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# Anger and Posttraumatic Stress Disorder Symptoms in Crime Victims: A Longitudinal Analysis

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Abstract

Among trauma-exposed individuals, severity of posttraumatic stress disorder (PTSD) symptoms is strongly correlated with anger. We used 2 longitudinal data sets with 282 and 218 crime victims, respectively, to investigate the temporal sequence of anger and PTSD symptoms following the assault. Cross-lagged regression analyses indicated that PTSD symptoms predicted subsequent level of anger, but that anger did not predict subsequent PTSD symptoms. Testing alternative models (common factor model, unmeasured third variable model) that might account for spuriousness of the relation strengthened confidence in the results of the cross-lagged analyses. Further analyses suggested that rumination mediates the effect of PTSD symptoms on anger.

*Key Words*: posttraumatic stress disorder, anger, rumination, crime victims

# Anger and PTSD Symptoms in Crime Victims: A Longitudinal Analysis

Besides fear, helplessness, and horror, traumatic events have the potential to cause strong feelings of anger. Indeed, the definition of posttraumatic stress disorder (PTSD) given in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev., American Psychiatric Association, 2000) lists irritability and outbursts of anger as one of the arousal symptoms. Moreover, Novaco and Chemtob (1998) pointed out in their review on anger and PTSD that early observations identified anger as a prevalent component of posttraumatic stress reactions.

For over a decade now, an increasing number of studies have shown that anger is significantly increased following traumatic events and that level of anger is strongly correlated with severity of PTSD (e.g., Chemtob, Hamada, Roitblat, & Muraoka, 1994; Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992; Schützwohl & Maercker, 2000). Of note, Novaco and Chemtob (2002) looked at whether the correlation between anger and PTSD symptoms decreased if items measuring anger and irritability within PTSD scales were removed. They found that the correlation was virtually as strong as it was with the full PTSD scales, indicating that the correlation was not a methodological artifact. A recent meta-analysis revealed that anger and severity of PTSD are strongly correlated with a mean effect size of $r = .48$, that the correlation is significantly higher with increasing time since the event, and that the correlation is highest in samples with military war experience with $r = .56$ (Orth & Wieland, 2006). However, in samples that experienced other traumatic events (e.g., criminal victimization, technological disaster, and health trauma) the correlation is strong as well, ranging from .30 to .48.

The question, then, is whether anger increases as a function of PTSD symptoms, whether PTSD symptoms increase as a function of anger, whether anger and PTSD symptoms reciprocally predict each other, or whether the relation is spurious because of third variables that

affect both anger and PTSD symptoms. Available evidence from longitudinal and treatment studies, which we review below, does not allow firm conclusions on the temporal sequence of anger and PTSD symptoms. Therefore, the aim of the present research was to investigate the temporal sequence of anger and PTSD symptoms following a traumatic event.

So far, only a few studies on anger and PTSD have employed longitudinal designs. In some of the studies, anger predicted PTSD severity at subsequent assessments (Ehlers, Mayou, & Bryant, 1998; Feeny, Zoellner, & Foa, 2000; Riggs et al., 1992); however, the effect size was small in Ehlers et al.'s (1998) study and PTSD severity at the prior assessment was not controlled for in Riggs et al.'s (1992) study. In other studies anger did not significantly predict subsequent PTSD severity (Andrews, Brewin, Rose, & Kirk, 2000; Zoellner, Foa, & Brigidi, 1999). Importantly, none of the studies tested the reverse direction, i.e., whether PTSD severity predicted anger at subsequent assessments.

Further evidence on the relation between PTSD symptoms and anger can be derived from treatment studies. Whereas Foa, Riggs, Masie, and Yarczower (1995) reported that anger was not significantly reduced by treatment of PTSD, Cahill, Rauch, Hembree, and Foa (2003) found that three different treatments of PTSD resulted in a significant reduction in anger. Chemtob, Novaco, Hamada, and Gross (1997) evaluated treatment targeting anger in Vietnam veterans with PTSD. Compared to a routine care condition, anger treatment resulted in a greater reduction in frequency of reexperiencing symptoms. Finally, studies investigating the moderating effect of pretreatment anger on PTSD treatment efficacy reported null results (Cahill et al., 2003; Pitman et al., 1996) or only tentative results (Foa et al., 1995; Taylor et al., 2001). To summarize, the results of treatment studies are inconsistent and do not allow clear conclusions regarding the temporal sequence of PTSD symptoms and anger.

Which psychological processes might account for the relation between PTSD symptoms and anger? Chemtob, Novaco, Hamada, Gross, and Smith (1997) hypothesized that individuals suffering from PTSD have a significantly lowered threshold for perceiving situations as threatening and that the perception of threat activates a biologically predisposed survival mode that includes anger reactions. Riggs et al. (1992) hypothesized that individuals with PTSD are motivated to avoid feelings of fear and that anger serves as a welcome distractor from fear-eliciting traumatic memories. Another possible explanation comes from cognitive models of PTSD suggesting that PTSD severity is correlated with rumination about the traumatic event and its consequences; for example, traumatized individuals ruminate about how the event could have been prevented or how life has been changed by it (cf. Ehlers et al., 1998; El Leithy, Brown, & Robbins, 2006). Rumination strengthens the associations between simultaneously activated cognitive and emotional elements of a memory structure. Thus, rumination can increase depressive affect (cf. Nolen-Hoeksema & Morrow, 1991) but also angry affect (Rusting & Nolen-Hoeksema, 1998). Given that the trauma-related associative network is likely linked to both PTSD symptoms (such as intrusive memories) and anger-eliciting memories, frequent trauma-related rumination could strengthen the association between PTSD symptoms and anger.

In the present research, we analyzed two longitudinal data sets consisting of crime victims. In Study 1, the analyses were based on four repeated assessments in the first three months after the assault, in Study 2, on two assessments at about five and seven months. The statistical analyses were conducted using cross-lagged regression analyses based on structural equation modeling (cf. Finkel, 1995).

## Study 1

In Study 1, we analyzed a data set that has been used by previous studies on anger and PTSD (Feeny et al., 2000; Zoellner et al., 1999). We decided to reanalyze the data set, as the analysis promised to extend previous studies and advance knowledge on the relation between anger and PTSD in several ways. First, we investigated reciprocal effects between the variables, whereas previous studies had only tested whether anger predicts PTSD symptoms. Second, we tested models based on multiple repeated assessments, increasing the reliability of the estimates. Third, we tested models that took into account the autoregression of constructs (done by Feeny et al., 2000, but not by Zoellner et al., 1999). Fourth, we took advantage of the full sample by using a maximum-likelihood procedure to deal with missing data, providing less biased and more reliable results than conventional missing data methods such as listwise or pairwise deletion (cf. Schafer & Graham, 2002). Fifth, structural equation modeling allowed us to control for random measurement error (by analyzing the constructs as latent variables) and nonrandom measurement error (by accounting for variance related to specific indicators and occasions).

## *Method*

### *Participants*

The data were collected as part of an extensive longitudinal study conducted in the Northeast of the United States. The sample consisted of 282 women who were victims of nonsexual assault (52%) or sexual assault (48%). In 75% of the cases, the perpetrator was a stranger; in 25% the perpetrator was known to the victim. Mean age of participants was 30.8 years (*SD* = 10.0, Range 17 to 65). Sixty-eight percent were African Americans, 29% were Caucasians, and 3% were of other ethnicity. Forty-six percent had attended or completed high school, 50% had attended or completed college, and 4% had attended or completed graduate school. The participants were largely untreated.

Participants were recruited through newspaper advertisements and police and hospital referrals. The assessments lasted about 2 hours and were conducted by trained interviewers who had at least a BA/BS in psychology. Participants were reimbursed $35 for each assessment. Assessments took place at *M* = 9.8 days (*SD* = 4.2) for Time 1, at *M* = 33.0 days (*SD* = 6.1) for Time 2, at *M* = 61.5 days (*SD* = 7.5) for Time 3, and at *M* = 91.2 days (*SD* = 10.4) for Time 4. Thus, mean time intervals between the assessments were 23.2 days, 28.5 days, and 29.7 days. We judged the time intervals to be similar enough to treat them as equal in the analyses. Data were available for 277 individuals at Time 1, 228 individuals at Time 2, 196 individuals at Time 3, and 193 individuals at Time 4. To investigate the potential impact of attrition we reran the SEM analyses without participants who dropped out of the study before Time 4. However, the results of the analyses (loading coefficients, regression coefficients, and fit indices) were virtually unaltered.

*Measures*

*PTSD Symptoms.* PTSD symptoms were assessed with the PTSD Symptom Scale-Interview, PSS-I (Foa, Riggs, Dancu, & Rothbaum, 1993).The PSS-I consists of 17 questions that correspond to the DSM-IV-TR PTSD symptoms. Symptoms were assessed with respect to the preceding two weeks or, if shorter, the time since the assault. Answers of participants were rated on a 4-point scale (0 = *not at all*, 1 = *once per week or less/a little*, 2 = *two to four times per week/somewhat*, 3 = *five or more times per week/very much*). Scale scores were computed as the mean of the underlying items. Foa et al. (1993) report a test-retest reliability of .80, an interrater reliability of .97., a sensitivity of .88 and specificity of .96 for the diagnosis of PTSD. Internal consistency in this study was high and ranged from .84 to .91 for Time 1 to Time 4.

*Anger.* Anger was assessed by self-report with the state scale of the State-Trait Anger Expression Inventory, STAXI (Spielberger, 1988), a measure which is widely used in clinical and nonclinical research (cf. Eckhardt, Norlander, & Deffenbacher, 2004). Evidence of its reliability and validity has been reported (cf. Spielberger, 1988). In this study, we decided to use the state scale but not the trait scale, because the state scale is more suitable for measurement of change. The state scale consists of 10 items. Participants assessed how they feel "right now". Answers were measured on a 4-point scale (1 = *not at all*, 2 = *somewhat*, 3 = *moderately so*, 4 = *very much so*). Scale scores were computed as the mean of the underlying items. In this study, internal consistency ranged from .93 to .97 for Time 1 to Time 4.

*Procedure for the Statistical Analysis*

For the computations we used Amos 5 (Arbuckle, 2003; Arbuckle & Wothke, 1999). We used fixation of factor loadings as the scaling method; for each factor, the unstandardized value of the first loading was set to 1. To deal with missing values, we employed the full information maximum likelihood (FIML) procedure included in Amos. Model fit was assessed by three fit indices that are currently recommended as most useful (Hu & Bentler, 1998, 1999): the Tucker-Lewis-Index (TLI), the Comparative Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA). Hu and Bentler (1999) suggest that good fit is indicated by values greater than or equal to .95 for TLI and CFI, and less than or equal to .06 for RMSEA. In addition, we report $\chi^2$-statistics and the confidence interval for RMSEA.

## *Results and Discussion*

*Preliminary Analyses*

The PTSD sample rate was 57% for Time 2, 42% for Time 3, and 36% for Time 4, following the guidelines given in Foa et al. (1993). For Time 1, we did not compute the PTSD

sample rate, because the diagnosis of PTSD cannot be given within the first month after the traumatic event (American Psychiatric Association, 2000).

We investigated whether the association between anger and PTSD symptoms is artificially inflated by the fact that the PTSD symptom measure used, the PSS-I, includes an item measuring an anger-related construct ("irritability," cf. Foa et al., 1993). The results showed that the cross-sectional correlations decreased only slightly when the item was omitted (from .38 to .36 for Time 1, from .40 to .37 for Time 2, from .51 to .49 for Time 3, and remaining at .41 for Time 4). Thus, the results of this study replicate the findings of Novaco and Chemtob (2002), indicating that the correlation between severity of PTSD and anger is not a methodological artifact.

*Measurement Models*

Table 1 shows means and standard deviations of the measures used in Study 1. For the structural equation models, we used subscales (PSS-I) and item parcels (STAXI), respectively, as indicators because they produce more reliable latent variables than individual items (Little, Cunningham, Shahar, & Widaman, 2002). For anger, we randomly aggregated the items into three parcels. The correlations of indicators are reported in Appendix 1.

First, we compared the fit of two measurement models. In the first measurement model, we freely estimated the factor loadings for eight latent variables measuring PTSD symptoms and anger at Time 1 to Time 4 (Model 1); all factors were correlated with each other and the uniquenesses of individual indicators were correlated over time to account for random measurement error. The fit of the first measurement model was good (see Table 2). The second measurement model was identical to the first except that we constrained the factor loadings of each indicator to be equal across time (Model 2). If the constrained model does not fit worse than

the unconstrained model, then the constraints are empirically justified and ensure that the latent constructs are measured similarly across time (i.e., factorial invariance).

To test for differences in model fit, we followed the recommendation of MacCallum, Browne, and Cai (2006) and used the test of small differences in fit instead of the more commonly used $\chi^2$-difference test. With sufficiently large samples, the $\chi^2$-difference test for nested models will always be significant, even when the true difference in fit is very small and theoretically irrelevant (cf. MacCallum et al., 2006). In contrast, the test of small difference in fit tests for differences greater than an a priori specified small difference, and thus a non-significant difference implies that the true difference is small, assuming that the sample size provides adequate statistical power. In conducting the test, we used the exact specifications given by MacCallum et al. (2006, cf. Program C): $\alpha = .05$, $RMSEA_A = .06$, and $RMSEA_B = .05$ ($RMSEA_A$ and $RMSEA_B$ represent the a priori specified small difference in fit). For all tests of small difference in fit reported in this article, statistical power was sufficiently large with values between .92 and 1.00.

The test of small difference in fit was nonsignificant for Models 1 and 2, indicating that they did not differ meaningfully from each other. Consequently, we favored the more parsimonious Model 2, and retained the longitudinal constraints on factor loadings in the subsequent analyses.

*Cross-Lagged Models*

Next, we tested the fit of two cross-lagged models, using the measurement model specified by Model 2. In cross-lagged models, a latent variable at Time 2 is predicted by the same variable at Time 1 and the other latent variable at Time 1 (cf. Figure 1). The cross-lagged paths indicate the effect of one variable on the other after controlling for the stability of the

variables over time. We accounted for nonrandom measurement error due to specific measurement occasions by cross-sectionally correlating the disturbances of the corresponding factors (cf. Cole & Maxwell, 2003).

In the first cross-lagged model (Model 3), all structural coefficients were freely estimated. Model fit was good (Table 2). In the second cross-lagged model (Model 4), we constrained the structural parameters (stability coefficients and cross-lagged coefficients) to be equal across all three time intervals. The difference in fit between Model 3 and 4 was nonsignificant. Consequently, we favored the more parsimonious Model 4, and retained the longitudinal constraints on structural coefficients in the subsequent analyses.

The structural coefficients for Model 4 are presented in Figure 1 with standardized values. The coefficients of the cross-lagged paths from PTSD symptoms to anger were significant (all *p*s < .01) and ranged from .14 to .20. In contrast, the coefficients of the cross-lagged paths from anger to PTSD symptoms were nonsignificant and ranged from -.04 to -.07.

The size of the cross-lagged effects can be assessed using the $f^2$ statistic for regression coefficients, which is defined as incremental variance of the outcome explained by the predictor ($\Delta R^2$) divided by the unexplained variance of the outcome ($1 - R^2$), as given by Cohen (1988). Values of .02 are interpreted as small effects, .15 as medium effects, and .35 as large effects (Cohen, 1988). We determined the incremental variance explained by the predictor by estimating two models, i.e., a model with the effect and a model without the effect (set to zero), and computed the difference in explained variance. The cross-lagged effects of PTSD symptoms on anger correspond to $f^2$ values of .06, .08, and .11, for the three time intervals, respectively, indicating small to medium effects. The cross-lagged effects of anger on PTSD symptoms correspond to $f^2$ values of .01, .00, and .00, for the three time intervals, respectively.

Furthermore, the results showed that the stability coefficients of PTSD symptoms were high, with values ranging from .83 to .88 (all *p*s < .01), and that the stability coefficients of anger were at a medium level, with values ranging from .41 to .51 (all *p*s < .01). The lower stability of anger compared to PTSD symptoms does not mean that the anger measure was less reliable. The findings reported in the method section indicate that both scales are reliable measures of the corresponding constructs. Moreover, if the low stability of anger across time indicated low reliability, the chances of explaining this variable by using other variables would be low. However, the analyses show that anger is significantly explained by PTSD symptoms measured at the preceding assessment.

*Common Factor Models*

Then, to investigate the validity of the cross-lagged model, we analyzed alternative structural models controlling for the potential spuriousness of the cross-lagged effects, as given by Finkel (1995). Figure 2 illustrates the alternative models tested. First, we analyzed a common factor model (Models 5 to 7, Figure 2A). In contrast to the cross-lagged model, the stability and cross-lagged paths between the PTSD and anger factors are set to zero, and four additional autoregressive common factors are included for Time 1 to Time 4, explaining the corresponding PTSD and anger factors. With Models 5 and 6 we tested whether the loadings of the common factors could be set equal across time (coefficients a1 to a4, Figure 2A; the loadings of PTSD symptoms had to be constrained to a fixed value, i.e., 1). The difference in fit was nonsignificant. Consequently, we favored the more parsimonious Model 6, and constrained the loadings of the common factors across time. Then, with Models 6 and 7, we tested whether the autoregressive paths between the common factors (coefficients b1 to b3, Figure 2A) could be set equal across time. Again, the difference in fit was nonsignificant. Consequently, we favored the more

parsimonious Model 7. However, the fit of Model 7 was lower compared to the cross-lagged model (Model 4). Because the common factor model and the cross-lagged model are non-nested, no formal test of difference in fit is possible; however, the fit values clearly indicate the favorability of the cross-lagged model. Thus, the rejection of the common factor model strengthens confidence in the results of the cross-lagged model.

*Unmeasured Third Variable Model*

Finally, we analyzed an unmeasured third variable model (Model 8, Figure 2B). In contrast to the cross-lagged model, this model included one additional latent variable, interpreted as an unmeasured third variable that explains all eight construct factors of PTSD symptoms and anger at Time 1 to Time 4. The stability effects and cross-lagged effects (coefficients c, d, e, and f, Figure 2B) had to be set equal across time to identify the model. As indicated by the fit values, the fit of Model 8 was slightly better than the fit of the cross-lagged model (Model 4), even if the difference in fit was nonsignificant. However, the structural coefficients (i.e., cross-lagged and stability coefficients) for Model 8 very closely matched the coefficients of the cross-lagged model. Thus, the unmeasured third variable model was a well-fitting alternative model of the data, but the coefficients that are crucial for the interpretation of the temporal sequence of PTSD symptoms and anger provided identical conclusions to those given by the cross-lagged model.

The results of Study 1 suggest that PTSD symptoms predict anger in the first months after a traumatic event, but that anger does not predict PTSD symptoms, and that the effect of PTSD symptoms on anger is not spurious. However, there is a need for cross-validating the findings. Therefore, we conducted a second longitudinal study.

## Study 2

Study 2 differed from Study 1 in terms of sample characteristics (mixed gender sample vs. the female sample in Study 1, German sample vs. the U.S. sample in Study 1) and frequency and timing of measurement (two assessments at about five and seven months postassault vs. four assessments in the first three months postassault in Study 1). A limiting feature of Study 1 consisted in the time frame of the anger measure (emotional state), which did not correspond to the measure of PTSD severity (symptoms in the preceding two weeks). Therefore, in Study 2 we used measures of PTSD symptoms and anger based on an identical time frame.

Besides replicating the analyses of Study 1, Study 2 extends the findings by testing rumination as a potential mediator of the relation between PTSD symptoms and anger. A mediation analysis in a two-wave design requires testing the significance of two longitudinal paths (Cole & Maxwell, 2003): the path from the predictor at Time 1 to the mediator at Time 2 controlling for the mediator at Time 1, and the path from the mediator at Time 1 to the outcome at Time 2 controlling for the outcome at Time 1. If stationarity (i.e., unchanging stability and cross-lagged coefficients across time) can be assumed, the product of the two paths provides an estimate of the mediational effect, even if the paths are estimated for the same time interval. A shortcoming of the two-wave design is that the assumption of stationarity cannot be tested (cf. Cole & Maxwell, 2003). However, the results of Study 1 provided evidence that the stationarity assumption is justified: the cross-lagged model with longitudinally constrained structural coefficients had virtually the same fit as the unconstrained model. Given that mediation analyses are frequently conducted with cross-sectional data, Cole and Maxwell (2003) judge the shortcomings of two-wave designs (potential violations of stationarity) to be less important than its advantages (controlling for prior level of the outcome).

## *Method*

*Participants*

The sample consisted of 218 individuals (67% female) who were victims of nonsexual assault (76%) or sexual assault (24%). In 44% of the cases, the perpetrator was a stranger; in 56% the perpetrator was known to the victim. Mean age of participants was 39.2 years (*SD* = 16.1, range 18 to 95 years). Forty-seven percent completed the obligatory 9 school years or less; 53% completed high school (10 years) or academic-track high school (ca. 13 years; university degrees were not recorded). At Time 1, 41% of the participants were making use of psychological counseling or therapy; at Time 2, the rate was 26%.

Participants were contacted with the help of the German victim assistance organization *Weisser Ring*. Inclusion criteria were a victim age of 18 years or older and German nationality. The assessments were originally intended to take place at four and six months following the criminal victimization. For practical reasons, the timing of the first assessment had to be relaxed to three to seven months. Thus, at Time 1, mean time since the assault was 5.2 months (*SD* = 1.4). For all participants, the second assessment was conducted two months after Time 1 (*M* = 7.2, *SD* = 1.4). Participants were paid 25 Euros to recompense their time and effort.

For Time 1, victims were sent a questionnaire with a request that they take part in the study; the response rate was 38%. The Time 2 questionnaire was sent to Time 1 responders, of which 81% responded. To investigate the potential impact of attrition, we reran the SEM analyses without participants who dropped out of the study (*N* = 177 vs. *N* = 218). However, the results of the analyses (loading coefficients, regression coefficients, and fit indices) were virtually unaltered.

*Measures*

*PTSD symptoms.* PTSD symptoms were assessed by self-report with the Impact of Event Scale-Revised, IES-R (Weiss & Marmar, 1997, for the German version see Maercker & Schützwohl, 1998). The IES-R consists of 22 items assigned to three symptom cluster subscales (intrusion with seven items, avoidance with eight items, and hyperarousal with seven items). To conform with the labels of the PSS-I used in Study 1, the remainder of this article refers to intrusion as reexperiencing and hyperarousal as arousal. PTSD symptoms were assessed with respect to the preceding seven days. In the German version, answers are measured on a 4-point scale using a non-equidistant scoring scheme (0 = *not at all*, 1 = *seldom*, 3 = *sometimes*, 5 = *often*). Scale scores were computed as the mean of the underlying items. Internal consistencies were high with .86 and .90 for reexperiencing (Time 1 and Time 2, respectively), .73 and .78 for avoidance, and .84 and .89 for arousal. In the German IES-R validation study, a regression equation was determined that can be used to estimate the PTSD sample rate, with a sensitivity of .70 to .76 and a specificity of .88 to .89 (Maercker & Schützwohl, 1998).

*Anger.* To employ an anger measure with a time frame identical to the IES-R (i.e., frequency in the preceding seven days), we adapted the state anger scale of the STAXI (Spielberger, 1988, for the German version see Schwenkmezger, Hodapp, & Spielberger, 1992). Data were collected by self-report. The tense of the items had to be changed from present to past. Due to restrictions of questionnaire length, we used only five out of ten items selected for the highest item-total correlations. Answers were measured on a 6-point scale ranging from 0 (*never*) to 5 (*very often*). Scale scores were computed as the mean of the underlying items. Internal consistencies were high with .88 and .91 (Time 1 and Time 2, respectively).

*Rumination.* Rumination was assessed with a scale consisting of three items measuring ruminative thoughts identified in the PTSD literature (cf. Ehlers et al., 1998). The items were “I

thought about how my life would have been if the event had not happened," "I thought about how the event could have been prevented," and "I thought about why the event had to happen to me." Participants were instructed to assess the frequency of rumination with respect to the preceding seven days (to parallel the time frame of the other measures used in this study). Answers were measured on a 6-point scale ranging from 0 (*never*) to 5 (*very often*). Scale scores were computed as the mean of the underlying items. Internal consistency was .78 and .82 (Time 1 and Time 2, respectively).

*Procedure for the Statistical Analysis*

The analyses were conducted using Amos 5 and full information maximum likelihood (FIML). Model fit was assessed using the same fit indices as in Study 1.

## *Results and Discussion*

*Preliminary Analyses*

The PTSD sample rate was 52% for Time 1 and 49% for Time 2. As in Study 1, we investigated whether the association between anger and PTSD symptoms was inflated by the fact that the PTSD symptom measure used, the IES-R, includes an item measuring anger ("I felt irritable and angry," cf. Weiss & Marmar, 1997). The cross-sectional correlations decreased only slightly when the item was omitted (from .49 to .47 for Time 1 and from .54 to .52 for Time 2).

*Cross-Lagged Models*

Table 3 shows means and standard deviations of the measures used in Study 2. As in Study 1, we used three subscales (PTSD symptoms) and three item parcels (anger) as indicators of the latent variables. The correlations of indicators are reported in Appendix 2.

The measurement models were identical to those tested in Study 1. Both the freely estimated and the constrained model provided a good fit to the data (Table 4). The difference in

fit was nonsignificant, leading us to retain the longitudinal constraints on factor loadings in the subsequent analyses.

Next, we tested the structural model (Model 11). The fit of the structural model was good (Table 4). Because the structural model and the constrained measurement model had the same model-implied covariance matrix, fit indices of these models are identical. The structural coefficients for Model 11 are presented in Figure 3A with standardized values. The cross-lagged path from PTSD symptoms to anger was .22 ($p < .05$), whereas the cross-lagged path from anger to PTSD symptoms was .03 (*ns*). The effect size of the cross-lagged effects corresponded to $f^2$ values of .02 and .00, respectively, indicating a small effect of PTSD symptoms on anger, and a zero effect of anger on PTSD symptoms. The stability coefficient of PTSD symptoms was high with .83 ($p < .01$), and the stability coefficient of anger was medium with .44 ($p < .01$). To summarize, the results of the cross-lagged analyses in Study 2 replicate the findings in Study 1 very closely.

*Mediation Models*

Then, we investigated whether rumination mediates the effect of PTSD symptoms on anger. Again, we tested two measurement models, one with freely estimated loadings (Model 12) and one with longitudinally constrained loadings (Model 13). The latent variables PTSD symptoms and anger were measured identically to Models 9 to 11; rumination was measured using the three items as indicators. The fit of both measurement models was good (Table 4). The difference in fit was nonsignificant, leading us to retain the longitudinal constraints on factor loadings in subsequent analyses. We tested whether the association between PTSD symptoms and rumination was artificially inflated by the fact that the IES-R includes two items tapping into the rumination construct (“Other things kept making me think about it,” “I thought about it when

I did not mean to," cf. Weiss & Marmar, 1997). However, the correlation was virtually unaltered when the items were omitted for both Time 1 and Time 2.

Next, we tested the structural model (Model 14). The fit of the structural model was good (Table 4). The structural coefficients for Model 14 are presented in Figure 3B with standardized values. The results show that rumination mediated the cross-lagged effect of PTSD symptoms on anger. The cross-lagged paths from PTSD symptoms to rumination and from rumination to anger were .38 and .28 (all *p*s < .01). In contrast, the direct cross-lagged path from PTSD symptoms to anger was only .04 (*ns*). The effect of PTSD symptoms on rumination corresponded to an $f^2$ value of .06, indicating a small to medium effect; the effect of rumination on anger corresponded to an $f^2$ value of .04, indicating a small effect; and the effect of PTSD symptoms on anger corresponded to an $f^2$ value of .00, indicating a zero effect.

## General Discussion

We investigated the temporal sequence of anger and PTSD symptoms in crime victims, using two longitudinal data sets with four repeated assessments in the first three months postassault (Study 1) and two assessments at about five and seven months (Study 2), respectively. Cross-lagged regression analyses indicated that PTSD symptoms predicted subsequent anger, with prior level of anger controlled for. In contrast, anger did not predict subsequent PTSD symptoms, with prior PTSD symptoms controlled for. The effects of PTSD symptoms on anger were significant and indicated a small to medium effect size; the effects of anger on PTSD symptoms were nonsignificant and indicated a zero effect size. This result could not be anticipated by the findings from previous longitudinal studies on anger and PTSD, because these studies only tested whether anger predicts PTSD symptoms, but not whether PTSD symptoms predict anger. In addition, the results of the present research replicated findings by

Novaco and Chemtob (2002), suggesting that the correlation between anger and PTSD symptoms is not artificially inflated by the fact that the PTSD measures used items assessing anger.

The multiple assessments in Study 1 allowed us to compare the cross-lagged model with alternative models controlling for potential spuriousness of the effect of PTSD symptoms on anger, i.e., a common factor model and a model accounting for an unmeasured third variable. The common factor model was not a plausible model of the data, given that it fit the data worse than the cross-lagged model. The unmeasured third variable model fit the data very well, but the cross-lagged effect of PTSD symptoms on anger remained unaltered and significant. Thus, the unmeasured third variable model suggested that the effect of PTSD symptoms on anger was not spurious.

Further analyses in Study 2 suggested that the effect of PTSD symptoms on anger was mediated by rumination. When the mediator was taken into account, the effect of PTSD symptoms on anger became nonsignificant, whereas the effect of PTSD symptoms on the mediator and the effect of the mediator on anger were significant. Consistent with findings of Ehlers et al. (1998) and El Leithy et al. (2006), PTSD severity was significantly related to rumination. The results suggest that rumination might be a psychological mechanism through which PTSD increases anger after a traumatic event. However, the results should be replicated using a validated rumination measure (e.g., the Ruminative Responses Scale, see Nolen-Hoeksema & Morrow, 1991).

A limitation of the present research is that none of the data sets included assessments of anger before the traumatic event. When analyzing the relation between PTSD symptoms and anger, future studies might control for pretraumatic level of anger, which might influence the

course of PTSD, even if posttraumatic anger does not predict subsequent PTSD symptoms. Moreover, future studies should analyze PTSD symptoms and anger for a longer time after the event. In the present research, assessments were carried out within the first seven months following the event. It might be interesting to test whether the pattern of results holds even several years after a traumatic event.

Another limitation is that the samples of both studies consisted exclusively of crime victims. Therefore, it is unclear whether the findings generalize to individuals who experienced other types of traumatic events (e.g., natural disasters, combat experience). As the available data show, the correlation between PTSD and anger is higher in samples with combat veterans compared to samples with other types of traumatic events (Orth & Wieland, 2006). Even if some of the psychological processes that account for the effect of PTSD symptoms on anger are the same following all types of traumatic events, the difference in size of correlation suggests that additional psychological processes might be at work in combat veterans. Therefore, the analyses of the present research should be replicated with combat veterans in particular.

Importantly, the study designs do not allow for strong conclusions regarding the possible causality of the effect of PTSD symptoms on anger. Nevertheless, longitudinal analyses are useful because they can indicate whether the data are consistent with a causal model of the temporal relationships between variables.

A strength of the present research is the convergence of findings across Study 1 and Study 2, which helps alleviate some methodological concerns and strengthens confidence in the results. For example, in Study 1 the time frames of the PTSD and anger measures were different, and the sample did not include both genders; however, these limitations were resolved in Study 2. In Study 2, a limitation consisted in the PTSD symptoms measure, the IES-R, which does not

completely correspond to the diagnostic criteria of PTSD in the DSM-IV-TR and whose validity is restricted due to the use of self-reports. However, this limitation was not present in Study 1, where the PTSD symptoms measure, the PSS-I, corresponded to the DSM-IV-TR and was based on clinical interviews. Also, in Study 2 the ethnic diversity of the sample was limited, whereas in Study 1 the sample was ethnically diverse. An additional strength of the present research, compared with previous studies, is the use of more appropriate statistical models based on latent variable modeling.

The results of the present research provide a basis for the advancement of theories about the relation between anger and PTSD. Theories of anger and PTSD should try to explain the psychological processes by which PTSD symptoms contribute to an increased level of anger, but theories should not seek to explain how posttraumatic anger influences PTSD symptoms. The theoretical accounts that have been introduced in the literature are not at odds with a unidirectional effect of PTSD symptoms on anger; as reported above, Chemtob, Novaco, Hamada, Gross et al. (1997) hypothesized that heightened perception of threat among traumatized individuals is responsible for increased anger, and Riggs et al. (1992) hypothesized that traumatized individuals are motivated to avoid fear and therefore more frequently shift their attention on anger. The present research suggests that still another process, rumination, might be responsible for the effect of PTSD symptoms on anger. However, because we did not test other potential mediators (e.g., threshold to perceive threat, fear avoidance), the results of the present research do not refute the hypotheses suggested by Chemtob, Novaco, Hamada, Gross et al. (1997) and Riggs et al. (1992). Therefore, future studies should test a broader range of processes that might mediate effects of PTSD on anger, and should take into account that the operating processes might vary depending on the type of traumatic event experienced by the sample.

This research suggests that recovery from PTSD (whether it be from treatment or spontaneous remission) entails recovery from posttraumatic anger but that recovery from posttraumatic anger does not entail recovery from PTSD. Nevertheless, it should not be overlooked that anger might still be important to target in treatment, as the effect of PTSD on anger may not be present in every individual and is, on average, only of moderate size. Therefore, anger treatment in individuals with PTSD may still be needed to better improve subjective well-being and social functioning.

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Table 1

*Means and Standard Deviations of Measures (Study 1)*

| Variable | Time 1 | | Time 2 | | Time 3 | | Time 4 | |
|---|---|---|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| Reexperiencing (PSS-I) | 1.82 | 0.73 | 1.15 | 0.75 | 0.89 | 0.79 | 0.68 | 0.72 |
| Avoidance (PSS-I) | 1.35 | 0.69 | 0.94 | 0.62 | 0.78 | 0.65 | 0.70 | 0.71 |
| Arousal (PSS-I) | 1.99 | 0.73 | 1.46 | 0.76 | 1.16 | 0.80 | 0.97 | 0.77 |
| Anger | 2.02 | 0.94 | 1.42 | 0.62 | 1.33 | 0.61 | 1.22 | 0.54 |

*Note.* PSS-I = PTSD Symptom Scale-Interview.

Table 2

*Fit Indices of the Models Tested (Study 1)*

| Model | $\chi^2$ | *df* | TLI | CFI | RMSEA | 90%-CI of RMSEA |
|---|---|---|---|---|---|---|
| Measurement models | | | | | | |
| 1. Free loadings | 277.2** | 188 | .97 | .98 | .041 | .030 - .051 |
| 2. Constrained loadings | 334.1** | 200 | .96 | .97 | .049 | .039 - .058 |
| Cross-lagged models | | | | | | |
| 3. Free structural coefficients | 363.7** | 212 | .96 | .97 | .050 | .042 - .059 |
| 4. Constrained structural coefficients | 377.3** | 220 | .96 | .97 | .050 | .042 - .059 |
| Common factor models | | | | | | |
| 5. Free loadings | 477.3** | 217 | .93 | .95 | .065 | .057 - .073 |
| 6. Constrained loadings | 479.2** | 220 | .93 | .95 | .065 | .057 - .073 |
| 7. Full constraints | 485.8** | 222 | .93 | .95 | .065 | .057 - .073 |
| Unmeasured third variable model | | | | | | |
| 8. Constrained structural coefficients | 351.3** | 212 | .96 | .97 | .048 | .039 - .057 |

*Note.* TLI = Tucker-Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; CI = confidence interval.

**$p < .01$.

Table 3

*Means and Standard Deviations of Measures (Study 2)*

| Variable | Time 1 | | Time 2 | |
|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* |
| Reexperiencing (IES-R) | 3.06 | 1.30 | 2.81 | 1.39 |
| Avoidance (IES-R) | 2.62 | 1.12 | 2.55 | 1.19 |
| Arousal (IES-R) | 3.12 | 1.38 | 2.80 | 1.52 |
| Anger | 3.03 | 1.41 | 2.75 | 1.40 |
| Rumination | 3.35 | 1.56 | 3.24 | 1.57 |

*Note.* IES-R = Impact of Event Scale-Revised.

Table 4

*Fit Indices of the Models Tested (Study 2)*

| Model | $\chi^2$ | *df* | TLI | CFI | RMSEA | 90%-CI of RMSEA |
|---|---|---|---|---|---|---|
| Bivariate Analyses (PTSD Symptoms and Anger) | | | | | | |
| Measurement models | | | | | | |
| 9. Free loadings | 62.6* | 42 | .97 | .99 | .048 | .019 - .071 |
| 10. Constrained loadings | 64.6* | 46 | .98 | .99 | .043 | .011 - .066 |
| Structural model | | | | | | |
| 11. Cross-lagged model | 64.6* | 46 | .98 | .99 | .043 | .011 - .066 |
| Mediation Analyses (PTSD Symptoms, Rumination, and Anger) | | | | | | |
| Measurement models | | | | | | |
| 12. Free loadings | 165.0** | 111 | .96 | .98 | .047 | .031 - .062 |
| 13. Constrained loadings | 169.9** | 117 | .96 | .98 | .046 | .030 - .060 |
| Structural model | | | | | | |
| 14. Mediation model | 173.0** | 120 | .97 | .98 | .045 | .029 - .059 |

*Note.* TLI = Tucker-Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; CI = confidence interval; PTSD = posttraumatic stress disorder.

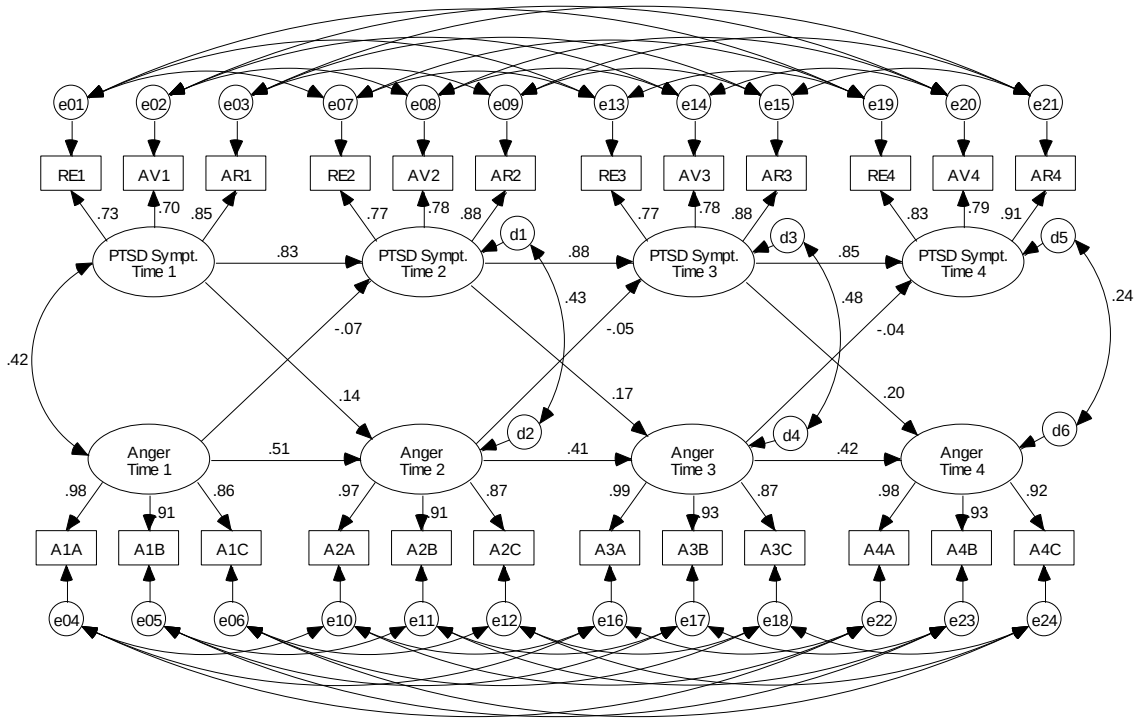
*$p < .05$. **$p < .01$.

## Figure Captions

*Figure 1.* Cross-lagged regression model of PTSD symptoms and anger with longitudinal constraints on structural coefficients (Model 4, Study 1). Values shown are standardized coefficients. To keep the figure simple, estimates of error variances and covariances are not shown. For all paths, *p*s < .01, except for paths from anger to PTSD symptoms (*ns*) and the correlation between the Time 4 disturbances of PTSD symptoms and anger ($p < .05$). PTSD = posttraumatic stress disorder; RE1 to RE4 = reexperiencing; AV1 to AV4 = avoidance; AR1 to AR4 = arousal. A1A to A4C = parcels measuring anger.

*Figure 2.* The figure illustrates alternative models accounting for potential spuriousness of effects between PTSD symptoms and anger, i.e., a common factor model (Figure 2A) and an unmeasured third variable model (Figure 2B). To keep the figure simple, only latent constructs are shown, and observed variables are omitted. The latent constructs were measured identically to the cross-lagged model (Figure 1). PTSD = posttraumatic stress disorder.

*Figure 3.* Cross-lagged regression model of PTSD symptoms and anger (Figure 3A, Model 11, Study 2) and mediation model with rumination mediating the effect of PTSD symptoms on anger (Figure 3B, Model 14, Study 2). To keep the figure simple, only latent constructs are shown, and observed variables are omitted. Values shown are standardized coefficients. In Figure 3A, for all paths, *p*s < .01, except for the path from anger to PTSD symptoms (*ns*) and the path from PTSD symptoms to anger ($p < .05$). In Figure 3B, for all paths, *p*s < .01, except for the path from PTSD symptoms to anger (*ns*) and the correlation between the Time 2 disturbances of rumination and anger ($p < .05$). PTSD = posttraumatic stress disorder.


e01
e02
e03
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e13
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e21
RE1
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AV4
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.70
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.77
.78
.88
.77
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.88
.83
.79
.91
PTSD Sympt. Time 1
PTSD Sympt. Time 2
PTSD Sympt. Time 3
PTSD Sympt. Time 4
d1
d3
d5
.83
.88
.85
.43
.48
.24
.42
-.07
-.05
-.04
.14
.17
.20
Anger Time 1
Anger Time 2
Anger Time 3
Anger Time 4
d2
d4
d6
.51
.41
.42
.98
.91
.86
.97
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.87
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.93
.87
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A1A
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A3C
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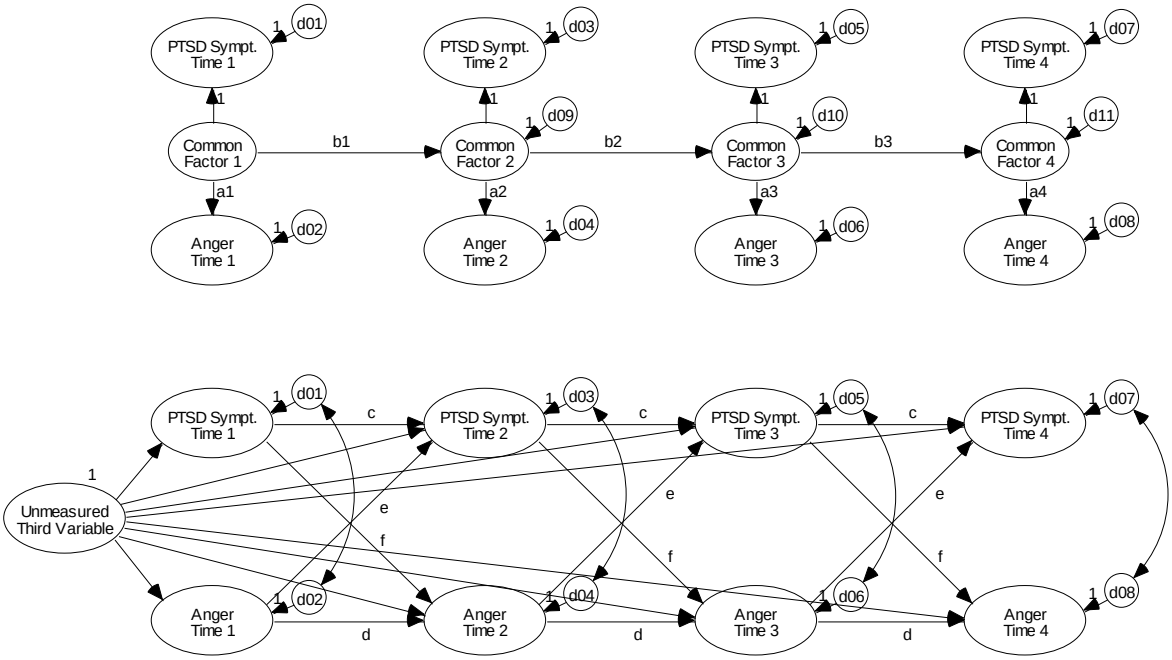
Figure 1


PTSD Sympt. Time 1
PTSD Sympt. Time 2
PTSD Sympt. Time 3
PTSD Sympt. Time 4
Common Factor 1
Common Factor 2
Common Factor 3
Common Factor 4
Anger Time 1
Anger Time 2
Anger Time 3
Anger Time 4
b1
b2
b3
a1
a2
a3
a4
d01
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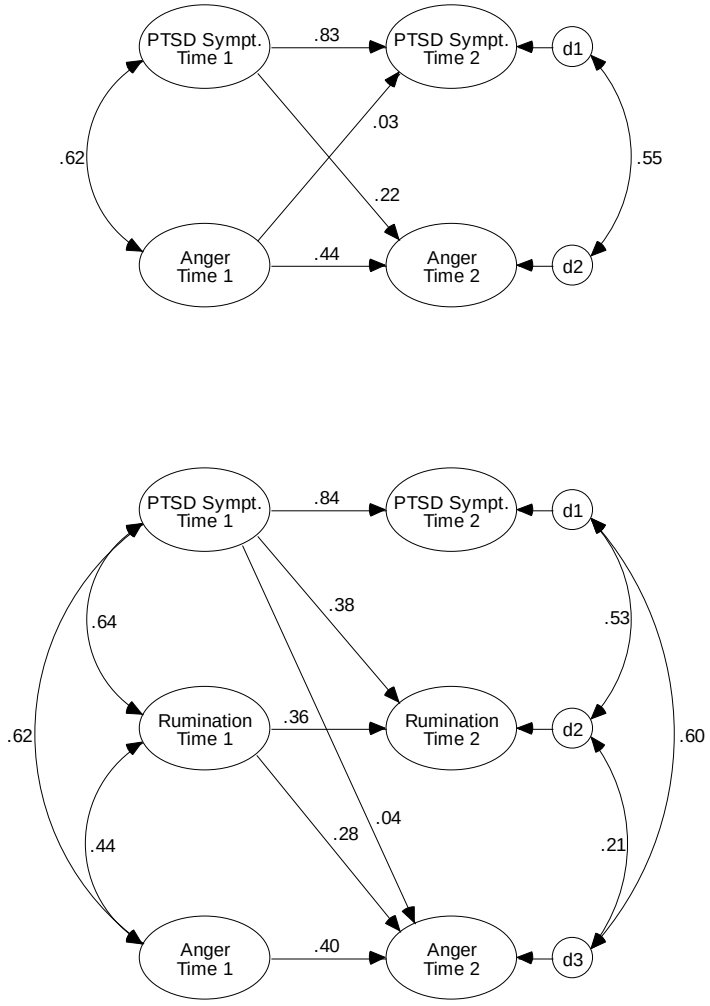
d10
d11
Unmeasured Third Variable
c
d
e
f

Figure 2


PTSD Sympt.
Time 1
.83
PTSD Sympt.
Time 2
d1
.03
.62
.55
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Anger
Time 1
.44
Anger
Time 2
d2
PTSD Sympt.
Time 1
.84
PTSD Sympt.
Time 2
d1
.38
.64
.53
Rumination
Time 1
.36
Rumination
Time 2
d2
.62
.60
.04
.28
.44
.21
Anger
Time 1
.40
Anger
Time 2
d3

Figure 3

Appendix 1

Means, Standard Deviations, and Correlations of Indicators of PTSD Symptoms and Anger (Study 1)

| Variable | M | SD | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. RE1 | 1.82 | 0.73 | -- | | | | | | | | | | | | | | | | | | | | | | | |
| 2. AV1 | 1.35 | 0.69 | .56 | -- | | | | | | | | | | | | | | | | | | | | | | |
| 3. AR1 | 1.99 | 0.73 | .63 | .60 | -- | | | | | | | | | | | | | | | | | | | | | |
| 4. RE2 | 1.15 | 0.75 | .59 | .52 | .51 | -- | | | | | | | | | | | | | | | | | | | | |
| 5. AV2 | 0.94 | 0.62 | .41 | .67 | .49 | .59 | -- | | | | | | | | | | | | | | | | | | | |
| 6. AR2 | 1.46 | 0.76 | .46 | .53 | .57 | .68 | .66 | -- | | | | | | | | | | | | | | | | | | |
| 7. RE3 | 0.89 | 0.79 | .50 | .43 | .40 | .72 | .51 | .55 | -- | | | | | | | | | | | | | | | | | |
| 8. AV3 | 0.78 | 0.65 | .42 | .63 | .43 | .59 | .73 | .53 | .62 | -- | | | | | | | | | | | | | | | | |
| 9. AR3 | 1.16 | 0.80 | .43 | .51 | .40 | .60 | .55 | .65 | .71 | .70 | -- | | | | | | | | | | | | | | | |
| 10. RE4 | 0.68 | 0.72 | .48 | .49 | .41 | .63 | .59 | .62 | .70 | .62 | .64 | -- | | | | | | | | | | | | | | |
| 11. AV4 | 0.70 | 0.71 | .43 | .55 | .39 | .49 | .67 | .50 | .46 | .75 | .59 | .69 | -- | | | | | | | | | | | | | |
| 12. AR4 | 0.97 | 0.77 | .42 | .43 | .38 | .49 | .55 | .62 | .52 | .54 | .69 | .72 | .74 | -- | | | | | | | | | | | | |
| 13. A1A | 1.90 | 0.95 | .23 | .28 | .37 | .20 | .11 | .26 | .15 | .11 | .12 | .09 | .11 | .12 | -- | | | | | | | | | | | |
| 14. A1B | 1.87 | 0.93 | .23 | .28 | .37 | .17 | .08 | .20 | .16 | .08 | .10 | .06 | .06 | .08 | .90 | -- | | | | | | | | | | |
| 15. A1C | 2.34 | 1.13 | .24 | .25 | .35 | .25 | .11 | .26 | .21 | .13 | .19 | .17 | .07 | .13 | .83 | .74 | -- | | | | | | | | | |
| 16. A2A | 1.35 | 0.60 | .17 | .23 | .24 | .31 | .24 | .41 | .23 | .24 | .28 | .25 | .23 | .24 | .45 | .38 | .33 | -- | | | | | | | | |
| 17. A2B | 1.39 | 0.60 | .16 | .24 | .26 | .34 | .24 | .43 | .24 | .22 | .27 | .19 | .16 | .19 | .37 | .37 | .26 | .86 | -- | | | | | | | |
| 18. A2C | 1.55 | 0.81 | .18 | .22 | .28 | .33 | .25 | .42 | .28 | .29 | .33 | .30 | .21 | .28 | .37 | .30 | .38 | .84 | .78 | -- | | | | | | |
| 19. A3A | 1.29 | 0.59 | .18 | .28 | .25 | .27 | .27 | .33 | .43 | .35 | .47 | .30 | .29 | .36 | .40 | .38 | .36 | .43 | .41 | .45 | -- | | | | | |
| 20. A3B | 1.27 | 0.53 | .21 | .26 | .25 | .30 | .25 | .34 | .48 | .34 | .47 | .30 | .27 | .35 | .35 | .37 | .28 | .37 | .41 | .40 | .91 | -- | | | | |
| 21. A3C | 1.45 | 0.80 | .20 | .28 | .28 | .32 | .29 | .36 | .48 | .40 | .51 | .35 | .34 | .40 | .36 | .33 | .39 | .44 | .40 | .55 | .90 | .83 | -- | | | |
| 22. A4A | 1.19 | 0.53 | .19 | .27 | .18 | .27 | .20 | .24 | .45 | .23 | .39 | .33 | .32 | .40 | .35 | .31 | .28 | .26 | .21 | .21 | .61 | .64 | .52 | -- | | |
| 23. A4B | 1.23 | 0.55 | .15 | .25 | .13 | .26 | .19 | .23 | .46 | .25 | .40 | .35 | .34 | .42 | .28 | .29 | .23 | .21 | .20 | .17 | .62 | .68 | .53 | .93 | -- | |
| 24. A4C | 1.25 | 0.60 | .18 | .24 | .16 | .26 | .16 | .20 | .45 | .22 | .40 | .35 | .33 | .42 | .31 | .27 | .32 | .22 | .17 | .24 | .61 | .63 | .57 | .90 | .88 | -- |

*Note.* PTSD = posttraumatic stress disorder; RE1 to RE4 = reexperiencing; AV1 to AV4 = avoidance; AR1 to AR4 = arousal. A1A to A4C = indicators of anger.

Appendix 2

Means, Standard Deviations, and Correlations of Indicators of PTSD Symptoms, Anger, and Rumination (Study 2)

| Variable | M | SD | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. RE1 | 3.06 | 1.30 | -- | | | | | | | | | | | | | | | | | |
| 2. AV1 | 2.62 | 1.12 | .42 | -- | | | | | | | | | | | | | | | | |
| 3. AR1 | 3.12 | 1.38 | .73 | .47 | -- | | | | | | | | | | | | | | | |
| 4. RE2 | 2.81 | 1.39 | .73 | .34 | .64 | -- | | | | | | | | | | | | | | |
| 5. AV2 | 2.55 | 1.19 | .39 | .71 | .38 | .36 | -- | | | | | | | | | | | | | |
| 6. AR2 | 2.80 | 1.52 | .67 | .33 | .71 | .80 | .40 | -- | | | | | | | | | | | | |
| 7. A1A | 3.11 | 1.54 | .51 | .18 | .53 | .47 | .18 | .40 | -- | | | | | | | | | | | |
| 8. A1B | 3.02 | 1.41 | .41 | .13 | .41 | .38 | .16 | .36 | .77 | -- | | | | | | | | | | |
| 9. A1C | 2.49 | 1.82 | .42 | .14 | .37 | .33 | .11 | .30 | .68 | .64 | -- | | | | | | | | | |
| 10. A2A | 2.81 | 1.49 | .40 | .13 | .41 | .53 | .21 | .56 | .48 | .46 | .36 | -- | | | | | | | | |
| 11. A2B | 2.83 | 1.42 | .37 | .20 | .33 | .49 | .25 | .49 | .39 | .51 | .23 | .81 | -- | | | | | | | |
| 12. A2C | 2.28 | 1.70 | .36 | .22 | .35 | .50 | .17 | .50 | .44 | .51 | .46 | .77 | .74 | -- | | | | | | |
| 13. R1A | 3.34 | 1.89 | .51 | .31 | .47 | .42 | .30 | .39 | .31 | .34 | .26 | .31 | .33 | .30 | -- | | | | | |
| 14. R1B | 3.30 | 1.81 | .36 | .26 | .40 | .25 | .28 | .27 | .30 | .16 | .17 | .26 | .20 | .16 | .43 | -- | | | | |
| 15. R1C | 3.41 | 1.91 | .42 | .30 | .41 | .36 | .30 | .35 | .32 | .34 | .24 | .37 | .37 | .37 | .59 | .62 | -- | | | |
| 16. R2A | 3.33 | 1.85 | .48 | .38 | .41 | .51 | .46 | .52 | .24 | .23 | .21 | .27 | .31 | .32 | .58 | .25 | .38 | -- | | |
| 17. R2B | 3.18 | 1.78 | .50 | .35 | .43 | .49 | .52 | .49 | .28 | .20 | .18 | .30 | .30 | .30 | .34 | .44 | .35 | .57 | -- | |
| 18. R2C | 3.21 | 1.87 | .42 | .37 | .36 | .47 | .45 | .45 | .22 | .25 | .16 | .33 | .33 | .36 | .42 | .31 | .58 | .59 | .65 | -- |

*Note.* PTSD = posttraumatic stress disorder; RE1 and RE2 = reexperiencing; AV1 and AV2 = avoidance; AR1 and AR2 = arousal. A1A to A2C = indicators of anger; R1A to R2C = indicators of rumination.