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Changes in anger and aggression after treatment for PTSD in active duty military

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Abstract

Objective: To examine whether treating posttraumatic stress disorder (PTSD) reduces anger and aggression and if changes in PTSD symptoms are associated with changes in anger and aggression.

Method: Active duty service members (n = 374) seeking PTSD treatment in two randomized clinical trials completed a pretreatment assessment, 12 treatment sessions, and a posttreatment

assessment. Outcomes included the Revised Conflict Tactics Scale and state anger subscale of the State-Trait Anger Expression Inventory.

Results: Treatment groups were analyzed together. There were small to moderate pretreatment to posttreatment reductions in anger (standardized mean difference [SMD] = -0.25), psychological aggression (SMD = -0.43), and physical aggression (SMD = -0.25). The majority of participants continued to endorse anger and aggression at posttreatment. Changes in PTSD symptoms were mildly to moderately associated with changes in anger and aggression.

Conclusions: PTSD treatments reduced anger and aggression with effects similar to anger and aggression treatments; innovative psychotherapies are needed.

Keywords

active military; aggression; anger; cognitive processing therapy; posttraumatic stress disorder

1 | INTRODUCTION

The emotion of anger and behavioral acts of aggression can lead to severe negative consequences, including family violence, legal charges, and death (Taft, Watkins, Stafford, Street, & Monson, 2011). Unfortunately, returning service members and veterans are reporting difficulties managing their anger and aggression and trouble with the subsequent consequences. Posttraumatic stress disorder (PTSD) contributes to anger and aggression, particularly in veterans and service members (Marshall, Panuzio, & Taft, 2005; Orth & Wieland, 2006). Nearly half of veterans with PTSD symptoms reported engaging in physical aggression, and 20% endorsed violent behavior that harmed another person within the first year of returning from deployment (Elbogen et al., 2014). Veterans with PTSD reported more anger and aggression than veterans without PTSD (Jakupcak et al., 2007). The associations between PTSD and anger and aggression are concerning, considering that 7%–20% of returning service members (Hoge et al., 2004) and veterans (Fulton et al., 2015) report clinical levels of PTSD symptoms. PTSD symptoms can be reduced with psychotherapies (Management of Posttraumatic Stress Disorder Work Group, 2017), but it remains unknown if completing PTSD treatment also decreases service members' anger and aggression.

2 | THE UTILITY OF ANGER AND AGGRESSION

Anger and aggression are distinct, yet related, constructs. Anger is an emotion that often precedes aggression, yet it is neither necessary nor sufficient for aggression. Aggressive behavior can be psychological (e.g., yelling insults at another person) or physical (e.g., striking, shoving, burning another person). It is generally the aggressive behaviors that lead to short-term negative consequences, while chronic anger has been linked to distal health outcomes such as cardiovascular disease (Suls, 2013) and stroke (Williams, Nieto, Sanford, Couper, & Tyroler, 2002).

While anger and aggression are different constructs, they both can promote survival. All mammals express anger to signal thwarted needs and goals (Harkness, Reynolds, & Lilienfeld, 2014). Aggression is similarly functional and can serve to protect family,

belongings, or an agenda. In combat situations, anger and aggression are energizing (fight-flight response) and can assist service members in completing missions even after events that trigger sadness (e.g., losing a comrade; Harkness et al., 2014). Service members may also use anger as a method to avoid other unwanted emotions (e.g., fear, sadness) and thoughts (Foa, Riggs, Massie, & Yarczower, 1995).

Anger and aggression can be adaptive and necessary in threatening situations, such as combat. They become less useful and potentially harmful when over-applied in safer situations, such as when a veteran returns home from deployment. Chemtob, Novaco, Hamada, Gross, and Smith (1997) have proposed the Survival Mode theory to explain the strong associations between PTSD and anger/aggression among combat veterans. In this model, perceived threats trigger increased arousal, hostile appraisals, and aggressive behaviors (Chemtob et al., 1997; Novaco, Swanson, Gonzalez, Gahm, & Reger, 2012). While protective in combat situations, this "survival mode" becomes maladaptive when individuals are no longer in danger. Consistent with this theory, many service members with PTSD have trouble distinguishing between safe and potentially unsafe people and places (Weber, 2008). Therefore, when a service member with PTSD encounters a stressful environmental event, s/he may already be aroused because of the variety of PTSD-related emotions and cognitions, interpret an ambiguous event as threatening, and respond in an angry or aggressive manner.

3 | RISK FACTORS AND PREVENTION

Predictors of problematic anger and aggression include trauma exposure, male sex, younger age, and combat exposure (Jakupcak et al., 2007; Miles et al., 2017). In addition, alcohol use is consistently a strong predictor of aggression and anger and is often comorbid with PTSD (Carter, Capone, & Short, 2011; Elbogen et al., 2014; Jakupcak et al., 2007). However, research has demonstrated that even while controlling for age, sex, and substance use, PTSD symptoms continue to predict impulsive aggression among veterans (Miles, Menefee, Wanner, Tharp, & Kent, 2015).

While it is important to understand the predictors of anger and aggression, it is equally important to develop methods to prevent and reduce them. Much of the treatment literature has focused on the relationship between PTSD and anger, while less research has studied aggression. Anger management treatment with cognitive behavioral techniques is often offered to patients who report anger and aggression. Meta-analyses in civilians found small to moderate reductions in anger and recidivism rates; however, the majority of studies focused on anger as the primary outcome and did not measure aggression (Dowden & Andrews, 2000; Lee & DiGiuseppe, 2018).

Veterans' reactions to anger management treatments have been studied less than civilians. There has been one small randomized clinical trial (RCT) with veterans with warzone trauma that found large reductions in anger (Shea, Lambert, & Reddy, 2013). Two larger RCTs with intent-to-treat samples demonstrated that veterans with PTSD showed small to moderate reductions in anger after completing cognitive behavioral therapy for anger problems (Chemtob et al., 1997; Morland, et al., 2010). Only one RCT has examined

the treatment of aggression in a military sample, comparing Strength at Home, a trauma-informed, 10-session couples' treatment, to supportive prevention. Strength at Home reduced interpersonal violence with small to moderate effect sizes as compared to supportive prevention (Taft et al., 2016).

Because anger and aggression can be symptoms of PTSD (American Psychiatric Association, 2013), evidence-based psychotherapies (EBPs) for PTSD may be a logical treatment choice. Cognitive Processing Therapy (CPT; Resick, Monson, & Chard, 2017) is an EBP for PTSD. The extant literature that examined changes in anger after CPT has predominately been conducted with civilians. In a sample of women who experienced interpersonal violence, completing CPT was associated with reductions in anger and anger directed inward (toward the self), regardless of if the women retained their PTSD diagnoses after treatment. PTSD symptom reduction was associated with less overall anger and better anger control (Galovski, Elwood, Blain, & Resick, 2014). Resick et al. (2008) also found that women who received CPT had moderate reductions in overall anger directed inward and smaller improvements in control of their anger directed outwards. Similarly, veterans with PTSD who completed CPT reported moderate to large reductions in anger as compared to treatment as usual (Forbes et al., 2012

Prolonged exposure (PE; Foa, Hembree, & Rothbaum, 2007) is another EBP for PTSD. In a study of civilians who completed PE and experienced PTSD symptom reduction, over half the sample continued to report anger/ irritability as problems (Zayfert & DeViva, 2004). Another pilot study examined male veterans with PTSD who served in Afghanistan or Iraq and found that PE reduced how much a participant was bothered by his hostility (behaviors) but did not reduce the emotion of anger (Ford, Grasso, Greene, Slivinsky, & DeViva, 2018). A final study combined civilian samples that completed either PE or CPT and found that irritability was only present in about 20% of the combined sample at posttreatment compared with about 80% at pretreatment (Larsen, Fleming, & Resick, 2019).

No study has examined the effect of completing PTSD treatment on frequency or severity of aggression, which is distinct from anger. Nor have service members who completed PTSD treatment been considered with adequately powered samples. Because anger and aggression may be adaptive in future combat situations, service members may respond differently to PTSD treatments. The first aim of the current study was to examine changes in anger and aggression as separate constructs for active duty service members who took part in two RCTs designed to study the efficacy of CPT (group and individual) for PTSD (Resick et al., 2017, 2015). A detailed explanation of CPT is beyond the scope of this manuscript (see Resick et al., 2017 for more information). In general, CPT teaches individuals to identify and challenge unrealistic/unhelpful thoughts. These skills can be used for trauma-related thoughts in addition to thoughts that lead to anger and aggression (Resick et al., 2017). The active control group in one of the RCTs received Present Centered Therapy (PCT; Resick, 2015) delivered in group format. PCT aims to reduce distressing symptoms by facilitating the patient in active problem-solving training and implementation. We predicted that CPT and PCT would lead to decreases in anger from pretreatment to posttreatment (Hypothesis 1a) and that the frequency of both psychological and physical aggression would decrease from pretreatment to posttreatment regardless of individual or group treatment format

(Hypothesis 1b). Our second aim was to determine whether changes in either anger or aggression were related to changes in PTSD over the course of treatment. We predicted that changes in PTSD severity would be positively associated with changes in anger (Hypothesis 2a) and psychological and physical aggression (Hypothesis 2b) for all the treatment groups.

4 | METHOD

4.1 | Participants

This study was a secondary data analysis of combined samples from RCTs that compared CPT administered in a group format to PCT administered in a group format (Study 1; Resick et al., 2015) and CPT administered in a group format to CPT administered in an individual format (Study 2; Resick et al., 2017) in active duty service members (see Table 1). Details regarding the South Texas Research Organizational Network Guiding Studies on Trauma and Resilience (STRONG STAR; www.STRONGSTAR.org) parent trials are described in detail elsewhere (Resick et al., 2017, 2015). Participants were 374 active duty U.S. service members (91% male) seeking treatment for PTSD; all participants served at least one deployment in support of combat operations following 9/11. Eligibility requirements included experience of a Criterion A traumatic event as defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; APA, 2000) that occurred during deployment. However, treatment may have focused on another Criterion A event (e.g., childhood sexual abuse). All participants met criteria for PTSD based on the PTSD Symptom Scale Interview (PSSI: Foa, Riggs, Dancu, & Rothbaum, 1993) that was administered by a trained and supervised master- or doctoral-level independent evaluator who was unaware of treatment condition. Interrater reliability for the PSSI was very good (Cohen's $\kappa = .83$ and .89 for Resick et al., 2015 and Resick et al., 2017, respectively). Participants taking psychotropic medications were required to be on a stable dosage for 6 weeks before study entry and were asked to remain on an unchanging regimen throughout the treatment. Participants received support from their unit commanders to participate and were not engaged in other PTSD treatments during the study. Exclusion criteria included current suicide or homicide risk meriting crisis intervention, active psychosis, and moderate to severe traumatic brain injury.

4.2 | Procedures

Trials were approved by institutional review boards at Brooke Army Medical Center, the University of Texas Health Science Center at San Antonio, VA Boston Healthcare System, and Duke University Medical Center. Participants signed informed consent documents and completed diagnostic assessments and self-report measures. All measures were common data elements administered in multiple trials testing treatments for PTSD in active duty service members and veterans being conducted as part of the STRONG STAR Consortium. Participants who met inclusion—exclusion criteria were randomized into the trials. Treatments consisted of 12 sessions, delivered twice weekly for 6 weeks. Individual CPT sessions were 50–60 min, and all group sessions were 90 min. To reduce the risk of losing service members who were redeployed, discharged, or relocated, the posttreatment assessment was conducted 2 weeks after the final treatment session.

4.3 | Measures

4.3.1 I **Posttraumatic stress disorder symptoms**—All participants met PTSD diagnostic criteria for the *DSM-IV-TR* based on the PSSI because Study 1 began when the *DSM-IV-TR* was in effect. However, knowing the DSM criteria were under revision, a draft version of the PTSD Checklist for *DSM-5* (PCL-5; Weathers et al., 2013) was used to evaluate PTSD symptom change from pretreatment to posttreatment. The draft version of the PCL-5 had nominal wording differences from the final PCL-5 and was validated in a military sample (Wortmann et al., 2016). There is a strong association between DSM-IV and DSM-5 PTSD diagnostic criteria, particularly when PTSD measures use minimum total severity scores (see Weathers, et al., 2018 for scoring details). The PCL-5 is a 20-item self-report measure that was used to evaluate changes in PTSD symptoms in response to a specific trauma. Scoring is based on how much the individual is bothered by the symptoms during the past month on a scale from 0 (*not at all*) to 4 (*extremely*). Scores at or above 31 indicate a potential PTSD diagnosis (Wortmann et al., 2016). Coefficient *α* for the PCL-5 was .88 at pretreatment and .95 at posttreatment.

- **4.3.2** I Anger—The state anger subscale of State-Trait Anger Expression Inventory (STAXI; Spielberger, 1988) is a 10-item self-report measure that assesses the severity of anger as an emotion state "right now." The subscale assessed incidence (dichotomous indicator of any anger at the current time) and severity (state anger subscale total score) of anger at pretreatment and posttreatment assessments. For severity of anger, item scores range from 1 (*Not at all*) to 4 (*Very much so*). For incidence of anger, total scores of 2 or more indicated the presence of anger at each assessment. Coefficient *a* was .96 at both pretreatment and posttreatment.
- **4.3.3** | **Aggression**—The Revised Conflict Tactics Scale (CTS2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996) assesses perpetration of physical (12 items; "I slapped someone.") and psychological (8 items; "I insulted or swore at someone.") aggression in the past month. While the original CTS2 assessed for aggression perpetrated against a romantic partner, we adapted the instructions to measure aggression against anyone, allowing for the assessment of aggression across many individuals. CTS2 was used to assess changes in incidence (dichotomous indicator of any aggression in past month) and frequency (physical aggression total score and psychological aggression total score) of aggression between the pretreatment to posttreatment assessments. For frequency of aggression, items are scored on a 0 (Never) to 6 (>20 times in the past month) scale. For incidence of aggression, scores of 1 or more on each subscale indicated the presence of aggression in the past month at each assessment. Coefficient α was .82 at both the pretreatment and posttreatment assessments for psychological aggression. For physical aggression, coefficient a was .86 at pretreatment and .71 at posttreatment. The discrepancy in α coefficients for the physical aggression subscale was likely due to standard deviation being reduced at the posttreatment assessment (SD = 2.78) compared to the pretreatment assessment (SD = 5.06).
- **4.3.4** I **Hazardous drinking**—Because alcohol use is a robust predictor of anger (Carter et al., 2011) and aggression in PTSD samples (Elbogen et al., 2014), hazardous drinking was examined as a potential covariate for the study and measured by the Alcohol Use

Disorders Identification Test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001). The AUDIT is a 10-item self-report measure that examines physiological and psychological dependence on alcohol as well as negative consequences associated with drinking. Scoring is based on how much alcohol a person consumes or how often they engage in a certain behavior (e.g., "How often during the last year have you had a feeling of guilt or remorse after drinking?") with scores that range from 0 to 4 for all items. Total scores can range from 0 to 40, where higher scores indicate a greater likelihood of an alcohol use disorder. Each item has unique scoring, and individuals endorse different items depending on their drinking behavior. Therefore it is not appropriate to calculate coefficient α for this measure.

4.4 | Data analytic plan

To investigate pretreatment to posttreatment changes in anger, psychological aggression, and physical aggression (Hypotheses 1a and 1b), total scores on the STAXI and each CTS2 subscale were examined using linear mixed model regressions with unstructured covariance matrices. Fixed effects included the effect of treatment group (Study 1 Group PCT [n = 52], Study 1 Group CPT [n = 56], Study 2 Group CPT [n = 133], Study 2 Individual CPT [n = 133]), time (pretreatment to posttreatment), and the group x time interaction. The main effect of time was a formal test of whether anger, psychological aggression, and physical aggression were reduced from pretreatment to posttreatment. The group x time interaction determined if any treatment group produced larger reductions in anger or both types of aggression compared to the other treatment groups. A nonsignificant interaction would allow us to combine the samples, affording more statistical power. Analyses were conducted using SPSS version 25.

To test Hypotheses 2a and 2b, we calculated change scores for anger, psychological aggression, physical aggression, and PTSD severity for each patient. We correlated changes in anger and both aggression subscales with changes in PTSD severity separately for each treatment group. Z statistics were used to test the significance of each correlation coefficient. As a secondary analysis we removed items related to anger (PCL-5 item 11) and aggression (PCL-5 item 15) to ensure our correlations were not due to shared variance of PCL-5 items with the STAXI and CTS2.

Each treatment group may have been underpowered to detect significant changes in anger and/or aggression, so we wanted to combine the treatment groups to increase power. The correlations described above were entered into the Comprehensive Meta-Analysis program in order to examine the Q-statistic (Hedges & Olkin, 1985). Nonsignificant p-values indicate homogeneity of effect sizes across treatment groups and provides permission to calculate pooled estimates of correlated changes. Pooled estimates were obtained using a fixed effects model given the small number of treatment groups (Cooper, 2017). We examined P, which is the proportion of variance in effect sizes due to the treatment groups (e.g., not attributable to sampling error). We calculated pooled estimates for each set of correlated outcomes with a nonsignificant Q-statistic and sufficiently small P value. When significant p-values for a Q-statistic were observed, we calculated and tested pairwise differences for the treatment groups and reported individual groups' correlated changes instead of a pooled effect. Finally, given the association between drinking and anger/aggression, we repeated all the analyses

while controlling for pretreatment AUDIT scores and any demographic variables upon which the groups differed.

5 | RESULTS

5.1 | Preliminary analyses

Participants in each treatment group (Study 1 Group PCT, Study 1 Group CPT, Study 2 Group CPT, Study 2 Individual CPT) did not differ on pretreatment demographic variables with the exception of ethnicity. Study 2 had more Black and Hispanic participants than Study 1 (Table 1). There were also no significant pretreatment differences in severity levels of anger, physical aggression, or psychological aggression between the treatment groups. However, a significant pretreatment difference existed for PTSD severity, with Study 2 participants having lower pretreatment PTSD severity than Study 1 participants. This difference was likely due to the military climate and needs changing over the course of the two studies, with fewer service members on active duty, fewer deployments, and fewer service members seeking PTSD treatment during Study 2. All treatment groups reduced their PTSD symptoms (PCL-5) from pretreatment to posttreatment with moderate to large effect sizes (Study 1 Group PCT, d = 0.65; Study 1 Group CPT, d = 0.85; Study 2 Group CPT, d = 0.48; Study 2 Individual CPT, d = 0.72).

Across all treatment groups, 88% endorsed anger, 97% endorsed psychological aggression, and 32% endorsed physical aggression at the pretreatment assessment. At posttreatment, the percentages dropped to 78% of the sample endorsed anger, 93% endorsed psychological aggression, and 20% endorsed physical aggression. Study 1 participants did not differ from Study 2 participants in incident rates of anger, psychological aggression, or physical aggression, at pretreatment [$X^2(1) = 0.66$, 1.78, and 0.07, respectively, all ps greater than .05] or posttreatment [$X^2(1) = 0.10$, 1.67, and 1.04, respectively, all ps < .05]. If examining the total sample (those who did and do not endorse anger and aggression), anger levels at pretreatment and posttreatment were of mild intensity. Also across the total sample, participants reported an average of less than one physical aggressive act and two to three psychological aggressive acts per month at both pretreatment and posttreatment.

5.2 | Changes in anger and aggression

Changes in anger were correlated with changes in psychological aggression (r= .35, p < .001), but not with changes in physical aggression (r= .12, p= .07). In addition, changes in psychological aggression were positively associated with changes in physical aggression (r= .19, p= .006). Changes in anger, psychological aggression, and physical aggression by treatment group are presented in Table 2. While there were differences between the treatment groups in reductions in anger, psychological aggression, and physical aggression, none of the group by time interactions were significant, indicating that pre- to posttreatment changes did not statistically differ across the treatment groups. Therefore, the data from the treatment groups in Studies 1 and 2 were combined for examining reductions in anger and aggression.

The pooled change in anger from pretreatment to posttreatment was significant at -2.00 points (SE = 0.53), and the standardized mean difference was -0.25 (small effect). The significant pooled effect for changes in psychological aggression from pretreatment to posttreatment was significant at -3.67 points (SE = 0.48) with a standardized mean difference of -0.43 (medium effect). Finally, a significant pooled effect was found for changes in physical aggression from pretreatment to posttreatment, with an effect of -1.26 points (SE = 0.38) and the standardized mean difference of -0.25 (small effect). Based on the presence of significant pooled effects for each outcome, Hypotheses 1a and 1b were supported and showed there were significant decreases in anger and both types of aggression from pretreatment to posttreatment. Finally, the changes in anger and aggression were analyzed while controlling for pretreatment AUDIT scores and ethnicity. None of the substantive conclusions changed; significant pooled effects were found for changes in anger (-1.98), psychological aggression (-3.01), and physical aggression (-0.89).

5.3 | Correlations between changes in PTSD and anger/aggression outcomes

Correlations between changes in PTSD severity and changes in anger, psychological aggression, and physical aggression by treatment group are presented in Table 3. Table 3 also includes information about the heterogeneity of the correlations across treatment groups and, where appropriate, estimates of the pooled correlation. Cochran's Q indicated that there was significant heterogeneity among the correlations between changes in anger and changes in PTSD severity ($X^2(3) = 8.81$, p = .03). \hat{I}^2 showed that 66% of the variance in the observed correlations were due to the treatment groups as opposed to sampling error. Medium to large sized correlations between changes in anger and changes in PTSD were observed for the Study 1 Group PCT (r = .41, p = .004), Study 1 Group CPT (r = .59, p < .001), and Study 2 Group CPT (r = .44, p < .001). The correlation was not significant for the Study 2 Individual CPT (r = .14, p = .24). Pairwise comparisons indicated that the correlation between changes in PTSD symptoms and reductions in anger for Individual CPT was significantly smaller than both Group CPT treatment arms but was not smaller than the Study 1 Group PCT arm. Thus, changes in PTSD symptoms were correlated with changes in anger with the exception of the Individual CPT condition (Hypothesis 2a was partially supported).

Cochran's Q and P^2 statistics indicated homogeneity among the correlations between changes in PTSD severity and changes in both forms of aggression, meaning the treatment groups did not differ in the size of their correlations between changes in PTSD symptoms and the outcomes. Therefore, it was appropriate and afforded more statistical power to analyze all treatment conditions as one group for aggression outcomes. The pooled correlation between changes in PTSD severity and changes in psychological aggression (r = .32, p < .001; medium sized) was significant, as was the pooled correlation between changes in PTSD severity and physical aggression (r = .18, p = .01; small to medium sized). Hypothesis 2b was supported. PCL-5 items 11 and 15 were removed, and correlations between changes in PTSD symptoms and changes in anger and aggression were run again. None of the substantive conclusions changed with correlations being almost identical to those in Table 3.

6 | DISCUSSION

Anger and aggression are serious problems for returning service members and veterans. This study examined a large sample of 374 active duty U.S. military personnel seeking PTSD treatment after a combat-related deployment. Most reported at least mild anger (88%) and one or more acts of psychological aggression (97%) in the month before beginning PTSD treatment. In addition, 32% endorsed engaging in physical aggression in the month before treatment. The incidence rates of anger and aggression are concerning and indicate that a considerable number of service members could benefit from learning strategies to manage these symptoms.

Previous research with civilians and veterans has demonstrated reductions in anger are associated with successful PTSD treatment (e.g., Forbes et al., 2012; Galovski et al., 2014; Larsen et al., 2019). Less attention has been paid to examining if PTSD treatment leads to reductions in aggression. To our knowledge, this is the first study to examine this question in active duty military. Our results demonstrated that anger, psychological aggression, and physical aggression were reduced after PTSD treatment. Controlling for hazardous drinking and ethnicity did not substantially change the results, thus the reductions were not explained by these factors.

The pooled effect sizes of these reductions were consistent with the effect sizes of treatments that directly aim to decrease anger (Morland, 2010) and aggression (Taft et al., 2016). The findings suggest that PTSD treatment may be as effective at reducing anger and aggression as treatments that target these symptoms. However, the reductions were small to moderate in size, and the majority of the sample continued to endorse anger (78%) and psychological aggression (93%) at posttreatment. Clearly, there are additional opportunities for service members to address anger and aggression outside of PTSD treatment.

To examine what mechanisms may be contributing to the reductions in anger and aggression, we correlated the changes in PTSD symptoms from pretreatment to posttreatment with the changes in anger, psychological aggression, and physical aggression. As PTSD symptoms decreased over time with moderate to large effect sizes, so did psychological and physical aggression in all treatment groups. However, the treatment groups were inconsistent in the relationships between changes in PTSD symptoms and changes in anger. Individual CPT had a small, nonsignificant correlation. All other treatment groups had stronger, statistically significant relationships between the changes in symptoms. It is unclear why this would be the case given that individual CPT was overall more efficacious in treating PTSD than group CPT (Resick et al., 2017). Additionally, in the present study service members participating in individual CPT showed the largest reduction in anger, although this decrease was not statistically different than the other treatment groups (see Table 2). One potential reason may be that group treatments allow members to support one another and model prosocial behaviors, which can extend to managing anger in an adaptive manner.

The correlations between changes in PTSD symptoms and changes in anger and aggression were small to moderate in strength. This finding is consistent with Forbes et al. (2003)

who found that anger change scores (pretreatment to posttreatment) did not covary with PCL change scores. PTSD is not the sole mechanism contributing to anger and aggression. Military culture may be another factor. Active duty service members may conceptualize anger and aggression as necessary to complete many of their work tasks (e.g., combat operations, intensive training) and may not be motivated or reinforced by the environment to decrease them. Qualitative interviews with service members may assist in our understanding of the perceived adaptive value of anger and aggression.

In our sample, the correlations between changes in anger and changes in PTSD symptoms appeared larger than the correlations between changes in both types of aggression and changes in PTSD symptoms. Anger and aggression can be influenced by different mechanisms (Barratt, Stanford, Felthous, & Kent, 1997). In the *DSM-IV-TR*, anger and irritability were considered indicators of physiological arousal. The DSM-5 (APA, 2013) separated the emotion from behavior, which is consistent with the current results.

Future research could benefit from examining potential mediators and moderators of the relationship between PTSD symptoms and aggression, including emotion regulation ability and insomnia. Emotion dysregulation mediated the relationship between PTSD symptoms and impulsive aggression in veterans with PTSD (Miles et al., 2015). It is not the presence of intense emotions but rather how one manages the emotions that is critical. Aggressive behavior can be decreased while level of anger remains constant (Barratt et al., 1997). It is also possible that insomnia is related to aggression as it is another symptom of PTSD that is resistant to treatment (Pruiksma et al., 2016). Active duty military are often required to do shift work resulting in a lack of sleep, which can then lead to anger and irritability.

Clinically, the findings that anger only decreased slightly with PTSD treatment and was only moderately correlated with PTSD symptom reduction are important because anger is associated with poorer PTSD treatment outcomes in civilians (Foa et al., 1995) and veterans (Forbes, Creamer, Hawthorne, Allen, & McHugh, 2003). Unfortunately, service members with PTSD have stronger relationships between PTSD and anger than civilians with PTSD (Orth & Wieland, 2006). Higher levels of anger may be one reason service members are likely to retain their PTSD diagnoses after treatment (Steenkamp & Litz, 2013).

For some service members with PTSD, PTSD treatment may be enough to help them manage their anger and aggression. In the present study, this was demonstrated in the mild levels of anger and low frequency of aggression in the overall total sample. However, there was variability in levels of anger and aggression, and even a single aggressive act can result in negative consequences. Providers would benefit from evaluating remaining intensity and frequency of anger and aggression after PTSD treatment. Some individuals will need additional anger- and aggression-focused treatments. Future research should determine which patients would benefit from a multifaceted approach and which psychological symptoms (anger, aggression, or PTSD) should be targeted first.

The results also suggest that novel methods are needed for those with some combination of PTSD, anger, and aggression. Manage Emotions to Reduce Aggression is a three-session emotion regulation treatment that is being piloted for veterans with PTSD and impulsive

aggression. This treatment may serve as a brief therapy option for those who are struggling with aggression (Miles and The Consortium to Alleviate PTSD, 2017; Miles, Thompson, Stanley, & Kent, 2016). Alternatively, mobile or computerized treatments that reduce anger may be helpful adjunctive treatments for some patients. For example, computerized treatments that target hostile interpretation bias may assist the service member in down regulating the survival mode response. Computerized treatments are also convenient to administer (Cougle et al., 2017; Smith, Dillon, & Cougle, 2018).

One limitation of the study was the use of self-report measures of aggression. Aggressors may underreport their aggressive acts because these undesirable behaviors can result in legal or administrative consequences. Despite this possibility, the majority of service members reported anger and psychological aggression. Future studies would be strengthened by using an independent aggression rater. To reduce participant burden in the original STRONG STAR trials, only the state anger subscale of STAXI was administered to assess for current levels of anger. Results of the current study may differ if the trait subscale was used as was done in previous studies (e.g. Galovski et al., 2014). Another limitation was that the DSM-5 was published during the conduct of the two trials. *DSM-IV-TR* criteria were used for the diagnosis of PTSD and study eligibility; however, the PCL-5 was available and used to assess symptom change. Finally, longer follow-up periods would be useful in determining if the reductions in anger and aggression continue with time.

Strengths of the study include the ability to combine and analyze data from two of the largest RCTs ever conducted testing PTSD treatments in active duty military. While any individual study may lack the sample size to detect changes in anger and aggression, the combined sample provided more statistical power. In addition, few exclusion criteria were used in the trials, making the sample more representative of all service members who are seeking treatment for PTSD. Importantly, anger and aggression were measured as separate constructs. In sum, this study demonstrates that PTSD treatment can be effective at reducing anger and aggression in active duty military; however, additional treatments may be needed to fully target the problematic emotion and behaviors.

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TABLE 1Demographic characteristics and symptoms by study

Characteristic	Study 1: Group CPT vs. Group PCT (n = 108)	Study 2: Group CPT vs. Individual CPT (n = 266)	X^2 , F	p-value
Age	32.76 (7.52)	33.20 (7.45)	0.52	.60
Male	100 (93%)	242 (91%)	0.26	.61
Ethnicity			9.68	.02
Black	22 (20%)	75 (28%)		
Hispanic	15 (14%)	61 (23%)		
White	62 (58%)	108 (41%)		
Other	9 (8%)	22 (8%)		
Education			1.96	.38
High school or less	35 (33%)	68 (26%)		
Some college/associate degree	64 (59%)	177 (66%)		
College/graduate degree	9 (8%)	21 (8%)		
Army	108 (100%)	261 (98%)	2.06	.15
Enlisted rank	105 (97%)	258 (97%)	0.01	.94
Months in military	123.82 (76.59)	130.49 (76.12)	0.77	.45
Typical duty			0.64	.72
Combat arms	45 (42%)	99 (37%)		
Combat support	24 (22%)	64 (24%)		
Combat service support	41 (36%)	103 (39%)		
Number of deployments			5.32	.15
1	29 (27%)	76 (29%)		
2	45 (42%)	88 (33%)		
3	24 (22%)	55 (21%)		
4+	10 (9%)	47 (19%)		
Pretreatment symptoms				
PCL-5	50.00 (13.80)	44.60 (12.57)	3.66	<.001
PCL-5 > = 31	99 (92%)	232 (88%)	1.49	.22
Anger (STAXI)	19.82 (8.26)	18.16 (7.99)	1.81	.07
Physical aggression (CTS2)	2.94 (7.26)	1.63 (3.77)	1.77	.08
Psychological aggression (CTS2)	15.15 (8.73)	13.48 (8.58)	1.69	.08
Posttreatment symptoms	(n = 92)	(n = 160)		
PCL-5	39.86 (18.55)	34.82 (19.42)	4.06	.04
PCL-5 > = 31	60 (65%)	96 (60%)	1.48	.22
Anger (STAXI)	17.91 (8.53)	16.37 (7.72)	2.22	.13
Physical aggression (CTS2)	1.22 (2.93)	0.85 (2.97)	1.02	.31
Psychological aggression (CTS2)	11.26 (8.09)	10.21 (7.68)	1.07	.30

Abbreviations: CTS2, Revised Conflict Tactics Scale; PCL-5, Posttraumatic Stress Disorder Checklist for DSM-5; STAXI, State-Trait Anger Expression Inventory.

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TABLE 2Pretreatment to posttreatment changes in outcome variables from mixed models

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Outcome by study/arm						95% CI	
Anger	Effect	StdErr	df	t	p-value	Lower	Upper
Study 1-Group PCT	-2.31	1.19	370	-1.94	.05	-4.65	0.04
Study 1-Group CPT	-1.81	1.23	370	-1.47	.14	-4.23	0.61
Study 2-Group CPT	-1.17	0.86	370	-1.36	.17	-2.86	0.52
Study 2-Individual CPT	-2.69	0.89	370	-3.04	.002	-4.44	-0.95
Overall effect	-2.00	0.53	370	-3.78	<.001	-3.03	-0.96
Overall SMD	-0.25						
Group x Visit f, p, df	0.54	.66	3, 370				
						95% CI	
Psychological aggression	Effect	StdErr	df	t	<i>p</i> -value	Lower	Upper
Study 1-Group PCT	-2.79	1.08	370	-2.58	.01	-4.93	-0.66
Study 1-Group CPT	-5.24	1.12	370	-4.69	<.001	-7.44	-3.05
Study 2-Group CPT	-2.27	0.77	370	-2.93	.003	-3.79	-0.75
Study 2-Individual CPT	-4.37	0.80	370	-5.46	<.001	-5.94	-2.79
Overall effect	-3.67	0.48	370	-7.67	<.001	-4.61	-2.73
Overall SMD	-0.43						
Group x Visit f, p, df	2.18	.09	3, 370				
						95% CI	
Physical aggression	Effect	StdErr	df	t	p	Lower	Upper
Study 1-Group PCT	-2.04	0.73	370	-2.81	.005	-3.47	-0.61
Study 1-Group CPT	-1.47	0.72	370	-2.04	.04	-2.89	-0.06
Study 2-Group CPT	-0.76	0.48	370	-1.58	.11	-1.71	0.19
Study 2-Individual CPT	-0.75	0.49	370	-1.53	.13	-1.71	0.21
Overall effect	-1.26	0.38	370	-4.08	< 0.001	-1.86	-0.65
Overall SMD	-0.25						
Group x Visit f, p, df	0.98	.40	3, 370				

Abbreviations: CI, confidence interval; CPT, Cognitive Processing Therapy; PCT, Present-Centered Therapy; SMD, standardized mean difference; .02, small effect; .05, medium effect; .08, large effect

TABLE 3

Correlations between changes in anger and aggression and changes in PTSD symptom severity and estimates of meta-analytic effects

_			95% CI		
Correlated outcomes by treatment and arm	r between change scores	Z	<i>p</i> -value	Lower	Upper
Anger and PTSD					
Study 1-Group PCT	.41 ^{ab}	2.91	.004	.14	.62
Study 1-Group CPT	.59 ^a	4.37	<.001	.36	.76
Study 2-Group CPT	.44 ^a	4.24	<.001	.25	.60
Study 2-Individual CPT	.14 ^b	1.17	.24	09	.35
Overall effect		_	_	-	_
Heterogeneity (Q/P)	8.81 */65.95				
			95% CI		
Psychological aggression and PTSD	r between change scores	Z	<i>p</i> -value	Lower	Upper
Study 1-Group PCT	.25	1.73	.08	03	.50
Study 1-Group CPT	.29	1.95	.05	.00	.54
Study 2-Group CPT	.37	3.52	<.001	.17	.54
Study 2-Individual CPT	.31	2.73	.006	.09	.50
Overall effect	.32	5.10	<.001	.20	.43
Heterogeneity (Q/P)	0.57 ^{ns} /0.00				
Dhysical aggression	r between			95% CI	
Physical aggression and PTSD	change scores	Z	<i>p</i> -value	Lower	Upper
Study 1-Group PCT	04	24	.81	32	.25
Study 1-Group CPT	.27	1.78	.08	03	.53
Study 2-Group CPT	.14	1.23	.22	08	.34
Study 2-Individual CPT	.29	2.54	.01	.07	.48
Overall effect	.18	2.74	.01	.05	.29
Heterogeneity (Q/I²)	3.65 ^{ns} /17.85				

Note: Z statistics are the normal curve test of the significant of the correlation coefficient. Tests for Q-values are with df = 3. Groups with different superscripts within an outcome are significantly different at .05 level. Superscripts are only provided for outcomes that lacked homogeneity of effect sizes (p value for Q is less than .05); consequently, a meta-analytic estimate of the overall effect size is not included given the inappropriateness of calculating such an effect across groups. \hat{P} represents proportion of total variance in effect sizes due to variance between the treatment arms and not due to sampling error.

** p<.01

*** p<.00

Abbreviations: CI, confidence interval; CPT, Cognitive Processing Therapy; PCT, Present-Centered Therapy; PTSD, posttraumatic stress disorder; ns, not significant.