency of abusive behaviors perpetrated by their current partner and most recent previous partner. Respondents were asked about IPV perpetrated by their current partner within the past year (i.e., violence from a current partner within the past year) and IPV by a previous partner during the last year they were together. If they had multiple partners who engaged in IPV in the past, they were asked to respond in terms of the one who most frequently used IPV. IPV scores were computed by summing the number of positively endorsed items, with total scores ranging from 0 to 11. This computation method, known as the variety score, has desirable psychometric properties (Moffitt et al., 1997). Additionally, variety scores reduce skewness and estimation errors common in the recall of high-frequency behaviors and circumvent the need to weight different aggressive acts by their presumed severity. IPV scores were the maximum score both from current and from previous relationships as reported at the pretreatment assessment, resulting in two IPV variables at pretreatment: *lifetime IPV* (i.e., IPV from a previous partner at some point during one's lifetime) and *pretreatment IPV* (i.e., IPV from a current partner within the past year). The only modification to this measure at the 6-month follow-up assessment was to ask about relationships within the past 6 months as opposed to a year, resulting in an *IPV at 6-month follow-up* variable. The Physical Assault subscale of the CTS had high internal consistency, with coefficient alphas of .89 for current IPV and .88 for past IPV reported at pretreatment and .83 for IPV reported between the period of posttreatment and the 6-month follow-up.

Beck Depression Inventory—II (BDI–II; Beck, Steer, & Brown, 1996). The BDI–II consists of 21 items assessing depressive symptoms corresponding with *DSM–IV* criteria for major depressive disorder, with higher total scores indicating more severe depressive symptoms. The BDI–II has demonstrated reliability and validity in heterogeneous outpatient samples (Beck et al., 1996). The alpha coefficient for the current study was .91.

Posttraumatic Diagnostic Scale (PDS; Foa, 1995). The PDS is a 49-item self-report measure that assesses trauma history and *DSM–IV* criteria for PTSD. Respondents rate the frequency of each symptom on a scale ranging from 0 to 3, with higher scores indicating higher frequency of PTSD symptoms. The PDS includes a symptom severity score (range 0–51) that is obtained by summing an individual's responses from each item. The PDS has demonstrated reliability and validity (Foa, Cashman, Jaycox, & Perry, 1997). The alpha coefficient for the current study was .88.

Procedures

Participants were assessed at nine time points throughout the trial: at pretreatment, at six points during treatment (i.e., every week of therapy), and at a 6-month posttreatment follow-up. As noted previously, after they completed the pretreatment assessment all participants were randomly assigned to one of three CPT therapy conditions: CPT, CPT-C, or WA. Therapists included one woman with a master's degree and seven women with doctorates in clinical psychology; each therapist conducted approximately equal numbers of therapy cases in each condition. All treatment sessions were videotaped, and treatment adherence and competence were closely monitored. All three treatments were scheduled to be completed in 6 weeks and involved 12 hr of therapy. A brief description of the three treatments is provided below (for further details, see Resick et al., 2008).

CPT. CPT followed the manual as written by Resick and Schnicke (1993) and included updated forms found in Resick (2001). CPT is a structured protocol in which the primary goal of treatment is to help clients learn skills to recognize and challenge cognitive distortions, first focusing on those related to their worst traumatic events and then the meaning of the events in terms of their self, others, and the world. Therapy includes education about PTSD; identification of relationships between events, thoughts, and emotions; and the development of alternative, more balanced thinking. The full CPT package includes detailed written accounts (WA) of the index traumatic event and daily readings of these written accounts during the early and middle sessions of therapy. Cognitive therapy is used during sessions and via worksheets completed between sessions to help clients identify cognitive distortions that interfere with recovery from PTSD and to promote balanced thinking.

CPT-C. The CPT-C protocol was identical to the full CPT protocol except for the omission of the detailed writing account and readings of the written trauma account. The therapy is also trauma focused but emphasizes additional Socratic questioning and cognitive work in lieu of the account. There is an additional emphasis on cognitive skills, including further applications of event–thought–emotion worksheets for cognitive skills practice and generalization.

WA. The design of the WA protocol was developed to maintain the integrity of the written account intervention in the full CPT protocol by expanding upon the written account component of CPT (i.e., participants were asked to engage in their writing during therapy sessions and to read it back to the therapist). Sessions 1 and 2 were 60 min each and consisted of a treatment overview, PTSD psychoeducation, instructions regarding subjective units of distress (SUDS), and script construction for the index trauma. During the remaining five sessions, participants briefly met with the clinician and then spent 45–60 min writing about their index trauma and provided SUDS ratings and the beginning and end of the writing assignment, as well as ratings of strong emotions. Following the end of the written assignment, the client read the account aloud to the therapist. Following the reading, therapists made nondirective and supportive comments, provided occasional education, and probed for the client's emotions, but they did not conduct any cognitive therapy or try to challenge the client's cognitive distortions. For homework assignments, clients were asked to complete their written account if they had not during the session, read it daily, and record SUDS ratings.

Statistical Analyses

Growth curve analyses from a multilevel regression framework (Singer & Willett, 2003) produced estimates of change over time in PTSD and depressive symptoms. This approach is particularly well suited for the current data because it accommodates varying time intervals between assessment points. Similarly, data can be analyzed for all women who were randomized to treatment, regardless of whether they began treatment or provided any follow-up assessment data (i.e., ITT sample). The analyses included eight assessment points (i.e., pretreatment, every week during the 6 weeks of therapy, and posttreatment) to assess changes in PTSD and depressive symptoms. There was some variability in the timing of the assessments across participants. Therefore, time was included in the models as a variable indicating the number of days that had elapsed since the initial assessment. Although a clinician-administered assessment of PTSD symptoms (i.e., the CAPS) was available at pretreatment and posttreatment, self-reported PTSD as measured by the PDS was examined for the primary analyses because it was administered at eight assessment points (i.e., pretreatment, every week during the 6 weeks of treatment, and posttreatment). This increases power and allows for the use of multilevel regression procedures to derive precise estimates of change during the course of treatment and the associated relationships with IPV at the 6-month follow-up. Additionally, the PDS has been shown to correspond well with the CAPS (Griffin, Uhlmansiek, Resick, & Mechanic, 2004). The growth curve analyses were conducted with the hierarchical linear and nonlinear modeling software program (Raudenbush, Bryk, & Congdon, 2005) using full maximum likelihood estimation.

Multilevel regression techniques were developed to analyze nested or hierarchical data structures (Raudenbush & Bryk, 2002). For a longitudinal design, repeated assessments are nested within individuals. The repeated-measures, or within-subjects, component of the model is referred to as Level 1, whereas the between-individuals component of the model is referred to as Level 2 (person level). Most applications of growth curve analyses examine the prediction of change over time (Level 1 processes) by person level variables (Level 2 predictors). Our primary goal in the study was to examine how changes in symptoms during treatment (Level 1 processes) predicted IPV (Level 2 outcome) at the 6-month follow-up assessment occasion. Standard growth curve analyses with PTSD and depressive symptoms as outcomes were conducted, and the Level 1 empirical Bayes estimates of change parameters (initial status and change over time) that hierarchical linear modeling derived for each participant were saved into a separate data file (Raudenbush et al., 2005). These change parameters were then included as predictors of a series of multiple regression analyses to examine how change over time in PTSD and depressive symptoms during treatment predicted IPV at the 6-month follow-up assessment occasion. Because we were interested in the overall symptom reductions from pre- to posttreatment, linear change models were evaluated. This produced estimates of two change parameters for all participants: initial status

(b_0) and change over time (b_1).[1] Because hierarchical linear modeling does not produce a standardized regression coefficient, to help interpret the strength of Level 1 (i.e., within-participants) time effects, we report the change in sigma square ($\Delta\sigma^2$), which represents how much of the within-subjects variance is accounted for by time.

Of the 150 women in the ITT sample, 81% ($n = 121$) completed the 6-month follow-up assessment and therefore provided IPV outcome data. Participants who completed the 6-month follow-up assessment attended more hours of treatment than participants who did not complete follow-up assessment, $M = 8.81$ ($SD = 4.71$) vs. $M = 4.41$ ($SD = 5.34$), $F(1, 149) = 16.60$, $p < .001$. Additionally, participants who completed the follow-up assessment reported significantly lower posttreatment PTSD severity, $M = 14.35$ ($SD = 4.71$) vs. $M = 26.50$, $F(1, 117) = 6.67$, $p < .01$, and depression severity, $M = 12.68$ ($SD = 12.94$) vs. $M = 27.00$ ($SD = 12.54$), $F(1, 117) = 9.18$, $p < .01$, than participants who did not complete the follow-up. Of note, however, there were no significant differences in lifetime IPV history or IPV at pretreatment in terms of completion of the 6-month follow-up assessment. For all subsequent growth curve analyses, all available PTSD and depression symptom data from the 150 women in the ITT sample were included in the growth curve analyses regardless of whether the women dropped out of treatment or did not complete one or more assessment occasions. Percentage of treatment completed was explored as a covariate in the analyses and treatment condition (CPT, CPT-C, WA) were explored in the analyses. Neither variable was a significant predictor of IPV victimization at 6-month follow-up; therefore, both variables were excluded from the current analyses. Exposure to IPV is a risk factor for further IPV victimization (Cougle et al., 2009), and we therefore explored lifetime IPV as a predictor of IPV at the 6-month follow-up. Lifetime IPV was not found to be a significant predictor of IPV at the 6-month follow-up; therefore, it was not included in the current analyses. Finally, because recent IPV and the frequency of prior interpersonal traumas may influence the likelihood of future IPV (Krause et al., 2006; McKinney et al., 2009), we controlled for both of these exposures in all of the analyses.

Results

Approximately 61% ($n = 91$) of the 150 women in the ITT sample reported a lifetime history of IPV, and 16% ($n = 24$) reported IPV perpetrated by their current partner within the year prior to the pretreatment assessment. Of the 118 women in the ITT sample who completed the 6-month follow-up assessment, 22% ($n = 26$) reported that they experienced IPV victimization in the 6 months following treatment.

The initial growth curve analyses indicated that both PTSD, $b_1 = -.17$, $t(149) = -12.38$, $p < .001$, $\Delta\sigma^2 = .56$, and depressive symptoms, $b_1 = -.16$, $t(149) = -12.16$, $p < .001$, $\Delta\sigma^2 = .50$, exhibited significant mean-level decreases from pre- to posttreatment. The growth curve analyses also indicated significant variability across participants in change over time both in PTSD and in depressive symptoms. Figure 1 depicts mean level change in PTSD over time as well as change at one standard deviation (*SD*) above and below the mean level change. The pattern of change in depressive symptoms mirrored that of PTSD symptoms.

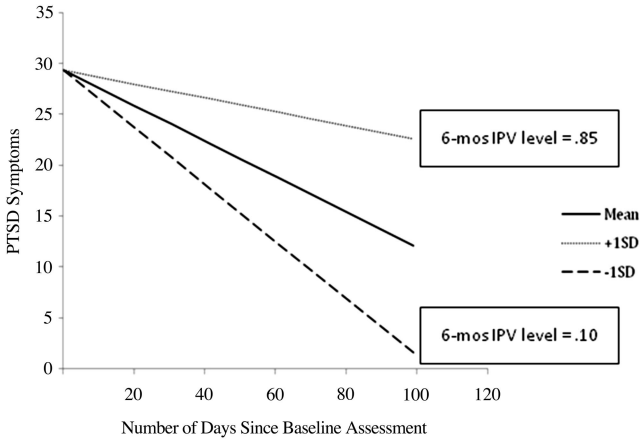

Figure 1. Depiction of the PTSD severity change model with time represented as the number of days since the pretreatment assessment. The solid line depicts the mean level of change in PTSD severity in the intent-to-treat (ITT) sample. The dashed lines are included to illustrate the variability in PTSD severity change in the ITT sample and depict change at 1 standard deviation (*SD*) above and 1 *SD* below the mean level of change. The boxes depict 6-month follow-up IPV levels for individuals exhibiting weaker (i.e., 1 *SD* above the mean, indicating a shallower decline) and stronger (i.e., 1 *SD* below the mean, indicating steeper declines) treatment responses. PTSD = posttraumantic stress disorder; IPV = intimate partner violence.

The next step of the analyses examined the impact of the change parameters from the growth curve analyses on IPV at the 6-month follow-up assessment. Descriptive statistics and bivariate correlations among study variables are presented in Table 1. PTSD and depressive symptoms were highly correlated at baseline, and change in PTSD symptom severity during treatment was also highly correlated with change in depression symptom severity. Pretreatment IPV (i.e., exposure to IPV from a current partner within the past year) was not associated with initial status (i.e., pretreatment PTSD and depression symptoms), which is likely a function of a restricted range of PTSD and depressive severity inherent in a PTSD clinical sample (i.e., ceiling effects resulting from the fact that all participants had to meet full diagnostic criteria for PTSD to be included in the parent trial). Additionally, although pretreatment IPV levels were not associated with change in PTSD or depressive symptoms during treatment, change in PTSD and depression symptoms during treatment were both associated with IPV at the 6-month follow-up assessment.

A series of hierarchical multiple regression analyses further examined the association between the PTSD and depression symptom change parameters and IPV levels at the 6-month follow-up assessment. For individual coefficients, the squared partial regression coefficient (pr^2) was calculated as an effect size indicator for significant effects, with .01, .06, and .14 as suggested cutoffs for small, medium, and large effect sizes (Kirk, 1996). As presented in

[1] Growth curve modeling conducted within a structural equation framework can include change parameters as predictors of distal outcomes. However, the current data set was not appropriate for this type of analysis because of the substantial variability in timing of assessments across participants.

Table 1
Descriptive Statistics and Correlations Among Study Variables

Variable	1	2	3	4	5	6	*M*	*SD*
1. Pretreatment IPV (CTS)	—						0.34	1.24
2. Prior interpersonal trauma	.04	—					12.5	8.90
3. PTSD initial status (β_0)	.13	.12	—				29.59	6.68
4. PTSD change over time (β_1)	−.05	−.03	.01	—			−0.18	0.10
5. Depression initial status (β_0)	.09	.03	.71*	.12	—		27.41	8.34
6. Depression change over time (β_1)	.01	.01	−.04	.82*	−.10	—	−0.16	0.10
7. Six-month follow-up IPV (CTS)	.06	.01	−.06	.27*	.03	.26*	0.50	1.29

Note. *SD* = standard deviation; IPV = intimate partner violence; CTS = Conflict Tactics Scale; PTSD = posttraumatic stress disorder; initial status = average scores at pretreatment.
* $p < .001$.

Table 2, significant associations were found between change in PTSD, $b = 3.37$, $t(118) = 3.06$, $p < .05$, $pr^2 = .07$, and depressive symptoms, $b = 3.49$, $t(118) = 2.93$, $p < .05$, $pr^2 = .07$, over time and IPV at the 6-month follow-up. Further, these associations remained significant when controlling for pretreatment IPV levels and prior exposure to interpersonal trauma.

To elucidate the nature of these effects, we evaluated the regression equations at different levels of symptom change to compute values for IPV at the 6-month follow-up assessment for individuals who responded well to treatment and for individuals who did not respond well to treatment (see boxes in Figure 1). Participants exhibiting a change in PTSD 1 *SD* below the mean (the dashed line in Figure 1 with the steeper slope, indicative of a stronger treatment response) had a corresponding value of .10 on the CTS at the 6-month follow-up IPV variable, and participants exhibiting a change in PTSD symptoms 1 *SD* above the mean (the beaded line in Figure 1 with the shallower slope, indicative of a weaker treatment response) had a corresponding value of .85 on the CTS at the 6-month follow-up IPV variable. In other words, better PTSD treatment response was associated with lower IPV levels at the 6-month follow-up; whereas those participants with weaker treatment responses reported higher levels of IPV at the 6-month follow-up. In looking at change in depression symptoms, these estimates were .28 and .95 on the CTS for individuals 1 *SD* below and above, respectively, the mean level of change in depression symptoms. Thus, individuals with stronger treatment responses on depression reported lower IPV levels at the 6-month follow-up and those with weaker treatment responses reported higher IPV levels at the 6-month follow-up. Examination of the change score in clinician-evaluated PTSD symptoms and diagnostic status from pretreatment to posttreatment revealed a pattern of results similar to those found with the self-report measure of PTSD.[2]

Discussion

To our knowledge, the current study is the first investigation of the effects of CBT for PTSD and depression symptoms on future IPV risk among interpersonal trauma survivors. Consistent with our hypothesis, women who experienced reductions in PTSD and depressive symptoms over the course of treatment reported less IPV at a 6-month follow-up relative to women who did not respond to treatment, in terms of reductions in PTSD and depression symptoms. Women who experienced improvements in PTSD and depression were less likely to report IPV at the 6-month follow-up, even after controlling for the effects of being in a current relationship with recent IPV at the pretreatment assessment and the total number of lifetime sexual and physical interpersonal traumas experienced. As one of the only studies to examine the effect of decreases in PTSD and depression symptoms on risk for future IPV among interpersonal trauma survivors, this study provides information that may aid intervention and efforts for women seeking services for interpersonal trauma.

Although in absolute numbers there were no differences from pretreatment to 6-month follow-up in the numbers of women who reported IPV, the proportion of IPV endorsed by the ITT sample at the 6-month follow-up period of the current study was quite high (22%). Some of these women represent new cases of IPV in that they had not reported IPV at pretreatment. The finding that reduction in PTSD and depression predicts lower IPV indicates that those who did not recover from their PTSD or depression were at particular risk for new IPV or IPV revictimization. Clearly, IPV victimization is a significant problem among individuals who have experienced interpersonal trauma, even among those who have recently sought mental health treatment. It is important to remember that this sample had been multiply traumatized over a long period of time, so those who did not respond to treatment may have needed longer intervention and assistance than the 12 hours of therapy that was offered in this study. Furthermore, although clinicians can help reduce women's risk for IPV by treating their PTSD and depressive symptoms, prevention of IPV also necessitates effective interventions with IPV perpetrators.

[2] The Clinician-Administered PTSD Scale (CAPS: Blake et al., 1995) was also administered pretreatment and posttreatment, and both changes in PTSD symptom severity and diagnostic status were examined. Change in PTSD symptoms severity (computed by subtracting posttreatment CAPS severity scores from pretreatment CAPS severity scores) was significantly associated with 6-month follow-up IPV levels ($r = .25$, $p < .01$), and this effect was maintained when controlling for pretreatment IPV levels. Additionally, a one-way analysis of variance with PTSD status as the factor and 6-month follow-up IPV levels as the dependent variable revealed that participants who met criteria for PTSD at posttreatment, $n = 32$, $M = 1.31$, ($SD = 2.12$), endorsed significantly higher levels of IPV at the 6-month follow-up than participants who no long met criteria for PTSD, $n = 78$, $M = 0.23$, ($SD = 0.66$), $F(1, 108) = 16.60$, $p < .01$, partial $\eta^2 = .24$. Again, a follow-up analysis of covariance confirmed that this effect was maintained when controlling for pretreatment IPV levels.

Table 2
Summary of Regression Analysis Predicting IPV at the 6-Month Assessment

Variable	*b*	95% CI	*SE* (*b*)	*B*	*t*
		PTSD			
Step 1 (R^2 = .07)					(*df* = 118)
Initial status (β_0)	−0.01	[−0.05, 0.03]	0.02	−.07	−0.75
Change over time (β_1)	3.37	[1.21, 5.53]	1.10	.27	3.06*
Step 2 (R^2 = .07)					(*df* = 116)
Initial status (β_0)	−0.02	[−0.06, 0.02]	0.02	−.08	−0.88
Change over time (β_1)	3.45	[1.27, 5.63]	1.11	.28	3.11*
Pretreatment IPV	0.08	[−0.10, 0.26]	0.09	.08	0.89
Prior interpersonal trauma	.01	[−0.01, 0.03]	0.01	.03	0.35
		Depression			
Step 1 (R^2 = .08)					(*df* = 118)
Initial status (β_0)	0.01	[−0.01, 0.03]	0.01	.06	0.67
Change over time (β_1)	3.49	[1.18, 5.80]	1.18	.26	2.93*
Step 2 (R^2 = .08)					(*df* = 116)
Initial status (β_0)	0.01	[−0.01, 0.03]	0.01	.06	0.62
Change over time (β_1)	3.47	[1.12, 5.82]	1.20	.26	2.90*
Pretreatment IPV	0.05	[−0.13, 0.23]	0.09	.05	0.57
Prior interpersonal trauma	0.01	[−0.01, 0.03]	0.01	.01	0.09

Note. IPV = intimate partner violence; PTSD = posttraumatic stress disorder; *b* = unstandardized regression coefficient; *SE* (*b*) = the standard error of the unstandardized regression coefficient; *B* = the standardized regression coefficient; *df* = degrees of freedom.
* $p < .05$.

The current findings add support for the growing consensus that mental health symptoms following interpersonal trauma are associated with risk of future victimization (Cattaneo & Goodman, 2005; Classen et al., 2005; Messman-Moore & Long, 2003). How might PTSD and depression symptoms confer risk for IPV among interpersonal trauma survivors? Presumably, certain PTSD-related symptoms of (i.e., dissociation and numbing) and depression (i.e., depressed mood and/or lack of energy) can be adaptive in the short run (i.e., to reduce painful emotions, thoughts, bodily sensations and memories; Follette, Iverson, & Ford, 2009; Resick & Schnicke, 1992), but such symptoms may reduce safety in relationships in the long-run (Chu, 1992). As we posited earlier, the emotional numbing symptoms of PTSD may put women at risk for IPV because these symptoms are directly related to reduced reactivity and distress, including anticipatory anxiety associated with danger cues, thereby reducing a survivor's ability to detect and/or respond to actual risk (Chu, 1992; Krause et al., 2006; Messman-Moore & Long, 2003). Although women should never be blamed for the IPV they experience, it is also possible that anger and/or emotion dysregulation, common responses associated with PTSD among interpersonal trauma survivors (Cloitre et al., 1997; Orth, Cahill, Foa, & Maercker, 2008), may increase women's risk for high levels of interpersonal conflict and IPV.

In a similar manner, depression symptoms may compromise cognitive and affective capacity, interfering with the ability to identify and avoid risky partners or situations or believe one is worthy of better treatment from one's partner. The low motivation levels characteristic of depression may also inhibit escape from dangerous situations or potentially violent relationships (Breslau et al., 1997; Cougle et al., 2009). The symptoms of depression, including feelings of guilt, worthlessness, helplessness, or hopeless, may also interfere with a woman's ability to pick safe partners or impede the termination of an abusive relationship. Although the mechanisms remain unknown, both PTSD and depressive symptoms may cause or exacerbate difficulties in the ability to adequately recognize risk and interfere with safety behaviors in intimate relationships. Moreover, suffering from PTSD and/or depression symptoms may make a woman look confused or distracted, marking her as a vulnerable target to potential abusers (Cloitre & Rosenberg, 2006).

It is also possible that other factors associated with CBT may have played a role in IPV risk reduction. It is plausible that living without significant symptoms of PTSD and depression may positively influence choices and dynamics in intimate relationships through access to more internal and external resources (Hobfoll, 1989). For example, successful CBT may have led to positive outcomes, such as more accurate perceptions of situations, increased safety behaviors, sense of self- and communal-efficacy, and hope. Although not examined in the current study, improvements in women's sense of empowerment, posttraumatic growth, self-esteem, emotion regulation abilities, as well as reductions in dissociation, self-blame, guilt, anger, and alcohol use may have impacted women's relationship decisions. Finally, reduced tolerance of IPV and increased assertiveness (e.g., "If you ever hit me I will leave you") may also impact the likelihood of future IPV. It is essential to examine potential mediators that explain why CBT for PTSD helps reduce risk for future IPV among interpersonal trauma survivors. Such future research will have important implications for the development and augmentation of targeted prevention and treatment efforts with interpersonal trauma survivors. Many interpersonal trauma survivors live with undiagnosed PTSD and depression and could benefit from our greater understanding of these complex relationships if this can be translated into effective interventions (Dutton, 2009).

Regardless of the exact mechanisms by which CBT for PTSD reduces IPV risk, the current findings suggest that enhancing

available community and social services to include CBT interventions for PTSD and depression symptoms will improve interpersonal trauma survivors' safety in intimate relationships. Therefore, outreach efforts aimed at identifying and treating individuals who experience PTSD and/or depression subsequent to interpersonal trauma may help to reduce the numerous cases of IPV that occur each year. Although research has shown that psychoeducation is helpful in reducing victimization among women who have not experienced interpersonal trauma (Breitenbecher & Gidycz, 1998; Breitenbecher & Scarce, 2001; Hanson & Gidycz, 1993), treatment aimed at reducing mental health symptoms may be essential in promoting the well-being and future safety of women who experience PTSD and/or depression subsequent to an interpersonal trauma. Given the promising findings of the current study, it is important to next examine how the current interventions and other treatment models (Iverson, Shenk, & Fruzzetti, 2009; Johnson & Zlotnick, 2006; Kubany et al., 2004) can be implemented and disseminated in community settings where such mental health services are immensely needed not only to improve women's health but also to reduce women's risk for IPV (Dutton, 2009). It is essential, however, that therapists who deliver psychotherapy to interpersonal trauma survivors be aware of the high risk for IPV among this population and consider the unique context of ongoing or future interpersonal violence during assessment and treatment.

The findings from this study should be interpreted in light of several limitations. First, this study included only women who reported an interpersonal assault as their index traumatic event. Thus, it is unclear whether the current results would generalize to men or to individuals suffering from PTSD secondary to other types of trauma (i.e., motor vehicle accidents, natural disasters, or combat). Second, all three groups received an empirically supported treatment for PTSD (Resick et al., 2008), and we therefore cannot be certain that gains were related to treatment instead of the passage of time alone, repeat assessments, or clinician attention. However, research clearly shows that PTSD and depression symptoms are chronic when left untreated (Campbell & Soeken, 1999; Resick et al., 2002; Zlotnick et al., 2006). Additionally, CPT has been found to effectively reduce PTSD and depressive symptoms in several clinical trials employing comparison treatment conditions and wait-list controls (e.g., Chard, 2005; Resick et al., 2002). Thus, it is unlikely that women would have experienced significant reductions in PTSD and depression symptoms without active treatment, especially given that the average length of time since the index event was 14 years, but it would have been ideal to compare these treatments to a minimal attention control condition in order to conduct a stronger test of the effect of CBT on reducing future risk for IPV. Moreover, women who reported higher PTSD and depression symptoms at posttreatment were less likely to attend the 6-month follow-up assessment. Therefore, they may have been more likely to have experienced IPV, and we cannot be sure that the current results would apply to those with high levels of distress who did not complete the follow-up assessment portion of the study.

A third limitation of this study is that it was not possible to examine simultaneously the effects of decreased PTSD and depression symptoms on IPV victimization in the same regression model due to the collinearity ($r = .82$) between these variables. Fourth, the current study did not include measures of sexual and emotional IPV, and therefore the study findings may not generalize beyond the reduction of physical IPV. Finally, although a strength of the current study is the use of statistically sophisticated methods to elucidate the prospective effect of psychiatric symptoms as risk factors for IPV; a more complete understanding of IPV risk necessitates the examination of the characteristics and processes of IPV perpetrators as well.

Given the promising findings of the current study, future research is warranted to investigate further the efficacy of psychosocial treatments for PTSD and depression symptoms for reducing risk for other forms of retraumatization (i.e., physical and sexual assaults from a nonintimate partner). Replication of the present findings with a larger, more heterogeneous sample is needed to bolster results. Also, a longer follow-up period is needed to fully assess the impact of treatment on long-term risk for IPV victimization. Sexual coercion and psychological abuse from an intimate partner should also be examined in future investigations. Similarly, future studies should examine the extent to which other positive outcomes associated with treatment (i.e., increased self-esteem or posttraumatic growth) and women's use of other types of community interventions (i.e., victim advocacy, legal actions) may also play a role in reducing interpersonal trauma survivors' risk for future IPV. Finally, dissemination and implementation research is needed to understand how psychosocial treatments, such as CPT, transfer to community and social services settings to reduce psychological distress and revictimization (Dutton, 2009).

In conclusion, women who experience IPV may be involved in a vicious cycle of abuse, with interpersonal victimization leading to psychiatric distress in the form of PTSD and depression, followed by additional victimization. Our results suggest that that treating PTSD and depression symptoms among interpersonal trauma survivors can function to reduce risk for future IPV and interrupt or prevent this cycle of abuse. Although responsibility for IPV always rests with the perpetrator, it remains essential to continue to identify risk factors that can be intervened upon to promote risk reduction for future IPV among interpersonal trauma survivors. Such intervention and prevention programs could have an impact on public health and may help to reduce the staggering financial costs associated with IPV through the reduction of its occurrence and its negative health consequences.

References

Acierno, R., Resnick, H., Kilpatrick, D. G., Saunders, B., & Best, C. L. (1999). Risk factors for rape, physical assault, and posttraumatic stress disorder in women: Examination of differential multivariate relationships. *Journal of Anxiety Disorders, 13,* 541–563. doi:10.1016/S0887-6185(99)00030-4

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Babcock, J. C., Green, C. E., & Robie, C. (2004). Does batterers' treatment work? A meta-analytic review of domestic violence treatment. *Clinical Psychology Review, 23,* 1023–1053. doi:10.1016/j.cpr.2002.07.001

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory.* San Antonio, TX: Psychological Corporation.

Bell, M. E., Cattaneo, L. B., Goodman, L. A., & Dutton, M. A. (2008). Assessing the risk of future psychological abuse: Predicting accuracy of battered women's predictions. *Journal of Family Violence, 23,* 69–80. doi:10.1007/s10896-007-9128-5

Black, D. A., Heyman, R. E., & Slep, A. M. S. (2001). Risk factors for male-to-female partner sexual abuse. *Aggression and Violent Behavior, 6,* 269–280. doi:10.1016/S1359-1789(00)00026-4

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., & Keane, T. M. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress, 8,* 75–90. doi:10.1002/jts.2490080106

Breiding, M. J., Black, M. C., & Ryan, G. W. (2008). Prevalence and risk factors of intimate partner violence in eighteen U.S. states/territories, 2005. *American Journal of Preventive Medicine, 34,* 112–118. doi:10.1016/j.amepre.2007.10.001

Breitenbecher, K. H., & Gidycz, C. A. (1998). An empirical evaluation of a program designed to reduce the risk of multiple sexual victimization. *Journal of Interpersonal Violence, 13,* 472–488. doi:10.1177/088626098013004004

Breitenbecher, K. H., & Scarce, M. (2001). An evaluation of the effectiveness of a sexual assault education program focusing on psychological barriers to resistance. *Journal of Interpersonal Violence, 16,* 387–407. doi:10.1177/088626001016005001

Breslau, N., Davis, G. C., Andreski, P., Peterson, E. L., & Schultz, L. R. (1997). Sex differences in posttraumatic stress disorder. *Archives of General Psychiatry, 54,* 1044–1048.

Campbell, J. C., & Soeken, K. L. (1999). Women's responses to battering over time: An analysis of change. *Journal of Interpersonal Violence, 14,* 21–40. doi:10.1177/088626099014001002

Cattaneo, L. B., & Goodman, L. A. (2005). Risk factors for reabuse in intimate partner violence: A cross-disciplinary critical review. *Trauma, Violence, & Abuse, 6,* 141–175. doi:10.1177/1524838005275088

Centers for Disease Control and Prevention. (2009). Understanding intimate partner violence: Fact sheet. Retrieved from http://www.cdc.gov/violenceprevention/pdf/IPV_factsheet-a.pdf

Chard, K. M. (2005). An evaluation of cognitive processing therapy for the treatment of posttraumatic stress disorder related to childhood sexual abuse. *Journal of Consulting and Clinical Psychology, 73,* 965–971. doi:10.1037/0022-006X.73.5.965

Chu, J. A. (1992). The revictimization of adult women with histories of childhood abuse. *Journal of Psychotherapy Practice and Research, 1,* 259–269.

Classen, C. C., Palesh, O. G., & Aggarwal, R. (2005). Sexual revictimization: A review of the empirical literature. *Trauma, Violence, & Abuse, 6,* 103–129. doi:10.1177/1524838005275087

Cloitre, M., & Rosenberg, A. (2006). Sexual revictimization: Risk factors and prevention. In V. M. Follette & J. I. Ruzek (Eds.), *Cognitive–behavioral therapies for trauma* (2nd ed., pp. 321–361). New York, NY: Guilford Press.

Cloitre, M., Scarvalone, P., & Difede, J. (1997). Posttraumatic stress disorder, self- and interpersonal dysfunction among sexually retraumatized women. *Journal of Traumatic Stress, 10,* 437–452. doi:10.1002/jts.2490100309

Coid, J., Petruckevitch, A., Feder, G., Chung, W., Richardson, J., & Moorey, S. (2001). Relation between childhood sexual and physical abuse and risk of revictimization in women: A cross sectional survey. *Lancet, 358,* 450–454. doi:10.1016/S0140-6736(01)05622-7

Cornelius, T. L., & Ressegui, N. (2007). Primary and secondary prevention programs for dating violence: A review of the literature. *Aggression and Violent Behavior, 12,* 364–375. doi:10.1016/j.avb.2006.09.006

Cougle, J. R., Resnick, H., & Kilpatrick, D. G. (2009). A prospective examination of PTSD symptoms as risk factors for subsequent exposure to potentially traumatic events among women. *Journal of Abnormal Psychology, 118,* 405–411. doi:10.1037/a0015370

Desai, S., Arias, I., Thompson, M. P., & Basile, K. C. (2002). Childhood victimization and subsequent adult revictimization assessed in a nationally representative sample of women and men. *Violence and Victims, 17,* 639–653. doi:10.1891/vivi.17.6.639.33725

Dutton, M. A. (2009). Pathways linking intimate partner violence and posttraumatic disorder. *Trauma, Violence, & Abuse, 10,* 211–224. doi:10.1177/1524838009334451

Foa, E. B. (1995). *Posttraumatic Stress Diagnostic Scale manual.* Minneapolis, MN: National Computer Systems.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment, 9,* 445–451. doi:10.1037/1040-3590.9.4.445

Follete, V. M., Iverson, K. M., & Ford, J. (2009). Contextual behavioral treatment for complex PTSD. In C. Courtois & J. D. Ford (Eds.), *Treating complex traumatic stress disorders: An evidence-based clinician's guide* (pp. 264–285). New York, NY: Guilford Press.

Gidycz, C. A., Coble, C. N., Latham, L., & Layman, M. J. (1993). Sexual assault experience in adulthood and prior victimization experiences: A prospective analysis. *Psychology of Women Quarterly, 17,* 151–168. doi:10.1111/j.1471-6402.1993.tb00441.x

Gidycz, C. A., Hanson, K., & Layman, M. J. (1995). A prospective analysis of the relationship among sexual assault experiences: Extension of prior findings. *Psychology of Women Quarterly, 19,* 5–29. doi:10.1111/j.1471-6402.1995.tb00276.x

Golding, J. M. (1999). Intimate partner violence as a risk factor for mental disorders: A meta-analysis. *Journal of Family Violence, 14,* 99–132. doi:10.1023/A:1022079418229

Griffin, M. G., Uhlmansiek, M. H., Resick, P. A., & Mechanic, M. B. (2004). Comparison of the Posttraumatic Stress Disorder Scale versus the Clinician-Administered Posttraumatic Stress Disorders Scale in domestic violence survivors. *Journal of Traumatic Stress, 17,* 497–503. doi:10.1007/s10960-004-5798-4

Hanson, K. A., & Gidycz, C. A. (1993). Evaluation of a sexual assault prevention program. *Journal of Consulting and Clinical Psychology, 61,* 1046–1052. doi:10.1037/0022-006X.61.6.1046

Hobfoll, S. E. (1989). Conservation of resources: A new attempt at conceptualizing stress. *American Psychologist, 44,* 513–524. doi:10.1037/0003-066X.44.3.513

Iverson, K. M., Lester, K., & Resick, P. A. (2011). Psychosocial treatments. In D. M. Benedek & G. H. Wynn (Eds.), *Clinical manual for the management of PTSD* (pp. 157–203). Arlington, VA: American Psychiatric Publishing.

Iverson, K. M., Shenk, C., & Fruzzetti, A. E. (2009). Dialectical behavior therapy adapted for women victims of domestic abuse: A pilot study. *Professional Psychology: Research and Practice, 40,* 242–248. doi:10.1037/a0013476

Johnson, D. M., & Zlotnick, C. (2006). A cognitive–behavioral treatment for battered women with PTSD in shelters: Findings from a pilot study. *Journal of Traumatic Stress, 19,* 559–564. doi:10.1002/jts.20148

Kirk, R. E. (1996). Practical significance: A concept whose time has come. *Educational and Psychological Measurement, 56,* 746–759. doi:10.1177/0013164496056005002

Krause, E. D., Kaltman, S., Goodman, L., & Dutton, M. A. (2006). Role of distinct PTSD symptoms in intimate partner reabuse: A prospective study. *Journal of Traumatic Stress, 19,* 507–516. doi:10.1002/jts.20136

Kubany, E. S., Hill, E. E., Owens, J. A., Iannce-Spencer, C., McCaig, M. A., & Tremayne, K. J. (2004). Cognitive trauma therapy for battered women with PTSD (CTT-BW). *Journal of Consulting and Clinical Psychology, 72,* 3–18. doi:10.1037/0022-006X.72.1.3

Marx, B. P., Calhoun, K. S., Wilson, A. E., & Meyerson, L. A. (2001). Sexual revictimization prevention: An outcome evaluation. *Journal of Consulting and Clinical Psychology, 69,* 25–32. doi:10.1037/0022-006X.69.1.25

McKinney, C. M., Caetano, R., Ramisetty-Mikler, S., & Nelson, S. (2009). Childhood family violence and perpetration and victimization of intimate partner violence: Findings from a national population-based study of couples. *Annals of Epidemiology, 19,* 25–32. doi:10.1016/j.annepidem.2008.08.008

Messman-Moore, T. L., Brown, A. L., & Koelsch, L. E. (2005). Posttraumatic symptoms and self-dysfunction as consequences and predictors of

sexual revictimization. *Journal of Traumatic Stress, 18,* 253–261. doi: 10.1002/jts.20023

Messman-Moore, T. L., & Long, P. J. (2003). The role of childhood sexual abuse sequelae in the sexual revictimization of women: An empirical review and theoretical reformulation. *Clinical Psychology Review, 23,* 537–571. doi:10.1016/S0272-7358(02)00203-9

Moffitt, T. E., Caspi, A., Krueger, R. F., Magdol, L., Margolin, G., Silva, P. A., & Sydney, R. (1997). Do partners agree about abuse in their relationship? A psychometric evaluation of interpartner agreement. *Psychological Assessment, 9,* 47–56. doi:10.1037/1040-3590.9.1.47

Noll, J. G. (2005). Does childhood sexual abuse set in motion a cycle of violence against women? What we know and what we need to learn. *Journal of Interpersonal Violence, 20,* 455–462. doi:10.1177/0886260504267756

Orth, U., Cahill, S. P., Foa, E. B., & Maercker, A. (2008). Anger and posttraumatic stress disorder symptoms in crime victims: A longitudinal analysis. *Journal of Consulting and Clinical Psychology, 76,* 208–218. doi:10.1037/0022-006X.76.2.208

Perez, S., & Johnson, D. M. (2008). PTSD compromises battered women's future safety. *Journal of Interpersonal Violence, 23,* 635–651. doi: 10.1177/0886260507313528

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2nd ed.). Thousand Oaks, CA: Sage.

Raudenbush, S., Bryk, A., & Congdon, R. (2005). HLM 6: Hierarchical linear and nonlinear modeling (Version 6) [Computer software]. Lincolnwood, IL: Scientific Software International.

Resick, P. A. (2001). *Cognitive processing therapy: Generic manual.* Unpublished manuscript, University of Missouri—St. Louis.

Resick, P. A., Galovski, T. E., Uhlmansiek, M. O., Scher, C. D., Clum, G., & Young-Xu, Y. (2008). A randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. *Journal of Consulting and Clinical Psychology, 76,* 243–258. doi:10.1037/0022-006X.76.2.243

Resick, P. A., Jordan, C. G., Girelli, S. A., Hutter, C. K., & Marhoefer-Dvorak, S. (1988). A comparative outcome study of behavioral group therapy for sexual assault victims. *Behavior Therapy, 19,* 385–401. doi:10.1016/S0005-7894(88)80011-X

Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of cognitive processing therapy, prolonged exposure and a waiting condition for the treatment of posttraumatic stress disorder in female rape victims. *Journal of Consulting and Clinical Psychology, 70,* 867–879. doi:10.1037/0022-006X.70.4.867

Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology, 60,* 748–756.

Risser, H. J., Hetzel-Riggin, M. D., Thomsen, C. J., & McCanne, T. R. (2006). PTSD as a mediator of sexual revictimization: The role of reexperiencing, avoidance, and arousal symptoms. *Journal of Traumatic Stress, 19,* 687–698. doi:10.1002/jts.20156

Rivara, F. P., Anderson, M. L., Fishman, P., Bonomi, A. E., Reid, R. J., Carrell, D. Y., & Thompson, R. S. (2007). Healthcare utilization and costs for women with a history of intimate partner violence. *American Journal of Preventive Medicine, 32,* 89–96. doi:10.1016/j.amepre.2006.10.001

Singer, J., & Willett, J. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence.* New York, NY: Oxford University Press.

Straus, M. A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics (CT) Scales. *Journal of Marriage and the Family, 41,* 75–88. doi:10.2307/351733

Straus, M. A. (1990). *Measuring intrafamily conflict and violence: The Conflict Tactics (CT) Scales.* New Brunswick: NJ: Transaction.

Tjaden, P., & Thoennes, N. (2000). *Extent, nature, and consequences of intimate partner violence: Findings from the National Violence Against Women survey.* Washington, DC: Department of Justice.

Whitfield, C. L., Anda, R. F., Dube, S. R., & Felitti, V. J. (2003). Violent childhood experiences and the risk of intimate partner violence in adults: Assessment in a large health maintenance organization. *Journal of Interpersonal Violence, 18,* 166–185. doi:10.1177/0886260502238733

Zlotnick, C., Johnson, D. M., & Kohn, R. M. (2006). Intimate partner violence and long-term psychosocial functioning in a national sample of American women. *Journal of Interpersonal Violence, 21,* 262–275. doi:10.1177/0886260505282564

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