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Anger, Hostility, and Posttraumatic Stress Disorder in Trauma-Exposed Adults: A Meta-Analysis

Ulrich Orth and Elias Wieland
University of Berne

This meta-analysis synthesizes the available data on the strength of association between anger and posttraumatic stress disorder (PTSD) and between hostility and PTSD, covering 39 studies with trauma-exposed adults. Effect sizes did not differ for anger and hostility, which could therefore be combined; effect sizes for anger expression variables were analyzed separately. The analyses revealed large effects. The weighted mean effect size (*r*) was .48 for anger–hostility, .29 for anger out, .53 for anger in, and −.44 for anger control. Moderator analyses were conducted for anger–hostility, showing that effect sizes were substantially larger with increasing time since the event and that effect sizes were larger in samples with military war experience than in samples that had experienced other types of traumatic events.

Keywords: posttraumatic stress disorder, anger, hostility

Common stereotypes portray traumatized individuals as full of fear and helplessness but not full of anger and hostility. Indeed, for a long time, theories of posttraumatic stress disorder (PTSD) focused on fear as the central emotion in the disorder and did not consider anger and hostility to be central attributes of individuals suffering from PTSD (cf. Brewin & Holmes, 2003). Consequently, empirical studies investigating anger and hostility in PTSD were sparse.

However, the definition of PTSD found in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev. [*DSM–III–R*], American Psychiatric Association, 1987; 4th ed. [*DSM–IV*], American Psychiatric Association, 1994) lists irritability and outbursts of anger as one of the hyperarousal symptoms. Moreover, Novaco and Chemtob (1998) pointed out in their review on anger and PTSD that in fact early observations showed anger to be a prevalent component of posttraumatic stress reactions, particularly in veterans with combat experience. For about the last decade, an increasing number of studies have explicitly focused on anger in individuals with PTSD (e.g., Chemtob, Hamada, Roitblat, & Muraoka, 1994; Frueh, Henning, Pellegrin, & Chobot, 1997; Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992; Schützwohl & Maercker, 2000). Taken together, the results of these studies suggest that anger is substantially associated with PTSD severity. Therefore, we decided to summarize the available data on the strength of association between anger and PTSD by means of a meta-analysis.

Of note, Novaco and Chemtob (2002) looked at whether the correlation between anger and PTSD is artificially inflated by the fact that anger is one of the diagnostic criteria for PTSD. However, they found that the correlation did not substantially decrease when items measuring anger and irritability within PTSD scales were removed. Orth, Cahill, Foa, and Maercker (2006) investigated this issue in another sample, female crime victims, with four repeated assessments of PTSD and anger. The results showed that the correlations likewise did not substantially decrease (from .38 to .36 for Time 1, from .39 to .36 for Time 2, from .51 to .49 for Time 3, and from .42 to .41 for Time 4). Thus, the results of both studies indicate that the correlation between PTSD and anger is not a methodological artifact.

The question, then, is which psychological processes cause anger to be related with PTSD symptomatology? One theory explaining the relation between anger and PTSD, which can be labeled *survival mode theory*, was formulated by Chemtob, Novaco, and colleagues (see Chemtob, Novaco, Hamada, Gross, & Smith, 1997; Novaco & Chemtob, 1998). They hypothesize that individuals suffering from PTSD have a substantially lowered threshold to perceiving situations as threatening and further that the perception of threat activates a biologically predisposed survival mode, including fear and flight reactions as well as anger and fight reactions. Another theory explaining the relation between anger and PTSD, which can be labeled *fear avoidance theory*, was introduced by Foa and colleagues (Feeny, Zoellner, & Foa, 2000; Foa, Riggs, Masie, & Yarczower, 1995; Riggs et al., 1992). They hypothesize that individuals with PTSD are motivated to avoid trauma-related feelings of fear, which are activated by posttraumatic intrusions, and that trauma-related anger serves as a welcome focus of attention because anger is an emotion with a more positive emotional valence than fear.

The theories described above focus on anger and PTSD but not on hostility. However, the concepts of anger and hostility are closely linked to each other. Anger is understood as an emotion and is defined by its characteristic cognitive, physiological, motivational, and behavioral components (cf. Berkowitz, 1999; Izard, 1991). In particular, the cognitive component involves the percep-

Ulrich Orth and Elias Wieland, Department of Psychology, University of Berne, Berne, Switzerland.

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Correspondence concerning this article should be addressed to Ulrich Orth, Department of Psychology, University of Berne, Muesmattstrasse 45, 3012, Berne, Switzerland. E-mail: ulrich.orth@psy.unibe.ch

tion that important personal goals are blocked by improper action of an external agent, and the motivational component frequently involves hostile and aggressive impulses. On the other hand, hostility is understood as an attitudinal construct and is defined as a predisposition to dislike and mistrust others and to interpret their behavior as egoistic and hurtful (cf. Miller, Smith, Turner, Guijarro, & Hallet, 1996). As a consequence, hostility causes an increased frequency of anger and aggression. Thus, anger and hostility may reciprocally activate each other and motivate the individual to aggressive behavior against others.

Measures of anger and hostility frequently contain overlapping content. For example, the Buss–Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957) comprises in its revised form (Aggression Questionnaire; Buss & Perry, 1992) four scales measuring anger, hostility, verbal aggression, and physical aggression, indicating that the BDHI measures not only an attitudinal construct but also the related affective and behavioral constructs anger and aggression. The Cook–Medley Hostility Inventory (Cook & Medley, 1954) measures affective and behavioral components besides hostility (Eckhardt, Norlander, & Deffenbacher, 2004). As a last example, the Hostility Scale of the Symptom Checklist—90 (SCL–90; Derogatis, Lipman, & Covi, 1973; Derogatis & Savitz, 1999) also contains items that measure anger. Therefore, it is no surprise that measures of anger and hostility are often strongly correlated (cf. Eckhardt et al., 2004).

Given the strong conceptual and empirical links between anger and hostility, we decided to include both anger and hostility in the meta-analysis and to test whether using a combined effect size for anger and hostility is justified. A combined effect size would have the advantage of greater reliability (because of a larger number of studies) and greater generalizability (because results would be valid for both anger and hostility). To combine the effect sizes, two conditions must be fulfilled: The effect sizes must be strongly correlated, and the mean effect sizes must be at the same level.

The first goal of the meta-analysis was to determine the strength of association between anger and PTSD and between hostility and PTSD, respectively, in trauma-exposed adults. Some of the frequently used measurement instruments of anger comprise, besides measures of anger intensity or frequency, measures of anger expression styles, such as *anger out* (expression of anger in verbal or physically aggressive behavior toward others or objects), *anger in* (inhibition and suppression of anger), and *anger control* (control of the experience and expression of anger). Effect sizes for these variables provide additional information, which might be useful in future theorizing about the relation between anger and PTSD. Therefore, we decided to include anger expression variables in the meta-analysis.

The second goal of the meta-analysis was to test potential moderators of the relation between anger, hostility, and PTSD. We hypothesized that type of event and time since event should be important moderators of the relation, for reasons outlined below. In addition, we included mean age and gender in the analysis for exploratory reasons, because age and gender are basic sample characteristics taken into account in meta-analyses as a matter of standard. However, we did not have hypotheses as to why age and gender might moderate the relation between anger and PTSD or between hostility and PTSD, respectively.

A first important moderator might be the type of traumatic event that led to PTSD. Anger as an important topic in PTSD emerged primarily in studies with Vietnam War veterans (e.g., Beckham et al., 1996; Chemtob et al., 1994; Frueh et al., 1997; Lasko, Gurvits, Kuhne, Orr, & Pitman, 1994), which might indicate that anger is a particularly salient problem in veterans suffering from PTSD. Therefore, we tested whether samples with military war experience show a particularly strong relation between anger and PTSD and between hostility and PTSD, respectively. A potential factor is that individuals selected for military service might have substantially higher assertiveness, aggressiveness, and trait anger even before experiencing a traumatic event.

A second important moderator might be the time that has passed since the event. Of interest, the few longitudinal studies that repeatedly measured both anger and PTSD suggest that the correlation between anger and PTSD becomes stronger with increasing time since the event. For example, in the study of Feeny et al. (2000), the correlation increased from .07 after 2 weeks to .21 after 4 weeks to .38 after 12 weeks. In the study by Ehlers, Mayou, and Bryant (1998) the correlation between anger and PTSD was .37 after 3 months and .47 after 1 year. Theories of associative networks in memory (see Bower, 1981) might provide an explanation for the time-dependent increase of the correlation. At the beginning, the association between fear and anger structures might be weak, but repeated simultaneous activation of the memory structures should increasingly strengthen the association. We hypothesized that the relation between the effect size (correlation of anger and PTSD or hostility and PTSD) and time since event does not follow a linear function but, rather, a logarithmic function, because under the assumption of stationary causal processes (i.e., stable causal effects), the correlation between two variables initially increases after the onset of the causal processes (i.e., the traumatic event) and then reaches equilibrium over time (i.e., converges to a fixed value; Cole & Maxwell, 2003). Indeed, a longitudinal study of anger and PTSD showed that the assumption of stationary causal processes is empirically justified (Orth et al., 2006).

Method

Selection of Studies

English-language journal articles, books, and book chapters were searched in the computerized databases PsycINFO and MEDLINE, as well as the PILOTS database produced by the National Center for PTSD, for all years covered in the databases through 2003. We did not search for dissertations or unpublished reports, as our later analyses strongly suggested that no publication bias exists (see Results section). We used the following search terms: *anger*, *hostility*, *PTSD*, *traumatic stress*, *posttraumatic stress* (thus including *posttraumatic stress disorder* and *posttraumatic stress reactions*). In PsycINFO, we additionally searched for *traumatic neurosis* and *stress reactions* in the entries before 1985, as recommended in the database. The assessment of the abstracts yielded 189 potentially relevant articles. These articles were then assessed in full text by both authors of this meta-analysis. The interrater agreement on inclusion or exclusion in the meta-analysis was high ($\kappa = .95$) and all differing assessments were discussed until consensus was reached.

Studies were included in the meta-analysis if the following criteria were fulfilled: (a) All participants of the sample were trauma exposed—that is, they had experienced a traumatic event as defined by Diagnostic Criterion A1 of the *DSM–IV* (American Psychiatric Association, 1994); if the sample comprised non-trauma-exposed participants and if it was possible to restrict the effect size computation to the trauma-exposed subsample, we included the study in the meta-analysis, and the values of sample charac-

teristics were computed only for the trauma-exposed subsample; (b) anger variables or hostility variables were assessed; relevant anger measures were trait and state measures of anger, as well as the anger expression variables anger out, anger in, and anger control, as measured, for example, by the State–Trait Anger Expression Inventory (STAXI; Spielberger, Sydeman, Owen, & Marsh, 1999); (c) PTSD diagnosis or severity of posttraumatic stress reactions was assessed; (d) enough information was given to compute an effect size; and (e) at the time of measurement, participants were 16 years or older.

If a sample was analyzed by more than one study, only one study was included in the meta-analysis to ensure independence of effect sizes. In these cases, we included the study that provided the most comprehensive coding information (e.g., the highest number of anger and hostility measures) and excluded the other studies. To the best of our knowledge, the following samples were analyzed by more than one study: The sample of Calhoun, Beckham, Feldman et al. (2002), which we included, was also used by Beckham, Feldman, Kirby, Hertzberg, and Moore (1997), Beckham et al. (1996), Beckham et al. (2002), and Calhoun, Beckham, and Bosworth (2002), which we excluded; the sample of Ehlers et al. (1998), which we included, was also used by Mayou, Ehlers, and Bryant (2002), which we excluded; the sample of Feeny et al. (2000), which we included, was also used by Zoellner, Foa, and Brigidi (1999), which we excluded; and the sample of Z. Solomon, Mikulincer, and Bleich (1988), which we included, was also used by Z. Solomon (1989), which we excluded.

Studies were also excluded if anger–hostility and PTSD were not assessed at the same time point, because our aim was to investigate the cross-sectional association between the variables. For this reason, we excluded the study of Riggs et al. (1992), in which anger was assessed 1 week after the event but PTSD was assessed 1 month after the event.

Finally, studies were excluded if conflicting information needed for the computation of effect sizes was given in, for example, the text, the abstract, tables, or figures. For this reason, we excluded the studies by Ford, Fisher, and Larson (1997), Hyer et al. (1986), and Roberts et al. (1982).

This procedure left 38 articles for analysis. All studies were published as journal articles, none as books or book chapters. The article of Hovens, Bramsen, and van der Ploeg (2002) provided two relevant samples; thus, our data set comprised 39 studies.

Coding of Studies

We coded the following data: sample size, country of origin, mean age of participants, proportion of female participants, type of traumatic event (criminal victimization including rape, military war experience, civilian war experience, technological disaster, natural disaster, health trauma, witnessing a traumatic event or being confronted with the traumatic event of a significant other, or mixed), mean time since event, measures of anger and hostility, measures of PTSD, and effect sizes. Effect sizes were coded separately for five variables (anger, anger out, anger in, anger control, and hostility).

Unfortunately, mean time since event was not reported in 22 of 39 studies. However, in 18 of these 22 studies, the event was clearly indicated in the text (e.g., Vietnam War) and the study reported exclusively on samples with military war experience. Thus, data were not missing at random, but missingness of data was substantially confounded with type of event and, consequently, with the variable for which data were missing itself (the average time since event was supposedly much larger for samples with military war experience compared with the remaining samples). However, for cases in which data are not missing at random and the number of missing values is not negligible, procedures to deal with missing data such as case deletion or missing data imputation yield significantly biased results (cf. Schafer & Graham, 2002). The only alternative allowing for an analysis of the variable was to estimate time since event on the basis of information explicitly given in the study, using the following procedure, which we judged as highly valid. Assuming that studies were published on average 3 years after data collection, we estimated time since event as the difference between year of data collection (year of the earliest publication with the sample minus 3, if not otherwise indicated in the text) and year of event. If the event covered more than 1 year (e.g., Vietnam War), we took the end of the event as the reference year (e.g., 1975) to prevent overestimation (other reference years were 1945 for World War II and 1953 for the Korean War). The interrater agreement, based on codings by both authors of this meta-analysis, was high ($r = 1.00$) for this variable.

Effect sizes were coded in the form of zero-order correlations (r type). For studies in which effect sizes had to be computed from summary statistics (means, standard deviations, frequencies, etc.), we used the Effect Size Determination Program by Daniel B. Wilson (see Lipsey & Wilson, 2001, Appendix C). For effect sizes that were computable for more than one measurement time, we coded the data of the first time, because these measures should have been least influenced by the study design. For studies that provided more than one effect size (because, e.g., two or more hostility measures were used), we averaged the correlations using Fisher's Z_r transformations. In contrast, for effect sizes that were computable for the relation with the dichotomous version of a variable (e.g., PTSD diagnosis) as well as with the continuous version of the same variable (e.g., PTSD symptom severity), we used only the effect sizes based on the continuous version because of higher precision.

All studies were coded by both authors of this meta-analysis. The interrater agreement was high ($\kappa \geq .85$ for categorical variables and $r \geq .97$ for continuous variables). All differing codings were discussed until consensus was reached.

Meta-Analytic Procedure

All computations with effect sizes were made using Fisher's Z_r transformations and using study weights with $\omega = n - 3$ (see Lipsey & Wilson, 2001). For the computations we used SPSS and the SPSS macros written by Daniel B. Wilson (see Lipsey & Wilson, 2001, Appendix D).

We conducted the following preliminary analyses. First, we searched for statistical outliers on effect size variables. Second, we determined whether we could use an effect size measure combining the effect sizes for anger and hostility. Third, we determined whether there was evidence of publication bias—that is, whether studies with nonsignificant findings had a lower probability of being published. To test for publication bias we followed the recommendations of Begg (1994). If publication bias exists, studies resulting in low effect sizes should have a low probability of being published if the sample size is small (because of a low probability of significant findings). In contrast, studies resulting in large effect sizes have a high probability of being published even if the sample size is small (because of a high probability of significant findings). The relationship of sample size and effect size may be visually examined using a funnel graph (cf. Begg, 1994). If the funnel graph does not show a symmetrical shape and if studies with small sample size show a bias toward larger effect sizes, there is evidence for publication bias. In this case, we would have to include dissertations and unpublished reports in the meta-analysis and return to the steps of selection of studies and coding of studies. Comparison of effect sizes for published and unpublished studies could then reveal the magnitude of the publication bias. However, if there is no evidence for publication bias, we can continue with the meta-analysis without consideration of dissertations and unpublished reports.

In the effect size analyses, we used a fixed effects as well as a random effects model. If the results of the fixed effects model show that the homogeneity statistics are not significant, a fixed effects model is appropriate, providing the advantage of a smaller confidence interval for the mean effect size. In this case, we report effect size statistics as determined by a fixed effects model. The mean effect size can then be taken as an estimate for the effect size in the population. However, if the fixed effects model results in significant homogeneity statistics, then the data must be analyzed using a random effects model (Hunter & Schmidt, 2004). In this

case, we report effect size statistics as determined by a random effects model.

In the moderator analyses, we then analyzed the influence of sample characteristics as potential moderators of effect size. In multiple regression analysis, only continuous or dichotomous predictors can be used. Therefore, we transformed the categorical variable type of event into a dichotomous variable contrasting samples with military war experience and samples with other types of events. We decided to focus on this contrast because this distinction is frequently discussed in the literature on anger and PTSD (see Novaco & Chemtob, 1998). Subsequently, in an analysis of variance, we investigate the influence of type of event in more detail using all of the original categories.

Results

Description of Studies

The 39 studies included in the meta-analysis were published between 1985 and 2003, with the median in 2000. Twenty-four studies were conducted in the United States, 9 in Europe, 2 in Israel, 1 in Australia, 1 in Canada, 1 in South Africa, and 1 in Sri Lanka. Sample sizes varied between 20 and 1,698 ($M = 226.1$, $SD = 366.1$, $Mdn = 94.0$). The average mean age of participants was 42.8 years ($SD = 10.6$), and the average proportion of female participants was 30%. Nineteen studies reported on military war experience, 7 on criminal victimization, 5 on civilian war experience, 4 on technological disasters, 2 on health traumas, and 2 on samples with mixed events. The average mean time since event varied between 0.03 and 47.50 years ($M = 12.4$, $SD = 12.2$, $Mdn = 9.0$).

A wide variety of measures were used in the studies. Anger ($k = 29$) was assessed by the STAXI (Spielberger et al., 1999) in 14 studies, by the Multidimensional Anger Inventory (MAI; Siegel, 1986) in 2 studies, and by other measures in 13 studies. Anger out ($k = 8$) was assessed by the STAXI in 6 studies and by the MAI in 2 studies. Anger in ($k = 9$) was assessed by the STAXI in 7 studies and by the MAI in 2 studies. Anger control ($k = 6$) was exclusively assessed by the STAXI. Hostility ($k = 16$) was assessed by the BDHI (Buss & Durkee, 1957) in 4 studies, by the SCL–90 (Derogatis et al., 1973; Derogatis & Savitz, 1999) in 3 studies, by the Cook–Medley Hostility Inventory (Cook & Medley, 1954) in 2 studies, and by other measures in 7 studies. PTSD ($k = 39$) was assessed by assignment of a PTSD diagnosis in 18 studies and by measures of symptom severity in 21 studies: PTSD Symptom Scale (Foa, Riggs, Dancu, & Rothbaum, 1993) in 5 studies; Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane, Caddell, & Taylor, 1988) in 5 studies; Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979) in 3 studies; and a range of other measures in 8 studies.

Preliminary Analyses

First, the data revealed that there were no statistical outliers on effect size variables. We therefore used the complete data set for the subsequent analyses.

Second, the data showed that it was empirically justified to combine the effect sizes for anger and hostility. There were eight studies that provided effect sizes for both anger and hostility. The effect sizes were correlated, with $r = .75$ ($p = .031$). In these studies, the weighted mean effect size was .55 for anger and .58 for hostility; the confidence intervals showed that the difference was not significant. Given these results, we computed an effect size for anger–hostility combined (with $k = 37$) and used this effect size in the analysis of publication bias and in the moderator analyses. The fact that the effect sizes for anger and hostility did not significantly differ does not mean that the distinction between these constructs is not meaningful, however. The result may be traced back to both conceptual links and imprecise measurement of anger and hostility, as discussed in the introduction.

Third, the data revealed strong evidence against a publication bias. A funnel graph showed that studies with small sample size did not show a bias toward larger effect sizes (Figure 1). The distribution of effect sizes exhibits a symmetrical shape typical of nonbiased meta-analytic data sets, which led to the decision not to include dissertations and unpublished reports in our meta-analysis. In addition, we computed the file drawer statistic (Rosenthal, 1979). The results showed that 86 unpublished studies with null effects would be needed to produce a nonsignificant overall effect size for anger–hostility. We judged it to be unlikely that so many unpublished studies on anger, hostility, and PTSD with null effects exist.

Effect Size Analyses

Weighted mean effect sizes were computed for anger, anger out, anger in, anger control, hostility, and anger–hostility. First, we computed effect size statistics for both a fixed effects model and a random effects model. For all effect size variables, the homogeneity statistics were significant, indicating that the variance of effect sizes cannot be attributed only to within-study sampling error, but also to between-study sampling error. Therefore, in the following, we report the effect size statistics based on a random effects model.

For all effect size variables, Table 1 shows the number of studies k, the population size, the weighted mean effect size r, the corresponding confidence interval for the weighted mean effect size, and the homogeneity Q with its level of significance. The mean effect size for anger amounts to .48, the mean effect size for hostility to .53, and the mean effect size for anger–hostility combined to .48. Effect sizes for anger and hostility are based on sufficiently large numbers of studies to yield relatively small

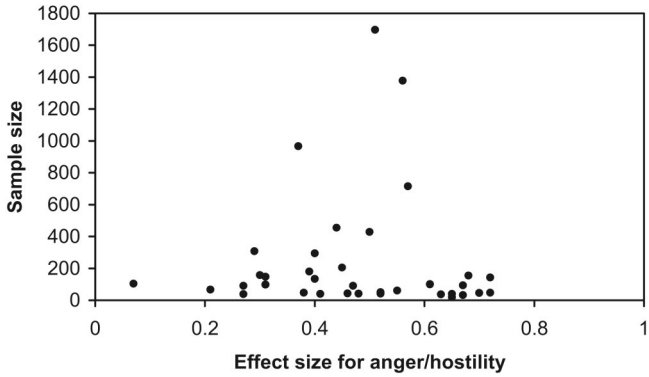

Figure 1. Funnel graph displaying the relation between the effect size (r) for anger–hostility and sample size of the studies.

Table 1
Summary of Effect Sizes

Variable	*k*	Population size	Weighted mean effect size (*r*)	95% CI for weighted mean effect size	Homogeneity (*Q*)
Anger	29	7,486	.48***	.43, .53	173.83***
Anger out	8	600	.29***	.09, .47	42.69***
Anger in	9	676	.53***	.39, .65	41.88***
Anger control	6	406	−.44***	−.19, −.63	35.21***
Hostility	16	1,788	.53***	.47, .59	34.71**
Anger/hostility	37	8,700	.48***	.44, .53	191.41***

Note. *k* = number of studies. Computations were made with a random effects model. CI = confidence interval.
** $p < .01$. *** $p < .001$.

confidence intervals. In contrast, the mean effect sizes for anger out, anger in, and anger control are based on relatively small numbers of studies; in consequence, confidence intervals are substantially larger for these effect size variables. The mean effect size for anger out amounts to .29, the mean effect size for anger in to .53, and the mean effect size for anger control to −.44.

Moderator Analyses

The above analyses revealed heterogeneity of effect size distributions; therefore, it was essential to investigate whether moderator variables would explain variation of effect sizes. We restricted the moderator analyses to the effect size for anger–hostility combined, because this effect size variable was based on the largest number of studies ($k = 37$). Because the number of studies determines the power of the moderator tests, we judged the number of studies to be too small to conduct moderator analyses for the other effect size variables.

First, we tested whether the mean time since event would be a better predictor if the variable was used in its original linear metric or in a logarithmic metric. Therefore, we computed a linear model and logarithmic model for the simple regression of effect size on mean time since event. The linear model was not significant, $F(1, 31) = 0.53$, $p = .47$. In contrast, the logarithmic model was significant, $F(1, 31) = 7.52$, $p = .01$. Therefore, we decided to use mean time since event in its logarithmic metric in the further analyses. Figure 2 illustrates the logarithmic relation between the effect size for anger–hostility and mean time since event; for reasons of illustration, the figure shows the *r* values of effect size, but the computations were made using Fisher's Z_r-transformed values of *r*. The corresponding logarithmic regression equation is $Z_r = 0.494 + 0.046 \times \ln(\text{time})$.

We next computed simple correlations for the effect size for anger–hostility and sample characteristics (Table 2). The results show that type of event and mean time since event were substantially correlated with effect size. The mean age of participants was virtually uncorrelated with effect size. The proportion of female participants in the sample correlated with effect size at a medium height, though not significantly so, due to low statistical power. To control for potential multicollinearity of the predictors, we computed a multiple regression analysis with sample characteristics as predictors of effect size for anger–hostility (Table 2). Overall, the variance explained by sample characteristics amounts to $R^2 = .38$. The results show that both type of event and mean time since event are important predictors, with regression coefficients of .42 and .47, respectively, even if the regression coefficient of type of event is not statistically significant due to low statistical power. The predictors mean age of participants and proportion of female participants did not yield regression coefficients with substantial weight.

Finally, because type of event was used only as a dichotomous variable in the preceding analyses, we computed an analysis of variance to investigate the influence of type of event on effect size for anger–hostility in more detail (Table 3). The results corroborate the importance of type of event as a predictor of effect size, as the mean effect sizes substantially differ between the categories.

Discussion

The results of this meta-analysis show that anger and hostility are substantially associated with PTSD among trauma-exposed adults. Overall, 39 studies were included in the meta-analysis. Effect sizes did not significantly differ for anger and hostility, and we therefore determined the mean effect size on an aggregate level, combining the results for anger and hostility. The weighted

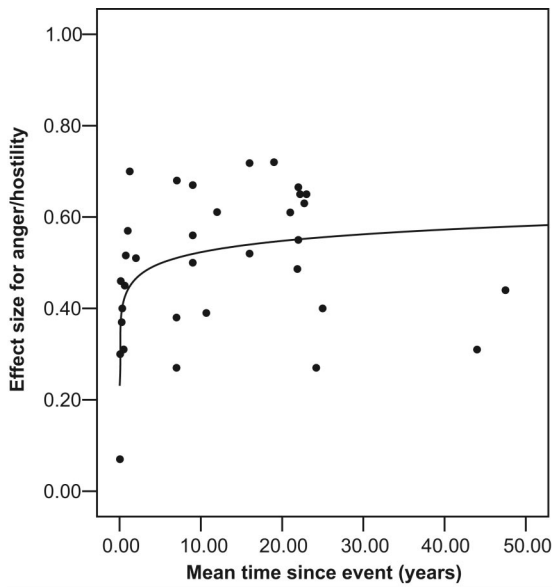

Figure 2. Scatter plot displaying the logarithmic relation between the effect size (*r*) for anger–hostility and mean time since event.

Table 2
Correlations and Standardized Regression Coefficients for Sample Characteristics Predicting Effect Size for Anger/Hostility (k = 32)

Predictor	*r*	β
Mean age	.07	−.13
Proportion of female participants	−.34	.14
Type of event[a]	.48**	.42
Mean time since event (log)	.49**	.47*

Note. Computations for the multiple regression analysis were made with a random effects model (full-information maximum likelihood). Homogeneity $Q_{model} = 21.17$ ($df = 4$, $p = .000$); homogeneity $Q_{residual} = 34.01$ ($df = 27$, $p = .166$). $R^2 = .38$. k = number of studies; log = logarithmically transformed variable.
[a] 1 = military war experience, 0 = other.
* $p < .05$. ** $p < .01$.

mean effect size for anger–hostility combined was .48, indicating a large effect (Cohen, 1992). The studies included in the analysis differed substantially with respect to sample characteristics such as type of traumatic event, time since event, mean age of participants, proportion of female participants, and country of origin. Moreover, the studies used a variety of measures to assess anger, hostility, and PTSD. The heterogeneity of sample characteristics and methodological characteristics strengthens the generalizability of the results.

The weighted mean effect sizes for the relation between anger expression variables and PTSD were .29 for anger out, .53 for anger in, and −.44 for anger control. Thus, the correlation between anger out and PTSD corresponds to a medium effect and is considerably lower than the correlation between anger in and PTSD, which corresponds to a large effect (Cohen, 1992). Anger in measures the frequency of inhibition and nondisclosure of anger; an inspection of the STAXI (Spielberger et al., 1999) and MAI (Siegel, 1986) items suggests that the anger-in scales also measure anger rumination. A ruminative style of emotion regulation might be closely linked to the reexperiencing symptom cluster in PTSD (e.g., Ehlers et al., 1998), which might explain the strong correlation between anger in and PTSD. In contrast, an expressive style of emotion regulation, as assessed in measures of anger out, is not among the characteristic posttraumatic stress reactions, explaining the lower correlation between anger out and PTSD. Finally, the correlation between anger control and PTSD corresponds to a large negative effect. It is possible that strong posttraumatic stress reactions reduce the ability of an individual to control anger by decreasing mental resources.

The analyses showed that the distribution of effect sizes was not homogeneous; therefore, we conducted moderator analyses of the effect size for anger–hostility, which revealed that mean time since event and type of event are sample characteristics that explain substantial proportions of variance in effect size (βs = .47 and .42, respectively). In contrast, the mean age of participants and proportion of female participants did not significantly contribute to the prediction of effect size in the multiple regression analysis.

Time since the event was, as expected, a significant predictor in a logarithmically transformed metric but not in a linear metric. Thus, the relation between time since event and effect size should be modeled as follows: Immediately after a traumatic event, the strength of association between anger and PTSD and between hostility and PTSD is low. In the first months, the strength of association increases strongly. Thereafter, the slope of the curve continuously decreases so that the curve begins to converge to a limiting value. Of course a logarithmic curve does not converge to a limiting value; however, with increasing time since event the slope of the curve decreases to a value that is, with respect to the purpose of the model, close enough to zero.

Type of event proved to be a second moderator of the association. In samples with military war experience the effect size was higher ($r = .56$) than in samples with other types of event. We determined the lowest effect size for victims of criminal victimization ($r = .30$). For all other types of event, the effect sizes ranged between .43 and .48. But does the high effect size for samples with military war experience really reflect a higher impact of military traumatic events on the relation between anger, hostility, and PTSD? Or are there methodological factors that might explain the finding? One factor might be that individuals with military war experience are preselected by the military administration. Presumably, individuals selected for military service differ from the general population on relevant traits—for example, they might have higher assertiveness, aggressiveness, and trait anger. We can only speculate whether higher pre-event trait anger leads to a stronger postevent association between anger and PTSD, because the psychological processes accounting for the association are still disputable. However, it is possible that it is not the type of event that causes the stronger association but instead the pre-event

Table 3
Analysis of Variance of Effect Size for Anger/Hostility by Event Type (k = 37)

Event type	*k*	Population size	Weighted mean effect size (*r*)	95% CI for weighted mean effect size	Homogeneity (*Q*)
Military war experience	19	5,367	.56***	.51, .60	17.57
Criminal victimization	6	575	.30***	.17, .41	8.56
Civilian war experience	5	789	.43***	.31, .54	6.32
Technological disaster	3	1,459	.48***	.36, .59	6.50*
Health trauma	2	230	.44***	.25, .60	0.47
Mixed	2	398	.37***	.19, .52	1.07

Note. Computations were made with a random effects model (full-information maximum likelihood). Homogeneity $Q_{between} = 21.31$ ($df = 5$, $p = .001$); homogeneity $Q_{within} = 40.50$ ($df = 31$, $p = .118$). k = number of studies; CI = confidence interval.
* $p < .05$. *** $p < .001$.

selection accounts for the larger effect size in samples with military war experience. Thus, the meta-analysis does not supply evidence for a causal influence of this moderating factor. Moreover, we would like to emphasize that the results of the meta-analysis show that anger and hostility are substantially related to PTSD among samples who have experienced all possible types of traumatic events, not only in individuals with combat-related PTSD.

A first crucial goal in future research on anger, hostility, and PTSD is to investigate the causality of the relationship. Is anger and hostility a causal factor maintaining PTSD? Or is increased anger and hostility a causal effect of strong posttraumatic stress reactions? To study the causality of the relation between anger, hostility, and PTSD, longitudinal data sets with multiple repeated measures are necessary. A recent study suggests that PTSD has a causal effect on anger but that anger has no causal effect on PTSD (Orth et al., 2006). Further studies should investigate the causal processes using other samples and other measures of anger, hostility, and PTSD.

A second crucial goal in future research should be to conduct more rigorous tests of theories that might explain the relation of anger and PTSD. In these tests, potential mediator variables (e.g., fear avoidance motivation) should be investigated so that the fit of alternative plausible models can be compared. Moreover, the symptom clusters of PTSD (reexperiencing, avoidance, hyperarousal) should be included, so that differing mediator models can be tested: For example, the hyperarousal cluster of PTSD might be a possible causal factor of the relationship, linking PTSD and anger by the physiological pathway. Indeed, the hyperarousal cluster has recently been shown to have a substantial and mainly unidirectional causal effect on the other PTSD symptom clusters (Schell, Marshall, & Jaycox, 2004). In addition, anger variables should be assessed with more precision. It might be crucial whether the relation between anger and PTSD can be traced back to increased trauma-related anger (e.g., anger against the perpetrator or against individuals who did not prevent the occurrence of the traumatic event) or to increased non-trauma-related anger (e.g., anger at the workplace or in the family). Finally, future studies should test further potential moderator variables of the relation between anger and PTSD (e.g., social support).

Knowledge about causality and the psychological processes that account for the association may help to design more efficient treatment strategies of PTSD and more efficient anger management interventions. Anger-specific treatment is necessary not only if anger has a causal effect on PTSD; effective treatment and prevention of chronic anger and hostility among traumatized individuals is required because anger and hostility is a severe problem for the individual, reducing subjective well-being and social functioning. Moreover, chronic anger and hostility among traumatized individuals is a severe problem for society, because anger and hostility might drive the cycle of violence, and traumatized individuals might themselves become perpetrators of aggression and violence (e.g., Beckham et al., 1997; Beckham, Moore, & Reynolds, 2000). Fortunately, the necessity of treatment for anger in PTSD has already been recognized, and treatment strategies have already been developed (e.g., Chemtob, Novaco, Hamada, & Gross, 1997).

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