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PTSD and anger: Evaluation of an indirect effect model in a civilian trauma sample

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ABSTRACT

Background and objectives: Theoretical models propose that PTSD symptoms and subjective anger are indirectly associated through hostile attribution bias, physiological reactivity, and aggressive psycho-motor scripts (Chemtob, Novaco, Hamada, Gross, & Smith, 1997). Originally developed to account for symptoms observed in military personnel, proposed anger mechanisms have received limited attention in civilian populations. The current study looked to evaluate the generalizability of Chemtob et al.'s model in trauma-exposed university students (N = 152).

Methods: Trauma exposure and corresponding symptoms were assessed during an initial screening procedure. Hostile attributions and aggressive scripts were examined prior to a laboratory-based anger induction procedure. Physiological reactivity was monitored throughout the provocation task. Ratings of subjective anger and anger recovery were completed following the induction period. Relations of post-trauma symptoms with subjective anger through hypothesized anger processes were examined using bootstrapped estimates of indirect effects.

Results: A significant indirect effect of PTSD severity on state-level anger was noted for hostile attribution bias (ab = 0.020, 95% CI [0.002, 0.041]) and a marginal effect through aggressive inclinations (ab = 0.015, 95% CI [-0.001, 0.039]). Data failed to provide evidence for physiological reactivity as an intervening variable. Trauma symptoms did not moderate anger recovery following the provocation task.

Limitations: Induction of anger in a sub-clinical sample may limit tests of hypothesized effects and the generalizability of the present findings.

Conclusions: Results indicate the proposed model may be applicable beyond combat trauma samples and suggest potential anger-related targets for PTSD treatment.

1. Introduction

Research evaluating the affective consequences of trauma exposure has primarily focused on the emotions of fear and anxiety. Though anxiety is believed to be central to the etiology of posttraumatic stress disorder (PTSD), prominent theoretical models suggest that other affective states may be relevant to a comprehensive understanding of post-trauma functioning (e.g., Resick & Schnicke, 1992). Anger, in particular, has received considerable attention in the extant literature, with multiple studies indicating associations between post-trauma symptoms and elevated anger (e.g., Orth & Wieland, 2006). In a model outlining the functional relation between anger and PTSD in military veterans, Chemtob, Novaco, Hamada, Gross, and Smith (1997) posit that trauma symptoms are indirectly related to subsequent anger through hostile attribution biases, physiological reactivity, and aggressive psycho-motor scripts. However, direct evaluation of this model is limited, particularly in survivors of non-combat trauma. Aims of the current study were to examine the indirect effects of post-trauma symptoms on subjective anger through mechanisms proposed by Chemtob et al. in university students reporting exposure to significant trauma.

1.1. Anger in PTSD

Elevations in anger have been proposed as a core symptom of PTSD since its introduction in DSM-III (American Psychiatric Association, 1980). Early research with Vietnam-era veterans noted that those with PTSD evidenced elevated levels of trait anger, anger expression, and hostility compared to both combat-exposed veterans with no psychiatric diagnosis and non-combat personnel presenting with other psychological conditions (Chemtob, Hamada, Roitblat, & Muraoka, 1994). Similar associations have been noted in veterans from other eras (e.g.,

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Jakupcak, et al., 2007; Renshaw & Kiddie, 2012; Teten et al., 2010), with meta-analyses by Orth and Wieland (2006) indicating consistent relations between combat-related PTSD and subjective anger.

1.2. Theoretical model of PTSD and anger

Whereas the existing literature provides a clear link between post-trauma symptoms and self-reported anger, the specific processes which account for this association remain unclear. The limited research in this area is guided by a model developed by Chemtob et al. (1997) to describe the relation between post-trauma symptoms and anger in military veterans. Similar to cognitive models of post-trauma psychopathology (e.g., Ehlers & Clark, 2000), Chemtob et al. propose that the core symptoms of PTSD are characterized by a sense of ongoing threat to the survivor. This perception of continued and immediate threat is believed to drive changes in hostile attribution biases, physiological reactivity, and aggressive psycho-motor scripts which potentiate the experience of anger in response to interpersonal conflict.

1.2.1. Hostile attribution bias

Chemtob et al. (1997) posit that individuals experiencing active trauma symptoms may exhibit characteristic biases in the attribution of others' intent. Specifically, survivors are believed to interpret the behavior of others in ambiguous interactions as intended to cause direct personal harm. Hostile attribution biases may be explicit or automatic but are thought to potentiate the subjective experience of anger as a response to perceived social conflict.

Existing research in veteran samples provides evidence for the proposed link between PTSD and hostile attribution biases. Novaco and Chemtob (2002) noted strong associations between post-trauma symptoms and self-reported suspicion and hostility among Vietnam-era veterans. Other studies suggest veterans with PTSD are more likely to report elevations in trait-level resentment, hostility, and suspicion than service members with no diagnosis (Beckham et al., 1996; Beckham et al., 2002; Lasko, Gurvits, Kuhne, Orr, & Pitman, 1994; Teten et al., 2010).

1.2.2. Physiological reactivity

Chemtob et al. (1997) also identify physiological reactivity to threat and/or provocation as an intervening variable in the relation between PTSD and anger. Trauma-related symptoms are thought to potentiate elevations in physiological arousal in response to anger-inducing stimuli. Increases in the intensity and duration of physiological reactions are believed to escalate the experience of subjective anger during frustrating encounters.

Preliminary research provides tentative support for relations between PTSD and anger-related physiological reactivity. Novaco and Chemtob (2002) identified associations between the severity of post-trauma symptoms and self-reported physiological arousal among Vietnam veterans. Beckham et al. (2002) also provide evidence of elevated cardiovascular reactivity in veterans completing an anger rumination task. In this study, participants were instructed to recall a time where they had felt angry with another person and were unsatisfied with the outcome. Veterans meeting formal diagnosis for PTSD exhibited greater increases in diastolic blood pressure and subjective anger in response to the provocation task than combat-exposed veterans who did not meet diagnostic threshold.

1.2.3. Psycho-motor scripts

A final mechanism identified in Chemtob et al.'s (1997) model is the activation of aggressive psycho-motor scripts. Specifically, trauma-related symptoms are thought to potentiate confrontational and aggressive strategies for addressing interpersonal conflict. Activation of scripts is assessed through reported thoughts of aggressive impulses and perceived difficulty inhibiting these behaviors. Aggressive psycho-motor scripts are believed to exacerbate feelings of subjective anger within the context of PTSD.

Behavioral scripts have received limited attention in the present literature due to their similarity to verbal and physical aggression. However, existing research in veteran samples does provide evidence of strong relations between post-trauma symptoms and self-reported impulsivity, displacement of expressions of anger onto other targets, and aggressive body language (Novaco & Chemtob, 2002). Further studies note reliable associations between PTSD and increased effort to control aggressive inclinations, difficulties managing anger, and increased aggressive urges (Beckham et al., 1996; Elbogen et al., 2010).

1.3. Evidence in civilian samples

As Chemtob et al.'s (1997) framework was developed to account for the relation of trauma reactions and anger-related processes in veteran populations, specific mechanisms have received limited attention in mixed-gender samples and survivors of civilian trauma. However, existing research does provide evidence for general relations between post-trauma symptoms and subjective anger in non-military samples, including survivors of sexual assault (Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992), violent crime (Orth, Cahill, Foa, & Maercker, 2008), and motor vehicle trauma (Ehlers, Mayou, & Bryant, 1998). Given that perceptions of ongoing threat are an underlying assumption in several prominent models of PTSD (e.g., Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989), it is reasonable to hypothesize that processes identified by Chemtob and colleagues may generalize to individuals exposed to civilian trauma.

Relations between post-trauma symptoms, attributional biases, and physiological reactivity have also been noted in non-military samples. For example, data suggest that female survivors of sexual assault evidence levels of hostile appraisal commensurate to those observed in veterans with combat-related PTSD (Castillo, Fallon, C'de Baca, Conforti, & Qualls, 2002). Research by Vrana, Hughes, Dennis, Calhoun, and Beckham (2009) further indicate that women meeting diagnostic threshold for PTSD exhibit greater elevations in hostile beliefs and cardiovascular reactivity following an anger rumination task than survivors with no PTSD diagnosis. Although Chemtob et al. (1997) do not specifically address the role of gender in their model of PTSD and anger, existing data offers preliminary support for generalization of proposed relations to mixed-gender and non-military samples. However, no full evaluation of Chemtob et al.'s (1997) model has been undertaken among civilian trauma survivors.

1.4. The current study

The aim of the current study was to evaluate the extent to which processes proposed by Chemtob et al. (1997) function as intervening variables in the relation of probable posttrauma symptoms and state anger following an anger induction task. Individuals endorsing a history of probable trauma and ongoing symptoms associated with a Criterion-A event (American Psychiatric Association, 2013) were recruited from undergraduate psychology courses. Self-report measures of hostile attribution bias and aggressive psycho-motor scripts were collected prior to completing an anger induction task. Indices of heart rate, blood pressure, and respiration were recorded throughout the induction procedure, with state anger and anger recovery assessed following the manipulation. Post-trauma symptoms assessed during the initial screening were expected to demonstrate indirect relations with state anger during the manipulation through indices of hostile attribution bias, physiological reactivity, and aggressive psycho-motor scripts. Post-trauma symptom severity was also expected to moderate decreases in subjective anger during a subsequent recovery period such that those with higher symptom levels would show more modest reductions in anger at the conclusion of the protocol. Participant sex was included as a covariate for all analyses to examine its relations with variables described in Chemtob et al.'s model.

2. Method

2.1. Participants

Participants included university students reporting ongoing distress in response to probable Criterion-A trauma (e.g., exposure to actual or threatened death, physical injury, or sexual violence; American Psychiatric Association, 2013) reported during a preliminary screening. Of the 153 individuals presenting for participation, one was excluded given evidence of active suicidal ideation. Physiological data for six individuals were also lost due to equipment failure. As a result, analyses involving physiological measures were conducted in a subset of $N = 146$ individuals, whereas models involving hostile attribution bias and behavioral scripts utilized the full sample of $N = 152$. Participants with missing physiological scores did not reliably differ from those with full data with respect to age, gender, minority status, or global PTSD symptoms (all $p \geq .584$). Participants in the final sample identified predominantly as White/Non-Hispanic (83.6%) and female (73.7%). Full sample characteristics are available in Table 1.

2.2. Measures

2.2.1. Trauma screening

Probable trauma history was assessed using a brief screening measure in which respondents indicated lifetime exposure to natural disaster, fire, traffic accidents, physical assault, and/or sexual assault. A final open-ended item permitted endorsement of Criterion-A events not included in the checklist. Ongoing distress in response to endorsed events was rated on a 1 to 5 Likert scale (1 = *None;* 5 = *Extremely*). Respondents were also asked to indicate which event of those identified was associated with the greatest level of continued distress.

2.2.2. PTSD checklist for DSM-5 (PCL-5)

The PCL-5 is a 20-item, self-report measure assessing cardinal symptoms of DSM-5 PTSD (Weathers et al., 2013). Items are rated on a 5-point Likert scale (0 = *Not at all*; 4 = *Extremely*) and summed to form an index of overall symptom severity. Preliminary research provides evidence for the factorial validity of PCL-5 scores in undergraduates reporting probable trauma (Armour et al., 2015; Blevins, Weathers, Davis, Witte, & Domino, 2015). The PCL-5 was administered following the initial trauma screening measure. Respondents were instructed to complete the PCL-5 with reference to the event associated with the most current distress. Approximately 30% of participants ($n = 44$) reported PCL-5 scores exceeding recommended cut scores for probable diagnosis of PTSD (PCL-5 ≥ 33; Weathers et al., 2013).

Table 1
Sample characteristics ($N = 152$).

Age	20.01 (3.01)
Sex (% female)	73.7%
Race/Ethnicity	
Caucasian	83.6%
Hispanic	9.9%
African American	2.0%
Asian	2.0%
Native American	1.4%
Other	1.3%
Index Event	
Sexual Assault	38.2%
Motor Vehicle Accident	31.6%
Physical Assault	14.5%
Disaster	5.3%
Fire	4.6%
Suicide	2.6%
Medical/Illness	1.3%
Emotional Abuse/Threatened Violence	1.3%
Witnessed death/injury	0.7%

2.2.3. Social Information Processing-Attribution and Emotional response Questionnaire (SIP-AEQ)

The SIP-AEQ (Coccaro, Noblett, & McCloskey, 2009) was administered as an index of hostile attribution bias. Respondents are asked to imagine eight vignettes involving ambiguous social situations (e.g., someone spilling coffee on them) and rate the likelihood that the actions of a hypothetical individual were directly hostile (i.e., *My co-worker wanted to burn me with the hot coffee.*). Items are rated on a 4-point Likert scale, (0 = *Not at all likely*; 3 = *Very likely*) and averaged to form a total score. The Hostile Attribution Bias scale of the SIP-AEQ demonstrated strong convergent and discriminant validity in previous research (Coccaro et al., 2009).

2.2.4. Physiological arousal

Existing research has identified cardiovascular reactivity including heart rate, blood pressure, and respiration rate as correlates of anger (Kreibig, 2010). Physiological measures were recorded using Biopac MP150 modules and a 500 series CNAP Monitor. Scores were screened off-line for movement errors and invalid values were removed. Continuous scores were averaged for baseline and rumination epochs and used to calculate percentage change (Response = [Manipulation – Baseline]/Baseline). Positive scores indicate an increase in physiological activation in response to the anger manipulation.

2.2.4.1. Cardiovascular response. Systolic blood pressure, diastolic blood pressure, and heart rate were assessed using cuffs placed on the upper arm and first two fingers of the non-dominant hand. Blood pressure and heart rate were sampled at 1000 Hz, which is considered standard for cardiovascular measures (Bhatia, Rarick, & Strauss, 2010). Heart rate was measured as inter-beat interval (IBI) and calculated as the mean time (s) between cardiac contractions.

2.2.4.2. Respiration. Respiration rate was assessed using a Biopac BN-RESP-XDCR module, sampled at a rate of 32 Hz. Respiration rate was measured as the mean time (s) between inhalations.

2.2.5. Hostile automatic thoughts (HAT) scale

Aggressive psycho-motor scripts were assessed using the Physical Aggression subscale of the HAT Scale (Snyder, Crowson, Houston, Kurylo, & Poirier, 1997). This scale measures the frequency of negative automatic thoughts about perpetrating physical aggression during social interactions. Respondents are asked to rate the frequency of thoughts over the past week (1 = *Not at all*; 5 = *All the time*). The subscale's eleven items are summed to create a total score. Previous research with this subscale suggests excellent internal consistency ($\alpha = 0.92$; Snyder et al., 1997).

2.2.6. State Anger Scale (SAS)

The SAS, a component of the State-Trait Anger Scale (Spielberger, Jacobs, Russel, & Crane, 1983), was administered as a measure of anger response to the rumination task. The SAS consists of 10 anger-related statements (e.g., "I feel angry") rated on a 4-point Likert scale (1 = *Not at all*; 4 = *Very much so*), summed to create a total score. Participants completed the SAS with respect to the level of subjective anger experienced during the rumination task.

2.3. Procedure

2.3.1. Screening

The trauma screener and PCL-5 were administered during a mass testing procedure. Students reporting ongoing distress in response to a probable Criterion-A event were recruited for participation. Eligible individuals received an email inviting them to participate in a study evaluating "physical and psychological responses surrounding emotional processing." Enrollment was coordinated using online participant management software.

2.3.2. Consent and questionnaires

Upon arrival to the lab, students were reminded of the purpose of the study. They then gave consent for participation and completed a battery of questionnaires. Participants were also asked to describe an anger-provoking memory using instructions similar to those employed by Beckham et al. (2002):

> Now recall a situation when you felt really angry, frustrated, or upset with another person. Choose a memory where you were unhappy with the outcome and feel that the situation remains unresolved.

Consistent with existing research, instructions were intended to prompt memories of interpersonal conflict or disagreement. Elicitation of a non-traumatic anger memory was intended to prevent the conflation of generalized anger with reactions linked directly to the reported exposure.

2.3.3. Physiological arousal manipulation

Following the completion of initial self-report measures, researchers aided participants in attaching physiological sensors. Students were instructed to sit quietly for a period of 10 min to permit habituation to the placement of electrodes. Following the adaptation period, participants were given an anger rating form and received the following pre-recorded instructions:

> For the next several minutes, we would like you to relax. In a moment, you will hear a tone. When you hear the tone, please sit back, close your eyes, and clear your mind.

A brief tone was administered (200 Hz, 0.25s) following a 10s delay to account for immediate orientation response to the pre-recorded instructions (Bradley, 2009). Baseline physiological data were gathered for a 2-min period following the tone. Upon completion of the baseline period, participants received instructions corresponding to the primary anger manipulation:

> In a moment, you will hear another tone. When you hear the tone, recall the angry memory you were asked to write about previously. Begin thinking about the memory, the details of the situation, and how angry you felt in that moment.

A neutral tone sounded after an additional 10 s delay, and participants were given a period of 2 min to focus on their angry memory. A final set of instructions followed:

> We would like you to stop thinking about your angry memory at this time, relax, and pick up the clipboard beside you. Remove the blank cover sheet from the clipboard. In a moment, you will hear a series of tones. Using the sheet provided, please rate your current level of anger, at that moment, each time you hear a tone. On this scale, 0 indicates no anger and 10 indicates maximum anger. Again, please rate your current level of anger, at that moment, each time you hear a tone.

Instructions were followed by a 10s delay. Tones were subsequently presented at 20s intervals over a period of 2 min to assess reductions in subjective anger. After presentation of the final tone, participants were informed:

> You have completed this portion of the study. Please continue to sit still and an experimenter will be with you shortly.

The researcher returned to the room to assist the participant in removing the physiological sensors, administer the SAS, and deliver a short debriefing questionnaire. Items from this measure assessed for prior knowledge of the procedure, levels of baseline anger upon entering the lab (0 = *No Anger*, 10 = *Extreme Anger*), and peak anger in response to the manipulation (0 = *No Anger*, 10 = *Extreme Anger*).

2.4. Analytic approach

2.4.1. Analyses of indirect effects

Three models of indirect effect were used to examine the relation between PTSD severity and anger response through hostile attributions, physiological reactivity, and behavioral scripts. For these analyses, PCL-5 scores were modeled as the primary independent variable, with state anger serving as the dependent variable. Interpersonal attribution (SIP-AEQ Hostile Attribution), reactivity (percent change in blood pressure, inter-beat interval, and respiration), and script (HAT Physical Aggression) indicators were examined as intervening variables in successive models to evaluate the indirect relation of post-trauma symptoms with subjective anger. Participant sex was included as a covariate in all analyses to control for possible differences in anger processes between men and women.

Intervening variable models were evaluated using Hayes' PROCESS macro (Model 8; Hayes, 2012) for SPSS. Analyses conducted in PROCESS utilize bootstrapping methods to test the statistical significance of indirect effects. Bootstrapping procedures address known biases in traditional tests of mediation (e.g., Sobel, 1982) and are considered a best-practice approach for the evaluation of indirect effects (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). For the current study, bootstrapped confidence intervals were calculated for point estimates of indirect effects using 5000 resamples from the original data. Confidence intervals failing to capture a null estimate were interpreted as statistically significant.

Effect sizes for individual regression coefficients are presented as both partial (*pr*) and squared semi-partial (sr^2) correlations. Partial correlations represent the strength of a unique association controlling for other predictors in the model and serve as a standardized index of effect. Values of pr = .14, pr = .36, and pr = .51 serve as benchmarks for small, medium, and large effects, respectively (Cohen, 1988). Squared semi-partial correlations provide an alternative metric of effect and reflect the percentage of total variability in the outcome uniquely shared with a predictor.

2.4.2. Moderation effects

Chemtob et al. (1997) suggests that post-trauma symptoms are related to broad deficits in the top-down regulation of anger, potentially increasing the duration of the anger response. As such, an additional regression model was used to examine the impact of post-trauma symptoms and ratings of peak anger on anger recovery during the post-manipulation cooldown period. Initial anger ratings recorded during the cooldown period (time 1) were modeled as the primary independent variable with the final anger rating (time 6) serving as the outcome. PCL-5 scores and ratings of peak anger (collected during debriefing) were examined as moderators of this relation, controlling for participant sex (Aiken & West, 1991).

3. Results

3.1. Data screening

All data were screened prior to analysis to determine the concordance of scores with the assumptions of multivariate analysis. The distribution of the HAT Scale exhibited a potential violation of univariate normality (skew = 1.863, kurtosis = 3.52). A visual inspection of these data indicated notable positive skew. A natural log transform was applied to improve the scale's distributional properties (skew = 1.21; kurtosis = 0.71). Univariate outliers were noted in the distributions of percent change in SBP, DBP, and IBI ($z \geq |3.44|$). Three extreme scores for SBP, three scores for DBP, and one score for IBI were smoothed by assigning these cases a value one unit greater than the adjacent score. This approach maintains the rank order of scores, consistent with recommendations for addressing problematic distributions (Tabachnick & Fidell, 2013). Subsequent screening identified little

Table 2
Means, standard deviations, and correlations between primary study variables.

Measure	Correlations									
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
1: PCL-5	–									
2: SAS	0.018	–								
3: TAS	0.167*	0.252*	–							
4: SIP-AEQ	0.161*	0.307**	0.418***	–						
5: PC-SBP	−0.095	0.001	−0.008	0.010	–					
6: PC-DBP	−0.048	0.132	0.099	0.078	0.599***	–				
7: PC-IBI	0.013	−0.142	−0.095	−0.086	0.029	−0.160†	–			
8: PC-RP	0.054	−0.150†	−0.130	−0.069	−0.078	−0.200*	0.117	–		
9: ln HAT	0.158†	0.193*	0.615**	0.310***	0.078	0.160†	−0.009	−0.020	–	
10: Sex	−0.162*	−0.104	0.046	0.024	0.007	0.015	−0.018	−0.020	0.133	–
M	25.68	18.21	17.70	0.93	0.01	0.06	−0.03	−0.14	2.67	0.26
SD	16.08	5.96	4.18	0.39	0.04	0.04	0.04	0.17	0.30	0.44

†p ≤ .055, *p < .05, **p < .01, ***p < .001.
Note: PCL-5 = PTSD Checklist for DSM-5; SIP-AEQ = Social Information Processing-Attribution and Emotion Questionnaire; SAS = State Anger Scale; TAS = Trait Anger Scale.
PC-SBP = Percent Change in Systolic Blood Pressure; PC-DBP = Percent Change in Diastolic Blood Pressure; PC-IBI = Percent Change in Interbeat Interval; PC-RP: Percent Change in Respiration Period; ln HAT Scale = natural log transform of Hostile Automatic Thoughts Scale; Sex: Male = 1, Female = 0.

Table 3
Change and effect size across baseline and anger epochs.

	Baseline (*M, SD*)	Anger (*M, SD*)	M_A - M_B	*95% CI*	Δ[a]
Subjective Anger	0.74 (1.35)	5.24 (2.16)	4.50	[4.16, 4.83]	3.32
SBP	118.82 (15.59)	119.05 (16.00)	0.23	[-0.73, 2.55]	0.02
DBP	68.60 (10.52)	68.81 (10.84)	0.21	[-0.77, 0.35]	0.06
IBI	0.84 (0.13)	0.82 (0.13)	−0.03	[-0.03, −0.02]	0.19
RP	4.72 (1.54)	3.89 (0.95)	−0.84	[-1.05, −0.63]	0.54

Note: SBP = Systolic Blood Pressure; DBP = Diastolic blood pressure; IBI = Interbeat Interval; RP: Respiration Period; HAT Scale = Hostile Automatic Thoughts Scale.
[a] Given the presence of meaningful baseline values, effect sizes were calculated as Glass's Δ using baseline standard deviation as the standardizer [$(M_A - M_B)/s_{baseline}$].

concern with respect to violations of linearity, homoscedasticity, or multicollinearity. Means, standard deviations, and correlations for primary study variables are provided in Table 2.

3.2. Manipulation check

3.2.1. Anger situations

Target memories identified for the rumination task were reviewed to determine whether recollections were associated with Criterion-A events. Students primarily reported past interpersonal conflicts (86.2%) or other frustrating situations (e.g., academic achievement). Two individuals (1.7%) reported memories that may have served as cues for identified index events. An additional participant selected a violent altercation as the target memory, although this assault was not identified as the index event in the initial trauma screen. As the removal of these individuals demonstrated minimal effect on the final results, cases were retained for all analyses.

3.2.2. Anger induction

Preliminary analyses verified that the anger induction task was effective in inducing emotional and physiological change (see Table 3). Comparisons of baseline emotion with peak response to the recall task indicated significant elevations in reported anger ($p < .001$; $\Delta = 3.32$).[1] Decreases in IBI ($p < .001$; $\Delta = 0.19$) and respiration interval ($p < .001$; $\Delta = 0.54$) across baseline and anger epochs were also consistent with increased physiological activation. Mean SBP ($p = .276$; $\Delta = 0.02$) and DBP ($p = .455$; $\Delta = 0.06$) were relatively insensitive to the anger manipulation.

3.3. Models of indirect effect: anger processes

3.3.1. Hostile attribution bias

An initial model assessed the indirect effect of PTSD severity on state anger through hostile attribution bias, controlling for participant sex (see Table 4). The relation between PCL-5 scores and hostile attribution bias was consistent with a small effect ($p = .048$, pr = .161), with hostile attribution bias demonstrating a small-to-medium association with anger ($p < .001$, pr = .308). No reliable relations were noted between model variables and participant sex. Consistent with hypotheses, the bootstrapped confidence interval for the indirect effect of PTSD on subjective anger through hostile attribution was significant in these data (*ab* = 0.019, 95% CI [0.002, 0.041], see Fig. 1).

3.3.2. Physiological reactivity

A second model tested the indirect effect of PTSD severity on state anger via physiological reactivity (see Table 5). PCL-5 scores failed to demonstrate reliable associations with percent change in SBP, DBP, IBI, or respiration rate (all $p \geq .240$). Percent change in SBP, DBP, IBI, and respiration also failed to demonstrate reliable associations with ratings of subjective anger (all $p \geq .153$). Participant sex did not demonstrate statistically significant relations with variables in these models. Confidence interval estimates for the specific indirect effects all contained null values.

[1] Effect sizes were calculated as Glass's Δ, using the standard deviation at baseline assessment as a standardizer. Interpretive benchmarks are consistent with those for Cohen's *d* (small = 0.20, medium = 0.50, large = 0.80).

Table 4
Coefficients, confidence intervals, and effect sizes for indirect effects of hostile attribution bias.

Outcome	Predictor	*b(SE)*	95% CI	*p*-value	*pr*	sr^2
SIP-AEQ						
R^2 = .029	PCL-5	.004 (.002)	[.001, .008]	.040	.168	.028
	Sex	.047 (.073)	[-.098, .191]	.526	.052	.003
SAS						
R^2 = .109	SIP-AEQ	4.80 (1.19)	[2.457, 7.153]	< .001	.315	.099
	PCL-5	-.019 (.030)	[-.078, .039]	.512	.054	.003
	Sex	−1.63 (1.06)	[-3.723, .473]	.128	.125	.014
Indirect Effect						
	SIP-AEQ	.020 (.010)	[.002, .041]			

Note: PCL-5 = PTSD Checklist for DSM-5; SIP-AEQ = Social Information Processing-Attribution and Emotion Questionnaire; SAS = State Anger Scale; Sex: Male = 1, Female = 0.
Interpretive benchmarks for *pr*: small ± 0.14, medium ± 0.36, and large ± 0.51.

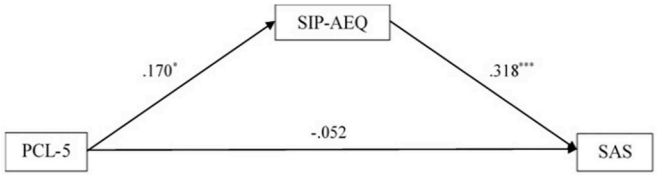

Fig. 1. Indirect effect of PTSD severity and subjective anger through hostile attribution bias. Path coefficients reflect standardized effects. PCL-5 = PTSD Checklist for DSM-5; SIP-AEQ = Social Information Processing-Attribution and Emotion Questionnaire; SAS = State Anger Scale $^{*}p < .05$, $^{***}p < .001$.
Note: Participant sex included as a covariate for all analyses.

3.3.3. Aggressive psycho-motor scripts

A third model examined the indirect effect of PTSD severity on subjective anger through aggressive psycho-motor scripts (see Table 6). The relation between PCL-5 scores and the natural log transform of HAT Physical Aggression was statistically significant (p = .024, pr = .184), with the point-estimate of this association consistent with standards for a small effect. Participant sex also demonstrated a significant relation with log-transformed HAT Scale scores, with men exhibiting elevated response on the measure (p = .046, pr = .163). Increases in log-transformed HAT scores were associated with increased anger response (p = .018, pr = .193). The indirect effect of PTSD severity on subjective anger through behavioral scripts approached significance in this sample (*ab* = 0.015, 95% CI [-0.001, 0.039], see Fig. 2).

3.4. Moderation model

A final model examined the impact of post-trauma symptom severity and peak anger ratings on anger recovery, controlling for participant sex. Results indicated a main effect of peak anger on anger scores at the final assessment point (p = .009, pr = .237). However, main effects for PCL-5 scores (p = .602; pr = −0.048), initial anger (p = .461; pr = −0.067), and participant sex (p = .124, pr = −0.140) failed to reach statistical significance. Results failed to support post-trauma symptoms (p = .893) or peak anger (p = .166) as moderators of the relation between initial and final anger ratings during the cooldown period.

4. Discussion

Existing data suggest that individuals reporting active trauma symptoms exhibit an increased propensity for anger experience (Orth & Wieland, 2006). The current project sought to determine the extent to

Table 5
Coefficients, confidence intervals, and effect sizes for indirect effects of cardiovascular arousal.

Outcome	Predictor	*b(SE)*	95% CI	*p*-value	*pr*	sr^2
PC-SBP						
R^2 = .010	PCL-5	-.001 (.001)	[-.001, .001]	.240	.098	.010
	Sex	-.001 (.009)	[-.019, .017]	.911	.001	< .001
PC-DBP						
R^2 = .001	PCL-5	-.001 (.001)	[-.001, .001]	.701	.032	.001
	Sex	.001 (.010)	[-.019, .022]	.906	.010	< .001
PC-IBI						
R^2 < .001	PCL-5	.001 (.001)	[-.001, .001]	.903	.010	< .001
	Sex	-.002 (.008)	[-.018, .015]	.847	.016	< .001
PC-RP						
R^2 = .003	PCL-5	.001 (.001)	[-.001, .003]	.500	.052	.003
	Sex	-.004 (.033)	[-.070, .063]	.917	.012	< .001
SAS						
R^2 = .038	PC-SBP	−8.96 (14.33)	[-37.30, 19.37]	.533	.053	.003
	PC-DBP	11.34 (12.66)	[-13.70, 36.37]	.372	.076	.005
	PC-IBI	−15.88 (11.87)	[-39.34, 7.59]	.183	.113	.012
	PC-RP	−4.14 (2.88)	[-9.82, 1.55]	.153	.121	.014
	PCL-5	.011 (.032)	[-.052, .075]	.533	.030	.001
	Sex	−1.62 (1.13)	[-3.849, .611]	.153	.121	.014
Indirect Effect						
	PC-SBP	.003 (.005)	[-.007, .015]			
	PC-DBP	-.001 (.007)	[-.014, .014]			
	PC-IBI	-.001 (.005)	[-.011, .010]			
	PC-RP	-.003 (.005)	[-.014, .005]			

Note: PCL-5 = PTSD Checklist for DSM-5; SAS = State Anger Scale; PC-SBP = Percent Change in Systolic Blood Pressure; PC-DBP = Percent Change in Diastolic Blood Pressure; PC-IBI = Percent Change in Interbeat Interval; PC-RP: Percent Change in Respiration Period; Sex: Male = 1, Female = 0; Interpretive benchmarks for pr: small = ± 0.14, medium = ± 0.36, and large = ± 0.51.

Table 6
Coefficients, confidence intervals, and effect sizes for indirect effects of aggressive behavioral scripts.

Outcome	Predictor	*b(SE)*	95% CI	*p*-value	*pr*	sr^2
HAT Scale						
R^2 = .051	PCL-5	.003 (.002)	[.001, .006]	.024	.184	.033
	Sex	.110 (.054)	[.002, .217]	.046	.163	.026
SAS						
R^2 = .056	ln HAT Scale	4.37 (1.64)	[1.117, 7.615]	.009	.213	.045
	PCL-5	-.014 (.031)	[-.075, .046]	.640	.038	.001
	Sex	−1.88 (1.11)	[-4.066, .306]	.091	.138	.018
Indirect Effect						
	HAT Scale	.015 (.010)	[-.001, .039]			

Note: PCL-5 = PTSD Checklist for DSM-5; SAS = State Anger Scale.
HAT Scale = Hostile Automatic Thoughts Scale; Sex: Male = 1, Female = 0.
Interpretive benchmarks for *pr*: small = ± 0.14, medium = ± 0.36, and large = ± 0.51.

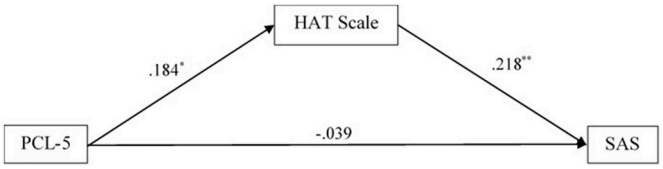

Fig. 2. Indirect effects of PTSD severity on subjective anger through aggressive inclinations. Path coefficients represent standardized effects. PCL-5 = PTSD Checklist for DSM-5; HAT Scale = Hostile Automatic Thoughts Scale; SAS = State Anger Scale $^{*}p < .05$, $^{**}p < .01$.
Note: Participant sex included as a covariate for all analyses.

which factors proposed to account for the relation between post-trauma symptoms and anger in military populations generalize to survivors of non-combat trauma. Consistent with Chemtob et al., (1997) hypotheses, cognitive biases and aggressive psycho-motor scripts were identified as intervening variables in the relation of posttraumatic symptoms and state anger in response to an anger induction. By contrast, indirect effects of posttrauma reactions on state anger through measures of physiological reactivity did not reach statistical significance in these data. Trauma symptoms also failed to moderate anger recovery as assessed through the relation of initial and final ratings of anger. Finally, participant sex demonstrated minimal relations with study variables, although men did report elevations in aggressive psycho-motor scripts as compared to women.

Results add to a growing consensus that while posttrauma symptoms appear to exhibit stronger relations with anger in veteran as compared to civilian samples (Orth & Wieland, 2006), those feelings in both groups are likely connected through similar mechanisms. In particular, evidence of an indirect path through cognitive biases suggests that post-trauma symptoms are associated with greater expectations of harm from others, which potentiate subsequent feelings of anger. Whereas attributions of hostile intent might be expected in survivors of interpersonal trauma (e.g., physical or sexual assault), it is worth noting that a substantial minority of participants in this study identified index events that are unlikely to have involved intentional harm from others (e.g., motor vehicle accidents, disasters). Results are consistent with theoretical models identifying perceptions of ongoing threat as a central feature of PTSD (i.e., Ehlers & Clark, 2000), as well as research demonstrating associations between posttrauma symptoms and hostile attribution biases among survivors of non-assaultive trauma (e.g., Heinrichs, et al., 2005).

The indirect effect of trauma symptoms on subjective anger through aggressive psycho-motor scripts was marginal in these data. It is possible that the lower trauma symptom load of the subclinical sample resulted in attenuation of observed effects relative to those expected among help-seeking survivors. Nevertheless, these data represent the first demonstration that this element of Chemtob et al.'s (1997) model may generalize to individuals exposed to non-combat trauma. This suggests the links between post-trauma symptoms and aggressive psycho-motor scripts observed in veteran samples are not solely attributable to military training. Results are consistent with modern theories of emotion that identify action tendencies (e.g., aggressive inclinations) as an important element of the subjective experience of emotion (e.g., anger; Carver & Harmon-Jones, 2009; Frijda, Kuipers, & ter Schure, 1989; Roseman, Weist, & Swartz, 1994). Men in the present sample were also found to demonstrate more frequent thoughts of engaging in aggression than women, controlling for PTSD symptom severity. Effects suggest differences in socialized gender role expectations may account for increased activation of aggressive scripts among men (Jakupcak, Lisak, & Roemer, 2002).

Contrary to hypotheses, results did not provide evidence of an indirect relation of PTSD on state anger through indices of physiological reactivity. It is possible the brief rumination task utilized for this study, combined with the evaluation of a non-clinical sample, failed to generate an adequate physiological response to the chosen anger memories. Alternatively, it is possible that sensitivity to physical arousal may be more relevant to potentiating subjective anger than absolute change in activation. Novaco and Chemtob (2002) found that veterans with elevated trauma symptoms scored higher on self-reported physiological reactivity during anger episodes as compared to veterans demonstrating less distress. Data supporting perceived change in arousal as an intervening variable in the relation between PTSD and anger would be consistent with research demonstrating robust associations between anxiety sensitivity and post-trauma symptoms (e.g., Marshall, Miles, & Stewart, 2010; Raines et al., 2017).

Results also failed to demonstrate an effect of PTSD severity on anger recovery following the provocation task. Though these analyses were exploratory, Chemtob et al. (1997) propose that individuals experiencing active trauma symptoms are likely to experience broad deficits in emotion regulation, resulting in greater persistence of state anger. As before, it is possible the induction procedure and the moderate sample available for these analyses was of insufficient strength to detect moderating effects of post-trauma symptoms on anger recovery. The strength of the relation between trauma symptoms and subjective anger may also vary between survivors. Research by Novaco and Chemtob (2015) noted that, among Vietnam-era veterans meeting criteria for PTSD, elevations in subjective anger and irritability were not exhibited by all individuals. This suggests that other variables, such as emotion regulation strategies or impulsivity, may serve to moderate the strength of the relation between PTSD symptoms and subjective anger in response to provocation (Novaco & Chemtob, 2015).

Interpretation of results should be made within the context of the study's relative strengths and limitations. The current project is the first to provide a comprehensive evaluation of Chemtob et al.'s (1997)

model in a sample of civilian trauma survivors. Analyses also capitalized on a prospective design, targeting predictive associations of PTSD symptom severity on state anger as well as hostile attribution bias, physiological reactivity, and aggressive psycho-motor scripts. Finally, evaluation of effects within a laboratory setting allowed for some standardization of the anger provocation and enabled investigators to monitor physiological activation in real-time.

Nevertheless, the prospective design may have contributed to the failure to detect a direct relation between posttrauma symptoms and state-level anger. Research exploring the association between PTSD and anger has focused primarily on concurrent associations between active symptoms and emotional responding (e.g., Beckham et al., 2002; Jakupcak et al., 2007; Novaco & Chemtob, 2002). Studies that have utilized prospective designs have not examined reactions to lab-based manipulations (e.g., Meffert et al., 2008; Orth et al., 2008). Although the presence of a base relation between PTSD and state anger would strengthen interpretations, current guidelines for the evaluation of intervening variable models support the assessment of indirect effects when a primary association is well-established or in situations where direct effects may be obscured due to temporal distance (Hayes, 2012; Shrout & Bolger, 2002). Continued research utilizing more intensive longitudinal designs (e.g., Van Voorhees et al., 2018; Possemato et al., 2015) may provide a more sensitive test of the relation between posttrauma symptoms and state anger in response to routine life events.

Regression models also suggest that trauma symptoms and intervening variables identified by Chemtob et al. (1997) account for a relatively small proportion of variability in subjective anger as elicited in this study. Although results offer continued support for proposed effects, other factors are likely involved in the relation between PTSD and anger processes. In addition, the use of trait measures of anger processes, rather than continuous measurement of individual emotional response (i.e., a daily diary design), may have limited the ability to capture individual variability in subjective anger. Future research should work to account other variables, such as impulsivity and emotion regulation, which could hold relevance to the relation between PTSD and anger.

As noted previously, it is possible the provocation task utilized in this study was limited in its ability to induce anger at an intensity necessary to capture indirect effects attributable to physiological activation. The moderate sample size may also have impacted the power of these analyses to detect indirect effects of physiological activation. Further, the extent to which the induction procedure elicited physiological reactivity outlined in the Chemtob et al. (1997) model is unclear. Designs utilizing confederates to generate ambiguously hostile situations could provide a more powerful activation of anger processes, particularly with respect to physiological arousal (Denson, Pedersen, Friese, Hahm, & Roberts, 2011; Vasquez et al., 2013). Whereas the evaluation of survivors reporting ongoing trauma symptoms precluded the use of more elaborate means of provocation, confederate-based designs could increase the ecological validity of analyses targeting anger processes.

Finally, effects observed in the current project may be attenuated by the assessment of a non-clinical sample. Index events identified by the majority of participants were unambiguously associated with Criterion A trauma. However, the utilization of self-report instruments restricted analyses to examining relations with probable PTSD symptoms. Symptom levels in the current sample were also less severe than those expected in help-seeking populations. Continued research incorporating interview-based assessments of PTSD among help-seeking survivors of civilian trauma would provide a stronger test of intervening variables in the relation of PTSD and subjective anger.

5. Conclusion

Examination of Chemtob et al.'s (1997) model of PTSD and anger in survivors of non-combat trauma suggests the underlying structure of the relation between these constructs may be similar in veteran and civilian trauma survivors. Results support the continued development and utilization of cognitive restructuring interventions, such as cognitive processing therapy (CPT, Resick & Schnicke, 1992), aimed at challenging hostile attribution biases in survivors with PTSD. Effects noted in this study also suggest that behavioral techniques including exposure and skills training may help to extinguishing aggressive inclinations while reinforcing alternative scripts for adaptive social interaction.

Conflicts of interest

The authors have no conflict of interest to declare.

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