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Changes in Anger in Relationship to Responsivity to PTSD Treatment

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Abstract

This study examined the clinical course of different dimensions of anger and their relationship to change in posttraumatic stress disorder (PTSD) in a sample of 139 female survivors of interpersonal violence suffering from PTSD. Specifically, this study evaluated differences in the rates of change in anger dimensions by responsivity to treatment status (responders, non-responders, and drop-outs). Responders and non-responders did not differ in rate of change on state anger and anger directed inward, suggesting that treatment led to improvements in these dimensions of anger regardless of final PTSD diagnosis. Responders did evidence statistically significantly more change in trait anger and control over one's anger than did the non-responders, suggesting that changes in these dimensions of anger may be related to recovery from PTSD. Individuals who terminated therapy prematurely did not experience the same gains in state anger, trait anger, or anger-in as those who completed treatment. Differences in rates of change (linear versus quadratic growth patterns), particularly with respect to continued improvement in anger following treatment completion are discussed.

Keywords

anger; PTSD; psychotherapy outcomes; cognitive therapy; sexual assault

The Role of Anger in Responsivity to PTSD Treatment

Posttraumatic stress disorder (PTSD) has been classified as an anxiety disorder in the *Diagnostic and Statistical Manual-IV* (DSM-IV; American Psychiatric Association [APA], 1994), but there is little doubt that a variety of emotions, including anger, can dominate the

clinical presentation of trauma survivors suffering from PTSD (Resick & Miller, 2009). Several studies have reported positive associations between general anger and PTSD in the trauma literature (e.g., Jakupcak et al., 2007; Novaco & Chemtob, 2002). However, anger is most typically conceptualized as a complex construct with multiple dimensions that are composed of specific cognitive, behavioral, motivational, and physiological components which may interact to influence its expression (Izard, 1991). Researchers (e.g., Deffenbacher et al., 1996) have described the presentation of anger as a current experience (state anger) or as a characteristic tendency to respond with anger (trait anger). The directional nature of the emotion further defines the general construct such that anger-in represents suppression of emotion and anger-out reflects an outward expression of anger (such as aggressive behavior; Funkenstein, King, & Drolette, 1954). A final dimension, anger control (one's success in exerting control over the experience and expression of anger), has developed based on factor analytic analyses of anger data (e.g., Spielberger, 1988). Each of these dimensions increases our understanding of an individual's experience of anger. Recent studies have distinguished the role of these dimensions of anger more specifically in the development, maintenance, and treatment of PTSD.

Two meta-analyses summarize the relationship between anger and PTSD with particular attention to the association of the specific dimensions of anger and PTSD. First, Orth and Wieland (2006) reported that overall anger and hostility were significantly associated with PTSD, with an average effect size of 0.48. Upon further examination, large effect sizes were reported for both anger directed inward (anger-in) and control over one's anger (anger control), while a medium effect size was obtained for anger directed outward (anger-out; Orth & Wieland, 2006). More recently, a second meta-analysis reported that the relationship between anger and PTSD was significantly greater than the relationships between other anxiety disorders and anger, supporting a unique relationship between anger and PTSD (Olatunji, Ciesielski, & Tolin, 2010). Consistent with previous findings, the relationships of PTSD with anger-out, anger-in, and anger control were specifically identified as stronger than for any other anxiety disorders.

Thus, it is fairly well established that general anger is associated with PTSD, and it appears from meta-analytic studies that anger-out, anger-in, and anger control may be specifically and uniquely associated with posttrauma responses. This is not entirely surprising as anger is indeed one of the seventeen symptoms of PTSD. The DSM-IV describes the PTSD symptom of anger as "irritability or outbursts of anger" (APA, 1994, p. 428) and the Clinician Administered PTSD Scale (the gold standard instrument for diagnosing PTSD) queries the symptom of anger as "Have there been times when you have felt especially irritable or showed strong feelings of anger? How strong was your anger? How long did it take you to calm down? Did your anger cause you any problems?" (Blake et al., 1998, p. 10). Thus, the PTSD symptom of anger emerges as a general construct of anger and irritability, with little information regarding the expression of the anger (inward or outward), the ability to control one's anger, or consideration of the characterological versus transient nature of the anger (trait or state). In light of the demonstrated robust relationship between anger and PTSD and the evidence suggesting that specific dimensions of anger may be dominant in posttraumatic distress, further consideration of the role of anger in trauma recovery may advance our understanding of PTSD. Understanding the course of anger

recovery within the context of trauma-focused interventions for PTSD may aid clinicians in prioritizing anger-related cognitions and behaviors through the course of therapy, increase retention of clients in therapy and enhance overall outcomes.

Although anger is commonly associated with male combat veterans, the previously described meta-analyses included a range of trauma samples. Approximately 40% of the clinical participants included in Olatunji and colleagues' meta-analysis were female, and findings revealed stronger anger effect sizes in PTSD samples with a greater percentage of female participants. Within Orth and colleagues' meta-analysis of anger and hostility, approximately 30% of the participants were female, and the percentage of female participants was not a significant predictor of the effect size of anger. Orth and Maercker (2009) investigated anger and PTSD in crime victims (sexual and nonsexual assault), 67% of which were women. Findings revealed that anger at the perpetrator was the most commonly reported target of anger, followed by anger at the self. Both were significant predictors of PTSD. Together, data suggests that the examination of anger and PTSD is warranted across PTSD groups, including female interpersonal violence victims.

Emotional processing theory posits that the presence of anger may impede PTSD treatment response due to interference in the reduction of the fear. Foa and colleagues (1995) suggest that anger is a type of avoidant coping and that individuals may develop a tendency to respond with anger as a strategy for avoiding fear and emotional pain. Alternatively, Novaco and Chemtob (2002) conceptualize anger within PTSD as survival-mode functioning in which anger is activated by perceptions of threat, with the survival mode function then taking priority over other cognitive processing. The anger response may bias the individual's information processing so that threat-consistent information receives priority while inconsistent information is overlooked. From either theoretical perspective, if anger increases rigid maintenance of cognitive biases, clinically elevated levels of anger may interfere with recovery.

Anger and PTSD Treatment Outcome

While successful treatments for PTSD have been identified, a review of randomized controlled trials for PTSD treatments indicates roughly half of participants are treatment responders (complete treatment and no longer PTSD positive). Approximately one quarter of the samples are treatment dropouts (do not complete treatment) and one-third are treatment non-responders (complete treatment but retain PTSD diagnosis or symptoms) (Bradley, et al. 2005). A number of studies have assessed the relationship between initial levels of anger and PTSD treatment outcome, particularly with treatment non-response. For example, Forbes and colleagues (2003) found that level of anger significantly predicted poor treatment response to group cognitive behavior therapy (CBT) for veterans (gender not specified, but likely predominantly male), such that those with high PTSD and low anger at baseline fared better than those with high PTSD and high anger at baseline. Similarly, Owens, Chard, and Cox (2008) administered cognitive processing therapy (CPT) to veterans (80% men) in combined group and individual format and identified an interaction between anger and initial PTSD severity. Their results suggest that the combination of severe PTSD and high anger may predict treatment non-response. When specific dimensions of anger

(including state, trait, anger-in, anger-out, and anger control) were assessed as predictors of response to prolonged exposure (PE) for two samples of predominantly female survivors of mixed trauma-types (Group 1: 41% men; Group 2: 38% men), only anger control and anger-in were positively associated with PTSD at post-treatment (i.e., treatment non-response; van Minnen, Arntz, & Keijsers, 2002).

Other studies have failed to demonstrate a link between anger and treatment non-response. Stapleton and colleagues (2006) compared individual format exposure therapy, eye movement desensitization and reprocessing (EMDR), and relaxation training in a mixed trauma sample (25% men) and found that initial levels of anger (trauma-related and trait) and guilt were not predictive of treatment outcome. Consistently, Cahill and colleagues (2003) administered PE, stress inoculation training (SIT), and combined PE/SIT in individual sessions to female assault survivors and found that baseline state anger did not predict post-treatment symptoms when pre-treatment scores were controlled. In summary, inconsistent, extant research suggests that initial anger (when generally defined) is associated with treatment non-response, particularly in the case of severe PTSD symptoms and, perhaps, more predominantly in male veteran samples. When considering specific anger dimensions, baseline lower levels of anger control (indicating greater impairment) may account for much of that treatment interference, while trauma-related, state and trait anger may be less influential on trauma recovery. However, these broad conclusions are tempered by the different dimensions of anger assessed across studies. One also cannot discount the variety of interventions and modalities used to treat PTSD and the differences in samples (both gender and trauma type) across these studies. No study to date has assessed the rate of change in various anger presentations in the context of recovery from PTSD.

Given the proposal that anger may be a type of avoidant coping (Foa et al., 1995), it seems likely that individuals with high levels of anger could be at risk for dropping out of treatment as a way of avoiding the negative emotional experiences involved in the treatment process. Additionally, high levels of anger can lead to poor interpersonal relationships, which could include difficulty building rapport, resulting in a higher likelihood of drop-out. For these reasons, anger could be an important variable to examine as a risk for drop-out from PTSD treatment. Unfortunately, few studies have reported findings examining differences in baseline anger between drop-outs and treatment completers. Interestingly, the bulk of studies that have reported these findings indicate that anger is not associated with attrition (Taylor et al., 2001; Taylor et al., 2003; van Minnen et al., 2002) with the exception of Rizvi and colleagues (2009), who found that, within the PE condition only (not CPT), women who dropped out reported higher pre-treatment trait anger than women who completed.

Anger clearly represents an important component of the clinical presentation of PTSD. Elevated levels of anger coupled with severe PTSD scores at baseline appear to predict worse outcomes across psychological treatments. Overall, interventions designed to treat PTSD have demonstrated efficacy in reducing pathological expressions of anger, albeit these effects appear to vary across type of anger assessed, type of intervention administered and type of trauma experienced by the survivor (e.g. Taylor et al, 2003; Arntz, Tiesema, & Kindt, 2007; Cahill, et al., 2003). However, less is known about the pattern of change across

the different dimensions and expressions of anger in the context of recovery from PTSD. This study thus posed the questions; does the pattern of change in anger depend on recovery from PTSD? Or does trauma-focused therapy independently influence anger expressions irrespective of full PTSD recovery? Does the answer to this question depend on the dimension of anger assessed?

Present Study

This study examined data collected as part of a previously reported trial comparing full protocol CPT to its two theorized main components, cognitive therapy only (CPT-C) and written accounts (WA; Resick et al., 2008). The main results of the parent study revealed no significant differences between the treatment conditions on attrition and all three conditions substantially improved in PTSD and depression, with CPT-C showing greater reductions in self-reported PTSD symptoms at several points during treatment as compared to WA. The parent paper reported outcomes on two dimensions of anger. All three treatment conditions endorsed significant reductions in anger-in, but did not evidence reductions in anger-out. No differences in anger reductions in these subscales emerged between the treatment conditions. Thus it appears that all three treatment conditions were equally effective in reducing anger.

The goal of this study was to examine the clinical course of dimensions of anger and their relationship to overall change in PTSD symptomatology through the course of PTSD treatment. Given the equivalent effects of all three treatment conditions in the study, we collapsed the sample across the three treatment conditions. Based upon findings suggesting that the presence of anger may be associated with reduced treatment response, we examined differences in the course of anger symptoms *first* between treatment responders and non-responders. We hypothesized that treatment responders would evidence greater improvements on all dimensions of anger. We *then* assessed differences in change on dimensions of anger between treatment responders and drop-outs. We once again hypothesized that responders would evidence greater improvement than those who dropped out of therapy prematurely. State anger, trait anger, anger-in, and anger control were considered separately in an effort to identify specific elements of anger that may be more dependent on recovery from PTSD. Finally, it was predicted that when the treatment responder group was utilized as a reference group, large effect sizes would emerge between the responders and drop-outs. We hypothesized that the non-responders would show some benefit (smaller effects) on anger symptoms by virtue of their exposure to therapy as compared to drop-outs who did not receive the same dose of therapy.

Method

Participants

Female participants were recruited from the St. Louis metropolitan area to the Center for Trauma Recovery (an outpatient clinic/research center located in a stand-alone building on a university campus) through referrals from victim assistance agencies, flyers, advertisements, and word of mouth. Participants, age 18–75, had experienced at least one incident of physical or sexual assault in their lifetime, were at least 3 months post-trauma, met criteria for PTSD at the initial assessment, and were stable on medication for at least one month

prior to baseline assessment. Exclusion criteria included current psychosis, suicidal intent, or dependence on drugs or alcohol. Additionally, participants could not currently be in an abusive relationship or being stalked. The intent-to-treat (ITT) sample included 150 women, 86 of whom completed all therapy sessions. All participants completed the pre-treatment assessment and were asked to complete post-treatment and a 6-month follow-up assessment conducted by trained, blind raters.

This study included a sample of 139 participants. Eleven participants in the ITT sample did not complete the STAXI and so were not included in these analyses. The participants averaged 35.14 years of age ($SD = 12.35$) and 13.78 years of education ($SD = 2.78$). Sixty-three percent of the participants were White, 32% Black, and 5% endorsed another racial identity. Three percent of the participants identified as Hispanic.

The sample was grouped according to treatment responsivity status (responder, non-responder, and drop-out). *Non-responder* was defined as PTSD-positive diagnostic status (at least one Cluster B symptom, 3 Cluster C and 2 Cluster D symptoms on the CAPS at a minimum of “at least once a month” frequency and at least a “moderate” intensity) met at post-treatment despite full completion of the protocol. Although the non-responder group varied in change in PTSD symptom severity, diagnostic cutoff was used to indicate an accepted standard of remaining psychopathology that is consistent with DSM-IV (APA, 1994) requirements for a current psychiatric disorder. Non-responders completed all available 12 hours of therapy in either the CPT, CPT-C, or WA protocol. *Drop-outs* were defined as those who left therapy before the conclusion of the protocol. Of the 57 drop-outs in this study, 56% never came to a single session or dropped out after only the first session of therapy. An additional 37% did not complete half the therapy, dropping out between sessions 2–5. The remaining 5% of the drop-out sample received less than 75% of the treatment protocol. *Responders* were identified as those who completed the protocol (all 12 available therapy hours with the exception of one individual who completed only 11 sessions and had to terminate therapy but had a CAPS score of four at post-treatment) and were PTSD negative at post-treatment. Several baseline differences emerged between responders, non-responders, and drop-outs. Forty-four percent of treatment responders and 45% of non-responders, compared to 75% of drop-outs, reported a yearly income of less than \$20,000, $\chi^2(10, N = 135) = 18.91, p = .041$. When racial origin was examined, 47% of the drop-outs were Black, while 21% of responders and 25% of non-responders identified as Black, $\chi^2(8, N = 139) = 16.83, p = .032$. Twenty-one percent of drop-outs reported less than 12 years of education, compared to 8% of responders and 0 non-responders, $\chi^2(38, N = 139) = 55.50, p = .033$. Non-responders reported significantly more severe PTSD symptoms than responders at baseline $t(80) = -2.68, p = .009$, while drop-outs did not differ from responders or non-responders at pre-treatment. Identification as Hispanic, age, and baseline anger (all dimensions) did not differ across treatment responsivity status.

This particular study design was complex with regard to power estimation. First, the grouping variable (responsivity status) had three, not two levels. Second, change was expected to be non-linear (initial rate of change flattens from post to follow-up measurement). These two issues rendered power estimation difficult (see Feingold, 2009 and Raudenbush & Liu, 2001). That said, using a repeated measures ANOVA approach, we

estimated that approximately 30 individuals in each condition would produce power at .80 for a medium between groups effect size (Cohen's $d = .5$). The multilevel strategy used in this study produces more power for a number of reasons than repeated measures ANOVA (e.g., no need to eliminate partial data). Therefore, given the data being analyzed, this study should have sufficient power to detect the group differences effect on change.

Measures

Clinician Administered PTSD Scale—(CAPS; Blake et al., 1995). The CAPS is a 30-item, structured clinical interview designed to assess PTSD symptoms. Items correspond to the diagnostic criteria for PTSD described in the DSM-IV (APA, 1994), including functional impairment. Items contain separate 5-point frequency and intensity rating scales (ranging 0–4). PTSD symptom severity is calculated by summing scores obtained for frequency and severity of each symptom. In order to be included in the study, participants needed to meet diagnostic criteria for PTSD as set forth by DSM-IV (APA, 1994). In order for a symptom to be counted towards the diagnosis, the participant had to endorse the symptom at a frequency of at least once per month and an intensity of at least moderate distress.

State-Trait Anger Expression Inventory—(STAXI; Spielberger, 1991). The STAXI is a 44-item measure of the experience, expression, and control of anger. The measure consists of six primary subscales including state anger, trait anger, anger-in, anger-out, anger control, and anger expression. The first five subscales were used in this study. Anger expression was not used as its score is derived from other subscales, there is limited normative data, and there is a lack of previous examination in relation to PTSD. *State anger* captures subjective anger experience at the time of administration, whereas *trait anger* measures general anger proneness. Anger-in represents the degree to which angry feelings are experienced and suppressed or held in. Conversely, anger-out measures the frequency with which angry feelings are experienced and outwardly expressed (i.e., aggressive behavior). The fifth subscale, anger control, taps the frequency of attempts to control the experience and expression of anger. Higher scores indicate more anger, with the exception of anger control where higher scores indicate lower symptoms.

Procedure

Eligible participants completed an initial assessment which included self-report measures and clinician-administered interviews. Participants were then randomly assigned to one of three experimental conditions (CPT, CPT-C, or WA). Two weeks after completing treatment, participants (including drop-outs) were invited to return for a post-treatment assessment and then again 6 months after treatment for a follow-up assessment. All assessments were conducted by trained, advanced doctoral level clinicians who were blind to the study conditions. Therapy (1-hour, individual sessions) was conducted twice a week for 6 weeks with the exception of the WA condition in which 2-hour sessions, once per week occurred during weeks 2–6. Thus each condition was designed to include 12 hours of therapy over 6 weeks. Therapy was conducted by clinical psychologists and treatment and adherence to each protocol was evaluated by independent raters. As this study collapsed across treatment conditions, a full description of the interventions will not be described here, but rather, the reader is referred to the parent paper (Resick, Galovski, et al., 2008).

Analytical Strategy

An intent-to-treat (ITT) philosophy was used for creation of the outcome models. One hundred and thirty-nine participants were enrolled and contributed a pre-treatment score, 113 of these participants contributed a post-treatment score and 113 contributed a follow-up assessment score. Descriptive statistics (e.g., means, standard deviations and correlations) are presented first.

We utilized a multilevel approach to analyzing longitudinal data (Singer & Willet, 2003) to examine the impact of trauma-focused therapy on anger, using the Linear and Nonlinear Mixed Effects Models package from R (Pinheiro, Bates, DebRoy & Sarkar, 2006). Multilevel modeling is ideal for studies which employ repeated measurements nested within individuals. Raw coding of the polynomials (Time, Time²) was used for increased interpretability (Biesanz, Deeb-Sossa, Papadakis, Bollen, & Curran, 2004), according to the following scheme: 0 (pre-treatment), 1 (post-treatment), and 2 (6-month follow-up). Several variants of this model were tested in a tear-down procedure (Cohen, Cohen, Aiken, & West, 2003) to determine whether growth was linear or quadratic (level 1). Responsivity status was dummy coded and tested as a level 2 predictor (treatment responders as the reference group).

The five subscales of the STAXI were the dependent variables (DVs): state anger, trait anger, anger-in, anger-out, and anger control. Analysis of each DV was conducted using the following steps: (1) analyze the variation in the DV via a null model to assess the between and within variance and compute an ICC, (2) model change in the DV to determine if a linear model would be sufficient, (3) determine if the slopes are random, and (4) determine if responsivity status groups differ in their patterns of change, (5) repeat the previous model with the addition of a block of covariates (i.e., those identified above as pre-treatment differences – PTSD, race origin, years of education, income status). Two models resulted in non-significant interaction terms (i.e., responsivity status was not associated with growth parameters – see results section below). To rule out the possibility that the lack of significance in the interaction terms from these two models was due to multicollinearity between the interaction terms and their component variables, an additional step was run using orthogonal polynomials for Time (Biesanz et al., 2004).

Results

Prior to the testing of the primary analyses, the associations between PTSD and levels of anger endorsed by the current sample were examined. Consistent with prior findings, each STAXI subscale was significantly correlated with the CAPS total score at pre-treatment, with the exception of anger-out (see Table 1). Table 2 presents sample means and available normative data (Spielberger, 1991) for the anger subscales. Trait anger and anger-out means were comparable to normative means for female, non-college adults. Given the lack of elevation and change in anger-out across treatment (Resick et al., 2008), anger-out was excluded from further analyses. Analyses on trait anger were not reported in the parent study, so trait anger was retained.

Table 3 displays the raw means, standard deviations, and Hedge's g effect sizes for the responsivity status groups across assessment intervals. Table 4 displays the multilevel models (described further below) for the final models with covariates included (covariate effects are omitted for space considerations). Each of the four anger dimensions changed significantly over the study period ($ps < .01$ for linear components and many of the quadratic – see Table 4 for final models¹). A quadratic growth model suggested significant change over time for state ($\gamma_{lin} = -7.12$, $\gamma_{quad} = 2.42$) and trait ($\gamma_{lin} = -4.09$, $\gamma_{quad} = 1.15$) anger and anger-in ($\gamma_{lin} = -5.33$, $\gamma_{quad} = 1.55$), with more rapid initial change (i.e., over the treatment period) followed by a slowing or maintenance of gains during follow-up (see Tables 3–4). Anger control also demonstrated significant change over time, but a linear growth model emerged ($b_{lin} = 1.06$), indicating that change was constant across treatment and continued through the follow-up interval.

Responsivity status (responder, non-responder, drop-out) as it relates to change

Responsivity status was significantly related to change in each anger dimension². Differing growth patterns emerged between responder groups. Figure 1 displays the fitted means for each anger dimension over time by responsivity status. For state anger, drop-outs experienced less initial change than responders ($\gamma_{linear*group2} = 6.56$, $p = .005$ – in comparison to $\gamma = -7.12$ for responders) and had a significantly flatter growth trajectory ($\gamma_{quad*group2} = -2.2$, $p = .031$ – in comparison to $\gamma = 2.42$ for responders), even after accounting for pre-treatment PTSD symptom severity and demographic control variables. Endpoint effect sizes indicate a large difference between responders and drop-outs at follow-up (Hedge's $g = -.77$; see Table 3). No differences were seen between the non-responders and responders in change ($\gamma_{linear*group1} = 3.39$ and $\gamma_{quad*group1} = -.85$, $ps > .1$), although effect size estimates indicate a sizable between group difference between non-responders and responders at follow-up (Hedge's $g = -1.10$; see Table 3).

Orthogonal polynomials were used to reduce multicollinearity between the growth variables and the interaction terms for *trait anger* and anger-in (i.e., the components and their product; Ployhart, Holtz & Bliese, 2002). In the final model, responders reported the most rapid initial change in *trait anger* and the most overall change (see Figure 1 for fitted values). Relative to responders ($\gamma_{linear} = -28.57$ – using orthogonal polynomials), non-responders ($\gamma_{linear*group1} = 30.05$, $p < .01$) and drop-outs ($\gamma_{linear*group2} = 26.47$, $p < .01$) displayed less initial change over the study period. This finding is mirrored in large endpoint effect size differences relative to the responder group at follow up for both non-responders and drop-outs respectively (Hedge's $g = -1.16$ and $-.69$).

Regarding anger-in, both responders and non-responders decreased over treatment and follow-up (see Figure 1 and Table 3). Drop-outs reported significantly less initial change (i.e., over the treatment period) relative to responders (cf. $\gamma_{linear} = -35.48$, vs. $\gamma_{linear*group2} = 29.29$, $p < .001$ – using orthogonal polynomials) and demonstrated fewer gains at follow-up

¹Earlier growth models are not depicted. Tabular results for all models are available from the first author upon request.

²For two of the outcomes, significance was only detected when using orthogonal polynomials when coding for time. For simplicity and consistency, the results in Table 4 report the raw coded time (0, 1, 2) rather than the more complex orthogonal polynomials. Alternate results are available from the first author upon request.

(Hedge's $g = -1.10$). Differences between non-responders and responders on anger-in did not reach significance ($p > .20$) and were in the medium effect size range at follow-up (Hedge's $g = -.61$).

Finally, responsivity status was significantly related to growth for anger control. Non-responders had a significantly lower growth rate than responders in anger control (cf. $\gamma_{linear} = 1.06$, vs. $\gamma_{linear*group1} = -1.49$). Thus, responders increased in anger control whereas non-responders experienced a decrease (see Figure 1 and Table 3). Endpoint effect sizes indicated a medium to large effect of responsivity status at follow-up (Hedge's $g = .86$). Drop-outs did not significantly differ from responders ($p > .15$; Hedge's $g = .37$).

Discussion

The extant literature has identified anger (particularly anger-in, anger-out, and anger control) as uniquely related to PTSD (Orth & Wieland, 2006; Olatunji et al., 2010). Consistent with previous research, this study found a significant relationship between initial PTSD severity and four out of five anger dimensions (state anger, trait anger, anger-in, and anger control) in a sample of interpersonal assault survivors. Interestingly, and in contrast with previous literature, these results did not reveal a significant relationship between PTSD symptoms and anger directed outward. Sample differences may explain this discrepancy. Because previous research has observed sex differences in various dimensions of anger presentations (Galovski, Mott, Young-Xu, & Resick, 2011), the absence of male survivors in this study may explain in part the baseline low levels of anger-out and resultant floor effects for change on this domain.

Although, by definition, responders and non-responders differed with respect to PTSD diagnostic status at the conclusion of treatment, it appeared both groups benefited from treatment on measures of state anger and anger-in, with no difference in the pattern of change. These results suggest that improvements in these dimensions of anger, following a course of PTSD treatment, occur even for those therapy participants who retain their PTSD diagnosis. It is possible that the grouping variable (PTSD diagnostic status at treatment's end) may be masking meaningful improvements in overall PTSD symptomatology which then generalize to anger symptomatology. In examining the raw data, approximately half of the "treatment non-responders" dropped at least 10 points on the CAPS (considered a clinically meaningful change; Schnurr et al., 2007). Sample sizes were too small to statistically assess differential patterns of change in anger dimensions and PTSD symptoms within this group of partial responders as compared to those individuals who evidenced no change on PTSD symptomatology after a course of trauma-focused therapy. However, we can conclude from the results of this study that improvements in state anger and anger-in for the non-responder group were on par with improvements in the responder group.

Differences in anger recovery did emerge between responders and non-responders on trait anger and anger control. Non-responders demonstrated significantly less change on trait anger than did the responders. Further, responders increased in control over anger while non-responders decreased in their ability to control their anger. These findings have clinical importance in informing treatment decisions. While it appears that individuals may improve

with respect to state anger and anger-in over a course of PTSD irrespective of retaining their PTSD diagnosis, this was not the case on trait anger and anger control. It can be speculated that the interventions may have some independent effect on the former dimensions of anger (state anger and anger-in), but improvements in anger control and trait anger may be more dependent on the remediation of PTSD.

Trajectories of change on state anger, trait anger, and anger-in all indicated that the rate of change was more pronounced during treatment (from pre- to post-treatment) but decelerated from post-treatment to follow-up. Interestingly, anger control was the only subscale on which a linear pattern of change emerged, such that the rate of change did not vary between assessment intervals and responders continued to improve between the post-treatment and follow-up assessments. This continued improvement bears some consideration. Responders not only improved in anger control over the course of treatment, but continued to improve on this dimension after treatment (in the absence of a PTSD diagnosis). It is unclear as to whether this continued improvement is due to the treatment or perhaps due to the effects of living without a PTSD diagnosis. In support of this latter hypothesis, previous research examining the temporal relationship of PTSD and anger (in the absence of treatment) has demonstrated that PTSD symptoms predicted anger, but the reverse was not true (Orth, Cahill, Foa, & Maercker, 2008). However, Orth et al.'s study looked at state anger only. Change in the ability to control one's anger has not been as closely examined, particularly after the remediation of PTSD, and may be particularly important to address clinically. While trait anger showed a significantly flatter change rate for the treatment non-responders versus the responders, the lack of control over anger actually increased for the PTSD non-responders. This is consistent with previous research suggesting that anger control is strongly associated with PTSD (Orth and Wieland, 2006; Olatunji et al., 2010) and that anger control alone may interfere with treatment recovery (van Minnen et al., 2002). Particular clinical attention to both cognitions and behaviors associated with lack of control over anger may augment trauma-focused interventions and enhance overall outcomes.

Based on the previous literature suggesting that elevated anger may contribute to attrition, change patterns in anger dimensions were examined between the reference group (treatment responders) as compared to drop-outs. These groups did not differ on pre-treatment levels of anger, which is consistent with previous findings (Taylor et al., 2003; van Minnen et al., 2002). Not surprisingly, participants who dropped out of treatment prematurely realized significantly less improvement in state anger, trait anger, and anger-in compared to those who responded to PTSD treatment. A limitation to this study is that the vast majority of drop-outs did so very early in the therapy, so it is difficult to assess the influence of anger on drop-out from any given session or particular portion of therapy. Future research may seek to assess anger on a session-by-session basis to more fully understand the role that anger may play in influencing attrition from therapy.

There are several limitations to this study. First, only women were included, rendering the results difficult to generalize to men. This limitation is tempered by the contribution to the relatively limited literature on anger recovery in PTSD-positive women. Second, this sample included survivors of interpersonal violence, once again introducing some difficulty in translating these results to survivors of other types of trauma. Third, the lack of a no-

treatment control group limits the ability to attribute changes to the treatment. The overall sample size is not large, particularly with respect to the drop-out and non-responder groups, potentially limiting the ability to detect differences among these groups. Finally, the groups were derived on PTSD diagnostic outcomes only, as this treatment is specifically designed to treat PTSD.

This study supported the larger literature suggesting that individuals with PTSD are likely to present with elevated anger symptoms. In light of the mixed findings in the PTSD and anger outcome literature, these results offer some clarity regarding the relationship between PTSD recovery and anger remediation. Surprisingly, the improvements in anger following treatment did not seem to be related to the loss of the PTSD diagnosis on two of the measures of anger, but did on trait anger and anger control. Not surprisingly, individuals who terminated therapy prematurely did not experience the same gains in anger outcomes as those who completed treatment. Future research may seek to examine trauma-related anger specifically and compare across genders and trauma types.

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Figure 1.

Anger subscale fitted values by responsivity status over time (0 = baseline, 1 = post-treatment, 2 = follow-up). Relative to responders, non-responders demonstrated significantly slower initial change in trait anger and less overall growth in anger control. Drop-outs displayed significantly slower initial change in state and trait anger, as well as anger control.

Table 1

Pearson Correlations of STAXI Subscales and CAPS Total Score at Pre-treatment

	State anger	Trait anger	Anger-in	Anger-out	Anger control
Trait anger	.50**	--	--	--	--
Anger-in	.36**	.41**	--	--	--
Anger-out	.36**	.65**	.28**	--	--
Anger control	-.30**	-.55**	-.06	-.46**	--
CAPS total	.32**	.26**	.21*	.14	-.18*

Note:

* $p < .05$,** $p < .01$.

Table 2

Pre-treatment Means and Standard Deviations by Responsivity Status and Normative Anger Data

Anger subscales	Adult Norms	Drop outs (n = 57)	Responders (n = 62)	Non-responders (n = 20)
State anger	12.82 (4.83)	15.66 (6.28)	15.83 (7.09)	16.77 (7.38)
Trait anger	19.44 (5.11)	19.19 (6.10)	18.94 (5.44)	20.00 (5.97)
Anger-in	15.70 (4.24)	19.70 (5.23)	18.66 (4.18)	20.35 (3.53)
Anger-out	14.92 (4.02)	14.91 (4.01)	14.69 (4.06)	15.50 (3.55)
Anger control	Not available	22.82 (4.77)	23.40 (5.33)	22.10 (4.70)
CAPS ^a	Not applicable	72.60 (18.16)	67.00 (17.79)	79.75 (20.72)

Note: Adults normative data are based on female, non-college adults (Spielberger, 1991).

^a Non-responders had significantly higher CAPS scores at pre-treatment than drop-outs or non-responders ($p = .009$)

Table 3

Pre-, Post-treatment, and Follow-up Values for ITT Groups across Anger Dimensions

	Responders (<i>n</i> = 62)				Non-responders (<i>n</i> = 20)				Drop-outs (<i>n</i> = 57)				Hedge's <i>g</i> (at Follow-up)	
	Pre-treatment <i>M</i> (<i>SD</i>)	Post-treatment <i>M</i> (<i>SD</i>)	Follow-up <i>M</i> (<i>SD</i>)		Pre-treatment <i>M</i> (<i>SD</i>)	Post-treatment <i>M</i> (<i>SD</i>)	Follow-up <i>M</i> (<i>SD</i>)		Pre-treatment <i>M</i> (<i>SD</i>)	Post-treatment <i>M</i> (<i>SD</i>)	Follow-up <i>M</i> (<i>SD</i>)		Responders vs Non- responders	Responders vs Drop-outs
State Anger	15.83 (7.09)	11.12 (2.28)	11.28 (2.56)		16.77 (7.38)	14.62 (4.91)	15.31 (6.11)		15.66 (6.28)	14.57 (6.22)	15.31 (7.18)		-1.10	-.77
Trait Anger	18.94 (5.44)	15.98 (4.50)	15.44 (3.51)		20.00 (5.97)	19.63 (5.07)	19.94 (5.09)		19.19 (6.10)	17.55 (6.37)	19.03 (6.69)		-1.16	-.69
Anger-in	18.66 (4.18)	14.87 (4.03)	14.14 (3.73)		20.35 (3.53)	18.42 (3.44)	16.82 (6.19)		19.70 (5.23)	17.09 (4.05)	18.73 (4.67)		-.61	-1.10
Anger Control	23.40 (5.33)	25.03 (4.49)	25.44 (4.40)		22.10 (4.70)	21.26 (4.36)	21.29 (6.23)		22.82 (4.77)	23.36 (4.50)	23.73 (4.84)		.86	.37
N	62	61	59		20	19	17		57	33	37			

Note. Entries are the means (standard deviations) from the raw data.

Table 4

Fixed Effects for Anger Models – Final model with Control Variables^c

	State Anger		Trait Anger		Anger-in		Control Anger	
	Est.	SE	Est.	SE	Est.	SE	Est.	SE
Intercept	12.94**	2.64	16.71**	2.86	17.48**	2.08	21.54**	2.47
Time ^a	-7.12**	1.41	-4.09**	1.17	-5.33**	1.10	1.06**	.30
Time ²	2.42**	.60	1.15*	.56	1.55***	.53		
Group1 ^b	-.01	1.73	.52	1.47	.99	1.15	-1.83	1.25
Group2 ^b	-.95	1.29	.29	1.11	.46	.87	-.61	.96
Group1 *Time	3.39	2.88	3.33	2.39	3.30	2.26	-1.49*	.63
Group1 *Time ²	-.85	1.23	-.71	1.16	-1.38	1.10		
Group2 *Time	6.56**	2.30	2.10	2.01	.78	1.88	-.71	.49
Group2 *Time ²	-2.20*	1.01	-.21	.98	.56	.92		
Level 1 df	213		213		213		216	
Level 2 df	121		121		121		121	
τ_{00}	34.13		16.56		6.87		11.79	
τ_{11}	68.40							
τ_{22}	9.39							
σ^2	8.34		12.75		11.39		10.98	
AIC	2144.16		2099.40		1998.94		2033.11	
LL	-1045.08		-1027.70		-977.47		-997.56	

Note.

*
 $p < .05$,**
 $p < .01$.^aTime is coded 0, 1, 2 for pre-, post- and follow-up respectively.^bFor responsibility status, responders is the reference category; non-responders (group 1) and drop-outs (group 2) are compared against responders. Statistical controls (PTSD, race, years of education, and income status) were groups entered simultaneously; fixed effects for controls (*11 in groups due to categorical nature*) are omitted from table. Off-diagonal elements of the variance terms were also assessed but not depicted here. Missing τ elements are due to random components not being estimated (e.g., a previous model deemed slope was not random).