

Anger: Cause or Consequence of Posttraumatic Stress? A Prospective Study of Dutch Soldiers

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Many studies have shown that individuals with posttraumatic stress disorder (PTSD) experience more anger over time and across situations (i.e., trait anger) than trauma-exposed individuals without PTSD. There is a lack of prospective research, however, that considers anger levels before trauma exposure. The aim of this study was to prospectively assess the relationship between trait anger and PTSD symptoms, with several known risk factors, including baseline symptoms, neuroticism, and stressor severity in the model. Participants were 249 Dutch soldiers tested approximately 2 months before and approximately 2 months and 9 months after their deployment to Afghanistan. Trait anger and PTSD symptom severity were measured at all assessments. Structural equation modeling including cross-lagged effects showed that higher trait anger before deployment predicted higher PTSD symptoms 2 months after deployment ($\beta = .36$), with stressor severity and baseline symptoms in the model, but not with neuroticism in the model. Trait anger at 2 months postdeployment did not predict PTSD symptom severity at 9 months, and PTSD symptom severity 2 months postdeployment did not predict subsequent trait anger scores. Findings suggest that trait anger may be a pretrauma vulnerability factor for PTSD symptoms, but does not add variance beyond the effect of neuroticism.

There is a growing interest for the role of anger in anxiety disorders, especially posttraumatic stress disorder (PTSD; Olatunji, Ciesielski, & Tolin, 2010). Individuals with PTSD experience more angry feelings currently (state anger), and over time in response to a variety of situations (trait anger) when compared to non-PTSD trauma-exposed controls (Chemtob, Hamada, Roitblad, & Muraoka, 1994; Jakupcak et al., 2007; Lasko, Gurvits, Kuhne, Orr, & Pitman, 1994). The relationship between anger and PTSD is observed in male and female samples, and across types of traumatic events, with the largest effect size in military samples (Orth & Wieland, 2006). Because trait anger in particular is related to problematic behavior such as (intimate partner) violent behavior (Norlander &

Eckhardt, 2005), it seems clinically relevant to understand the role of trait anger in PTSD.

How can the association between trait anger and PTSD be explained? First, it may be a methodological artifact because experiencing irritability or outbursts of anger is a PTSD symptom (American Psychiatric Association [APA], 2000). Removal of this anger item from a PTSD instrument, however, does not substantially decrease the association, which excludes a methodological artifact (Novaco & Chemtob, 2002; Orth, Cahill, Foa, & Maercker, 2008). Second, trait anger may be a vulnerability factor for the onset of PTSD (i.e., a diathesis-stress model, which states that individual differences in personality traits may predispose one to a disorder that is triggered by a stressor; McKeever, & Huff, 2003), or the persistence of PTSD (i.e., a pathoplasty model, which posits that personality traits may influence the course or expression of symptoms; Clark, Watson, & Mineka, 1994). Third, elevated levels of trait anger may be a consequence of PTSD (i.e., a “scar model,” which states that a personality trait may be affected by the disorder; Clark et al., 1994). These models are not mutually exclusive, and the relationship between trait anger and PTSD may be reciprocal.

Most studies about the anger-PTSD link are limited by cross-sectional designs. Some longitudinal studies, however, have been conducted using state anger measures. These studies showed that levels of anger after a traumatic event predicted later posttraumatic stress (Ehlers, Mayou, & Bryant, 2003; Feeny, Zoellner, & Foa, 2000; Jayasinghe, Giosan, Evans, Spielman, & Difede, 2008; Riggs, Dancu, Gershuny,

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This study was supported by an open competition grant (400-07-181) and a Vidi innovational research grant (awarded to I.M.E.) from the Netherlands Organization of Scientific Research (NWO). The authors thank the participants for their time and effort, Col-MD Kees IJzerman from the Dutch Military Mental Health Care for support, and Arne Leer and research assistants for their help in the data collection.

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DOI: 10.1002/jts.21904

Greenberg, & Foa, 1992). Because level of trait anger reflects level of state anger over time and across situations, these constructs correlate positively (Deffenbacher, 1992). Consequently, the longitudinal data seem to support the diathesis-stress model and pathoplasty model. The evidence, however, is not unequivocal. A recent study with a large sample of crime victims found that PTSD symptoms predicted subsequent levels of state anger, but not the reverse (Orth et al., 2008), supporting the scar model. These longitudinal studies with assessments after trauma exposure lack control for the effects of trauma exposure or baseline (pretrauma) PTSD symptoms on anger levels. Prospective research in which anger is assessed before trauma exposure, and baseline symptoms are taken into account is scarce. To our knowledge, only three such studies have been published so far. One study tested 43 firefighters immediately after and at several time points 6–24 months after their training, showing that initial higher levels of hostility predicted PTSD symptoms at 24 months after training (Heinrichs et al., 2005). Levels of hostility in 470 soldiers before deployment also predicted PTSD symptoms 6 months after deployment (van Zuiden et al., 2011). Another study among 180 police cadets showed that trait anger during training predicted PTSD symptoms 12 months later (Meffert et al., 2008). These data support the diathesis-stress model, but there are several methodological limitations. First, these studies did not all account for initial PTSD symptoms (Heinrichs et al., 2005; Meffert et al., 2008), and trauma exposure (Meffert et al., 2008; van Zuiden et al., 2011). Second, none of these studies tested whether PTSD symptoms predicted subsequent trait anger. Thus, the temporal relationship between trait anger and PTSD remains fairly unclear.

Another important question is whether anger predicts PTSD symptoms beyond well-known vulnerability factors. Increased anger levels may reflect a specific negative emotion at a lower-order level of the more general trait of neuroticism (or “negative affectivity”; Watson & Clark, 1992), which is related to anxiety disorders and depression (Clark et al., 1994). Prospective research has shown that pretrauma neuroticism scores predict later PTSD symptoms (Engelhard, van den Hout, & Lommen, 2009; van den Hout & Engelhard, 2004). Therefore, it seems important to test whether the relationship between trait anger and PTSD symptoms remains after accounting for pretrauma neuroticism.

In the current study, 249 Dutch soldiers were tested 2 months before their deployment to Uruzgan, Afghanistan, and twice (2 months and approximately 9 months) after returning home. Using structural equation modeling, we tested the temporal relationship between trait anger and PTSD symptom severity, with known risk factors including pretrauma symptoms and stressor severity (e.g., Brewin, Andrews, & Valentine 2000) included in the model. The unique predictive value of the predeployment trait anger on PTSD symptom severity was tested by including neuroticism (e.g., Engelhard et al., 2009; Parslow, Jorm, & Christensen, 2006) in the model. Furthermore, we tested if the relationship could be explained by content overlap by exclud-

ing the irritability item from the PTSD measure. We hypothesized that trait anger before deployment would predict PTSD symptom severity 2 months after deployment, and PTSD symptoms at 9 months directly or indirectly through earlier PTSD symptom severity. We expected a positive relationship between PTSD symptom severity and trait anger after deployment, and that the predictive value of trait anger would remain after accounting for pretrauma symptoms, stressor severity, neuroticism, and content overlap.

Method

Participants and Procedure

Before a 4-month deployment to Afghanistan (November 2009–March 2010), 250 Dutch Royal Army soldiers were asked to participate in this study. One refused, and the others agreed and were tested about 2 months before their deployment (T1; $N = 249$). They filled out questionnaires measuring anger, PTSD symptom severity, and neuroticism.

The sample (98.4% were male) had a mean age of 23.82 years ($SD = 4.94$). The highest attained educational level was primary (2.0%), secondary (92.0%), or higher education (6.0%). About 34.0% were married or cohabiting, 38.1% were in a relationship but not cohabiting, and 27.9% were single. About 43.4% had not been deployed before.

Approximately 2 months after returning home (T2), 247 soldiers were retested, using a diagnostic interview for PTSD (response rate = 99.2%), administered by trained research psychologists, and questionnaires measuring anger, PTSD symptom severity, and deployment stressors (response rate = 96.8%). At follow-up (T3), about 9 months after returning home, 221 soldiers (response rate = 88.8%) were reassessed using questionnaires. Testing typically took place on a military base, but for about a third of the group the final assessment was by postal mail. Nonresponse was partly due to soldiers who were unreachable (e.g., after a transfer) or withdrew from the study.

Soldiers were recruited on military bases across The Netherlands and selection was based on availability during their preparation program. After individual oral and written information about the study was given by the researcher, written informed consent was obtained. Participation was strictly voluntary without financial compensation. This study was part of a larger project (see Lommen, Engelhard, Sijbrandij, van den Hout, & Hermans, 2013), and was approved by the Institutional Review Board of Maastricht University.

Measures

Trait anger was assessed with the Dutch version (van der Ploeg, Defares, & Spielberger, 1982) of the State-Trait Anger Scale (STAS-T; Spielberger, Jacobs, Russell, & Crane, 1983). Trait anger was assessed with 10 items scored on a 4-point scale (1 = *almost never*; 2 = *sometimes*; 3 = *often*; 4 = *almost always*). The mean of the underlying items was computed.

Internal consistency was good, with .85, .86, and .88 at T1, T2, and T3, respectively.

PTSD symptom severity was assessed with the Dutch version (Engelhard, Arntz, & van den Hout, 2007) of the Posttraumatic Symptom Scale-Self Report (PSS; Foa, Riggs, Dancu, & Rothbaum, 1993). The 17 *DSM-IV* PTSD symptoms according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; APA, 2000) were each rated on a 0 = *not at all* to 3 = *almost always* scale for the prior month. Participants were instructed to rate the PSS with respect to an event (before deployment: their most aversive life-event, after deployment: deployment-related event) that troubled them the most at the time of testing. The sum score was used for descriptive purposes. For the statistical analyses, the mean score of the three subscales (five reexperiencing items, seven avoidance items, five hyperarousal items) was used. The PSS is valid and reliable (Engelhard et al., 2007; Foa et al., 1993). Internal consistency was .85, .81, and .85 at T1, T2, and T3, respectively.

PTSD diagnosis was determined using the Dutch version (van Groenestijn, Akkerhuis, Kupka, Schneider, & Nolen, 1999) of the Structured Clinical Interview for *DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996).

Stressful events were assessed using a Dutch version (Engelhard & van den Hout, 2007) of the Potentially Traumatizing Events Scale (PTES; Maguen, Litz, Wang, & Cook, 2004). Of the original 21 items representing warzone-related stressors, one was omitted, four were added, and another item was changed to reflect the situation in Afghanistan (see Lommen et al., 2013). For each of the 24 events, participants indicated whether they had experienced it in Afghanistan. Number of endorsed events was used as indicator of stressor severity.

Neuroticism was assessed with the Dutch version (Sanderman, Arrindell, Ranchor, Eysenck, & Eysenck, 1991) of the neuroticism scale of the Eysenck Personality Questionnaire-short version (EPQ-N; Eysenck & Eysenck, 1975). The EPQ-N consists of 22 items that were answered with *yes* = 1 or *no* = 0. The sum score was used. The scale has good psychometric properties (Sanderman et al., 1991). Internal consistency was .81 at T1.

Data Analysis

Analyses were conducted with Mplus 6.11 (Muthén & Muthén, 2010). Cross-lagged analyses were used to assess the temporal relationship between the latent variables PTSD symptom severity (PSS) and trait anger (STAS-T). First, subscales of the PSS and item parcels of the STAS-T were used in the structural equation model as indicators of latent variables (cf. Orth et al., 2008) because of the limited sample size in combination with our complex statistical model. Three unidimensional sets of items (item parcels; two of three items and one of four items) of the STAS-T were created based on statistical grounds (Kline, 2010; high item intercorrelations). Second, to ensure that comparisons of the latent variables PSS and STAS-T over time are valid, the underlying structure of the latent variables should

be invariant over time. Separate measurement models for PSS and STAS-T tested for measurement invariance (Brown, 2006; van de Schoot, Lugtig, & Hox, 2012). Third, the cross-lagged panel model with PSS and STAS-T was run, when accounting for stressor severity (PTES). Stability paths of PSS and STAS-T over time were estimated to control for previous PSS and STAS-T scores. Identical parcels of PSS and STAS-T over time were allowed to correlate. Cross-sectional and partial correlations between PSS and STAS-T were also estimated. The main interests concerned the cross-lagged paths between the PSS and STAS-T. Satorra-Bentler scaled χ^2 difference (*TRd*) tests were used to examine whether the cross-paths contributed significantly to the model fit. Fourth, to test if the effects that have been found in the cross-lagged panel model were robust, additional structural models were run including covariates like neuroticism (EPQ-N).

A robust maximum likelihood estimator (MLR) was used because it is robust to nonnormality of the data. Data appeared missing at random (i.e., missingness at T2 did not depend on age, education, PSS at T1, or STAS-T at T1, missingness at T3 did not depend on age, education, PSS at T1 or T2, or STAS-T at T1 or T2, smallest $p = .081$). Therefore, participants with missing data were included in the analyses and full information maximum likelihood (FIML) was used to estimate our model. Model fit was evaluated using root mean square error of approximation (RMSEA; Steiger, 1990), comparative fit index (CFI; Bentler, 1990), and Tucker-Lewis index (TLI; Tucker & Lewis, 1973). Conventional guidelines were followed, considering $< .08$ as an adequate fit for the RMSEA value, and $> .90$ as adequate fit for CFI and TLI. The χ^2 is reported to compare models. Paths in the cross-lagged panel models were tested one-sided (see Kline, 2010 for an overview of fit statistics).

Results

On average, participants reported experiencing 14.21 ($SD = 4.65$) warzone-related stressors, including witnessing an explosion (84.6%), being shot at (60.6%), having to remove human remains (37.8%), and seeing dead or injured Dutch soldiers (22.4%). At T2, 2.8% met the diagnostic criteria for partial or full PTSD according to the SCID. One participant met criteria for PTSD, six persons met criteria for partial PTSD (Engelhard, van den Hout, Arntz, & McNally, 2002), mainly due to not meeting avoidance criteria. At T2, 2.9% of the participants scored above the clinical cutoff of 14 (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006) on the PSS, and 5.0% screened positive for PTSD based on symptom count (with a score of at least 1 indicating symptom presence; Foa & Tolin, 2000). At T3 these rates were 6.3% and 10.0%, respectively. The mean PSS scores were 4.07 ($SD = 4.79$), 3.10 ($SD = 3.81$), and 4.05 ($SD = 4.73$), for T1, T2, and T3, respectively. PSS and STAS-T scores did not differ between individuals with or without prior deployment, largest $t = 1.69$, smallest $p = .092$.

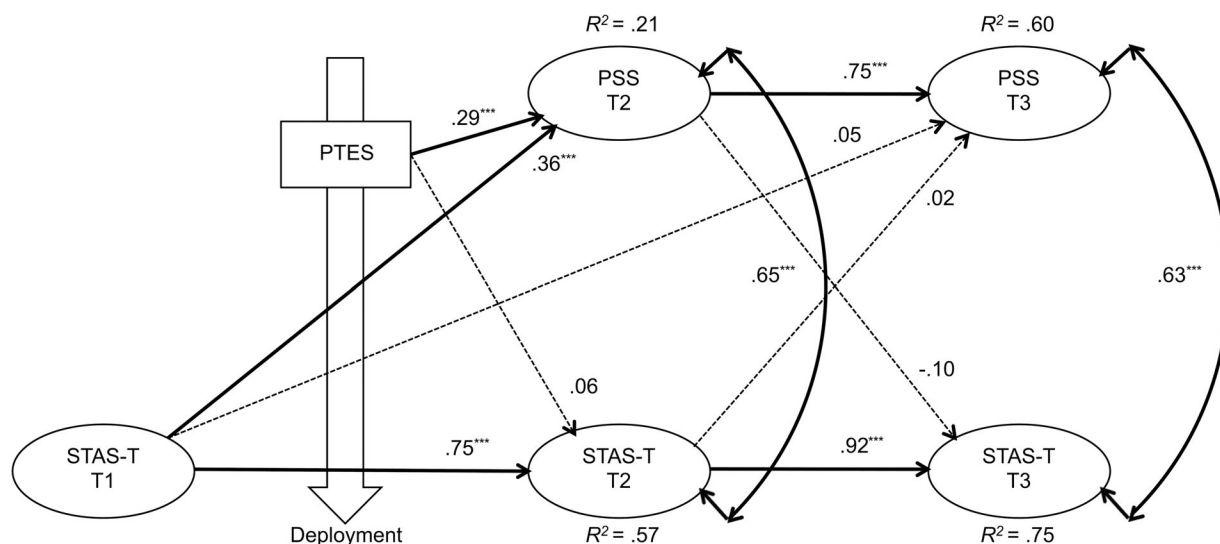


Figure 1. Model 1, including trait anger (STAS-T; State-Trait Anger Scale) at T1, T2, and T3; PTSD symptom severity (PSS; Posttraumatic Symptom Scale-Self Report) at T2 and T3; and stressor severity (PTES; Potentially Traumatizing Events Scale). Values shown are standardized coefficients. Solid lines represent significant relations; dashed lines represent nonsignificant relations. *** $p < .001$.

To test for measurement invariance, model fit of the measurement models was evaluated with confirmatory factor analysis (CFA). The unconstrained CFA model (factor loadings and intercepts of the latent variable were freely estimated), CFA model with loading invariance (constrained factor loadings), and the CFA model with intercept invariance (constrained factor loadings and intercepts) were compared.

For PSS at T1, T2, and T3, the unconstrained CFA model fit was acceptable, $\chi^2(16) = 31.07$, RMSEA = .06, CFI = .97, TLI = .93. The model with loading invariance and the model with intercept invariance showed degraded, and unacceptable model fit, smallest $TRd(4) = 27.23$, largest $p < .001$. Both factor loadings and intercepts appeared to be different over time. From a theoretical perspective it is arguable that the PSS before (T1) and after (T2 and T3) deployment differ, as the PSS at T1 refers to different stressful events. Therefore, we decided to test measurement invariance of the CFA model including only PSS at T2 and T3. The unconstrained model showed acceptable fit, $\chi^2(5) = 4.32$, RMSEA = .00, CFI = 1.00, TLI = 1.00. The model fit did not significantly degrade when loading invariance was imposed, $TRd(2) = 5.66$, $p = .059$. Compared to the latter model, the model that imposed intercept invariance showed degraded model fit, $TRd(2) = 6.63$, $p = .036$. Nevertheless, all measurement models showed acceptable model fit, largest RMSEA = .06, smallest CFI = .97, smallest TLI = .96. For this reason, the constrained model was used in further analyses, despite the partial measurement invariance. To make clear that the relationship of the indicators to the construct differed between PSS at T1 and PSS at T2/T3, PSS at T1 has been renamed as baseline symptoms and was included in the cross-lagged model as a covariate with factor loadings and intercepts that were freely estimated.

For STAS-T at T1, T2, and T3, the model fit of the unconstrained CFA model, $\chi^2(16) = 19.96$, RMSEA = .03, CFI = 1.00, TLI = .99 was acceptable and was not different for the model that imposed loading invariance, $TRd(4) = 2.19$, $p = .701$. Compared to the latter model, the model that imposed intercept invariance showed degraded model fit, $TRd(4) = 56.27.00$, $p < .001$. Nevertheless, all measurement models showed acceptable fit, largest RMSEA = .09, smallest CFI = .95, smallest TLI = .93. The RMSEA value was .087, which is at the boundary of an acceptable fit. But because this cutoff is debatable (e.g., $< .10$ according to Kline, 2010, p. 206) and other fit indices (CFI and TLI) showed good fit, we evaluated the model fit as acceptable and the constrained CFA model was used in further analyses.

After evaluation of the measurement models, cross-lagged models were used to test the temporal relation between PSS and STAS-T. In Model 1, a cross-lagged panel model including PSS at T2 and T3, STAS-T at T1, T2, and T3, and PTES was run (see Figure 1). Model fit was acceptable, $\chi^2(94) = 172.49$, RMSEA = .06, CFI = .95, TLI = .93. Stability paths, correlations, and autocorrelations were all significant. PTES predicted PSS at T2. The only significant cross-lagged path was trait anger at T1 as a predictor of PSS at T2. The results remained similar in a multigroup analysis examining the model in participants with and without prior deployment experience, and when intercepts of the latent variables were freely estimated. An additional analysis was run to assess the indirect effect of STAS-T at T1 on PSS at T3 through PSS at T2, which indicated a significant indirect effect, $\beta = .27$, $p = .002$.

A χ^2 difference test showed the necessity of the cross-lagged paths in the model, as omitting these paths led to a degraded model fit, $TRd = 15.68$, $df_{diff} = 4$, $p = .003$. Further, to make sure

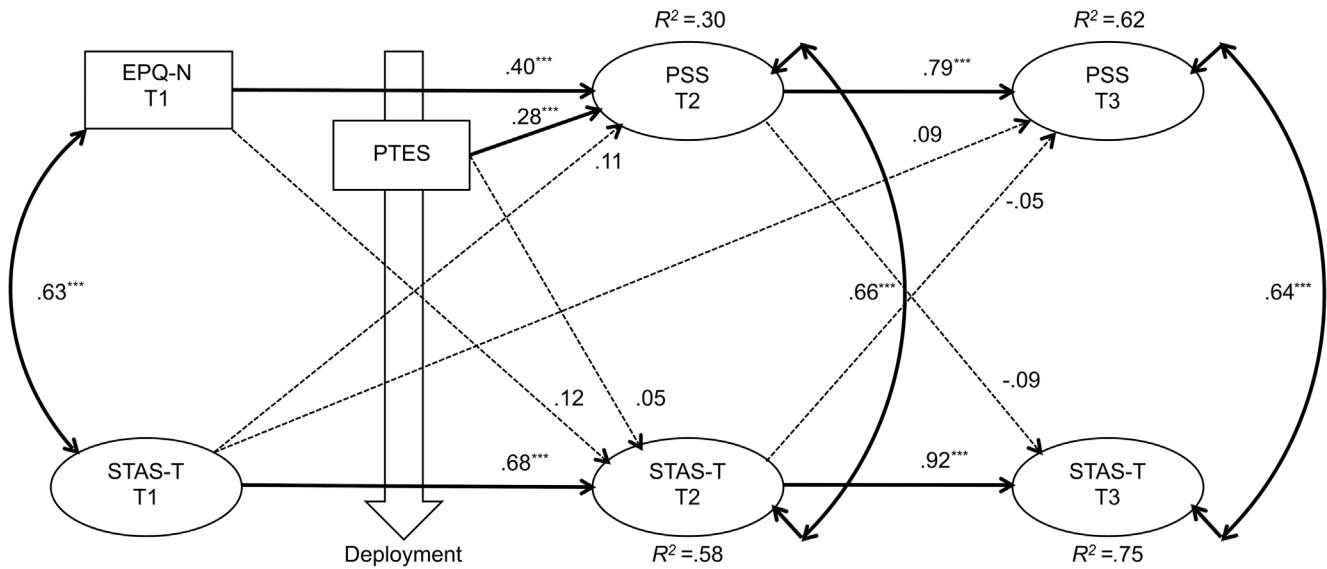


Figure 2. Model 3, including trait anger (STAS-T; State-Trait Anger Scale) at T1, T2, and T3; PTSD symptom severity (PSS; Posttraumatic Symptom Scale-Self Report) at T2 and T3; stressor severity (PTES; Potentially Traumatizing Events Scale); and neuroticism (EPQ-N; Eysenck Personality Questionnaire-short version). Values shown are standardized coefficients. Solid lines represent significant relations; dashed lines represent nonsignificant relations. $***p < .001$.

that the relation between PSS and STAS-T did not depend on content overlap, the model was reanalyzed with the irritability item removed from the PSS. Results showed similar patterns and conclusions as the model including the irritability item (trait anger at T1 predicted PSS at T2, $\beta = .32, p = .001$). Moreover, decrease in correlation between PSS and STAS-T at T2 ($r = .60$) and T3 ($r = .58$) was minor, which indicated that the relation between PSS and STAS-T was not a methodological artifact.

Because of the lack of measurement invariance, baseline symptoms could not be included in the cross-lagged model. Theoretically, it is of great importance to include baseline symptoms in the model to rule out that symptomatology after deployment does not simply reflect predeployment symptomatology. Baseline symptoms were therefore added as a covariate to the cross-lagged panel model, with freely estimated factor loadings and intercepts (Model 2). Model fit was acceptable, $\chi^2(139) = 256.08, RMSEA = .06, CFI = .93, TLI = .92$, and earlier model results were replicated. Both covariates, PTES and baseline symptoms significantly predicted PSS at T2, and again the only significant cross-path was STAS-T at T1 predicting PSS at T2.

To test the robustness of the cross-lagged panel model with neuroticism (EPQ-N) in the model, Model 1 was extended with EPQ-N as a covariate (Model 3, see Figure 2). Model fit was acceptable, $\chi^2(107) = 188.22, RMSEA = .06, CFI = .95, TLI = .93$. No cross-lagged paths reached significance. Both EPQ-N and PTES at T1 significantly predicted PSS at T2, but not STAS-T at T2.

In sum, the addition of covariates to the cross-lagged panel model showed that STAS-T at T1 significantly predicted PSS at T2 with baseline symptoms at T1 in the model, but not

with EPQ-N in the model. No other cross-lagged paths were significant.

Discussion

This prospective study examined the temporal relationship between trait anger and PTSD symptom severity in Dutch soldiers who were assessed before, and twice after their deployment to Afghanistan, using a cross-lagged panel analysis in a structural equation modeling framework. The main findings can be summarized as follows. First, in line with our hypothesis, trait anger before deployment predicted PTSD symptom severity 2 months after deployment, and indirectly at 9 months after deployment through PTSD symptom severity shortly after deployment. Second, against our expectations, trait anger 2 months after deployment did not predict PTSD symptom severity at 9 months, and PTSD symptom severity 2 months after deployment did not predict trait anger at 9 months after deployment. Third, the predictive effect of predeployment trait anger on subsequent PTSD symptom severity remained when stressor severity, content overlap, and baseline symptoms were included in the model, but not when neuroticism was included.

To the best of our knowledge, this is the first prospective study that assessed trait anger and PTSD symptoms over time, and taking baseline symptoms into account. The current finding that predeployment trait anger predicts subsequent PTSD symptom severity is in line with other prospective studies (Heinrichs et al., 2005; Meffert et al., 2008; van Zuiden et al., 2011), and supports the diathesis-stress model. Trait anger after deployment did not predict later PTSD symptom severity, which contradicts the pathoplasty model, and PTSD symptom

severity initially after deployment did not predict trait anger at follow-up. The latter suggests that trait anger is not affected by PTSD symptom severity, which is in contrast with predictions according to the scar model. An earlier longitudinal study (Orth et al., 2008) that tested the temporal relation between anger and PTSD symptoms did not find effects of anger on PTSD symptoms either, but in contrast with our results, PTSD symptoms did predict subsequent anger. Methodological differences may account for the different findings because Orth et al. (2008) measured state anger, which might be more sensitive for change than trait anger.

Our findings replicate earlier findings that neuroticism predicts PTSD symptoms (Bramsen, Dirkzwager, & van der Ploeg, 2000; Engelhard et al., 2009; Parslow, Jorm, & Christensen, 2006; van den Hout & Engelhard, 2004). The results of the current study indicate that neuroticism and trait anger are both vulnerability factors for PTSD symptom severity, with neuroticism as the stronger predictor. Trait anger seems to reflect a lower-order factor of the more general vulnerability factor neuroticism (cf. Watson & Clark, 1992). Because the analyses did not examine the lower-order components of neuroticism, however, it remains unclear if the effect for neuroticism in the final model is driven primarily by the anger facet as opposed to neuroticism overall. A challenging question is through what mechanism pretrauma neuroticism and trait anger predict later PTSD symptom severity. In concordance with the survival-mode theory (Novaco & Chemtob, 2002), anger, similar to neuroticism and trait anxiety, may facilitate threatening interpretations of ambiguous stimuli (Barazzone & Davey, 2009; Wenzel & Lystad, 2005). Furthermore, neuroticism is associated with avoidance of (harmless) ambiguous stimuli (Lommen, Engelhard, & van den Hout, 2010). Anger and neuroticism may also lead to negative appraisals of (the sequelae of) a traumatic event and avoidance strategies intended to control perceived threat, which are both involved in the maintenance of PTSD symptoms (e.g., Bovin & Marx, 2011; Ehlers & Clark, 2000; van den Hout & Engelhard, 2004).

Several limitations of this study should be taken into account. First, only a small number of participants had high levels of PTSD symptoms. Earlier studies also reported relatively low deployment-related PTSD rates in Dutch and British soldiers (about 4%; Engelhard et al., 2007; Sundin, Forbes, Fear, Dandeker, & Wessely, 2011), and U.S. soldiers (about 7%; Bonanno et al., 2012; see also, McNally, 2012) deployed to Afghanistan or Iraq. The low levels of symptoms in the current study might affect generalizability of the findings, and leave little variance for predictive variables. There was likely low power in the models to detect small effects. Second, PTSD symptom severity was assessed with self-reports. It cannot be ruled out that some participants endorsed PTSD symptoms after deployment that were unrelated to a traumatic event. The prospective design, however, allowed us to assess initial PTSD symptoms (i.e., before deployment) to account for general response tendencies and for symptoms that were present before deployment. Third, most participants were healthy, young male soldiers. It

is unclear to what extent the current findings generalize to other trauma types and samples. Three earlier studies, however, that tested other occupational samples at risk of trauma exposure have found similar results on the predictive value of pretraumatic anger (Heinrichs et al., 2005; Meffert et al., 2008; van Zuiden et al., 2011). Fourth, baseline symptoms assessed with the PSS before deployment loaded on different factors than the PSS after deployment, probably reflecting the heterogeneity of the group before deployment. The different factor loadings before versus after deployment are not surprising, as these are anchored to different stressful events, and the events are likely more recent and less variable after deployment than at baseline. Nevertheless, it makes the understanding of the results somehow more complex, as some of the change in the latent variables may be explained by temporal variation in the measurement properties of the indicators of these variables (Brown, 2006). Fifth, trait anger reflects the experience of angry feelings over time and does not necessarily lead to (problematic) anger expression (Eckhardt, Barbour, & Stuart, 1996). Although trait anger may predispose to PTSD psychopathology, it might not be problematic in itself. In the current study, mean trait anger scores were relatively low compared to a reference group of randomly selected young men (<40 years) in The Netherlands (van der Ploeg, Defares, & Spielberger, 1982). Before deployment, only 14.8% of our sample scored above the average score of the reference group. About 2 months after deployment, this was 11.6%, and about 9 months after deployment, it was 18.0%. It seems unlikely that trait anger was underreported in our study because the PTSD self-report questionnaire showed that 41.2% reported they *sometimes* have had heightened irritability or anger outbursts during the prior month.

Strengths of this study include the prospective, longitudinal design with a measurement before trauma exposure, the large sample with low attrition rates, inclusion of a trait anger measure over time, inclusion of significant risk factors of PTSD, and the statistical method to analyze the data.

To summarize, our findings suggest that trait anger is a pretrauma vulnerability factor for the development of PTSD symptoms, but does not explain unique variance over and above the higher-order trait variable of neuroticism. Individuals with a high level of trait anger or neuroticism before trauma exposure might be at greater risk to develop later PTSD symptoms. Trait anger did not seem to be a consequence of PTSD symptomatology.

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