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The Emotional Stroop Task and Posttraumatic Stress Disorder: a Meta-Analysis

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Abstract

Posttraumatic stress disorder (PTSD) is associated with significant impairment and lowered quality of life. The emotional Stroop task (EST) has been one means of elucidating some of the core deficits in PTSD, but this literature has remained inconsistent. We conducted a meta-analysis of EST studies in PTSD populations in order to synthesize this body of research. Twenty-six studies were included with 538 PTSD participants, 254 non-trauma exposed control participants (NTC), and 276 trauma exposed control participants (TC). PTSD-relevant words impaired EST performance more among PTSD groups and TC groups compared to NTC groups. PTSD groups and TC groups did not differ. When examining within-subject effect sizes, PTSD-relevant words and generally threatening words impaired EST performance relative to neutral words among PTSD groups, and only PTSD-relevant words impaired performance among the TC groups. These patterns were not found among the NTC groups. Moderator analyses suggested that these effects were significantly greater in blocked designs compared to randomized designs, towards unmasked compared to masked stimuli, and among samples exposed to assaultive traumas compared to samples exposed to non-assaultive traumas. Theoretical and clinical implications are discussed.

Keywords

PTSD; trauma; Stroop; attentional bias

Traumatic events are common among adults in the general population, with prevalence rates of approximately 50% (Kessler, 2000; Resnick et al. 1993). Despite ubiquitous exposure to traumatic events, posttraumatic stress disorder (PTSD) has an estimated prevalence of about 8% (Kessler et al., 1995, 2005; Kilpatrick et al., 2003). Though specific trauma types in specific populations have been linked with higher rates of PTSD (e.g., approximately 20% of women who have been sexually abused meet criteria for PTSD; Kessler et al., 1995; Resnick et al., 1993), even the highest prevalence estimates of PTSD represent a minority of

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the number of individuals exposed to traumatic events. Thus, mere exposure to a traumatic event is an insufficient explanation of PTSD (Rosen & Lilienfeld, 2008; Yehuda & LeDoux, 2007), which highlights the need for a deeper understanding of the mechanisms mediating PTSD in order to foster a better explanation of why PTSD develops and maintains. In a related vein, a better understanding of the mechanisms mediating PTSD may inform novel treatment techniques that may map more directly onto the psychopathology of PTSD and thus improve treatment efficacy.

The elucidation of key mechanisms in PTSD is accordingly important for the understanding of how PTSD develops and maintains, the identification of individuals likely to develop PTSD, and the treatment of individuals who have developed PTSD. One means of elucidating core deficits in PTSD has been through examination of cognitive processes and information processing biases (Brewin et al., 2007; Buckley et al., 2000; McNally et al., 1990). One of the most common tasks used in this line of research is the emotional Stroop task. The current paper presents a meta-analytic synthesis of the emotional Stroop effect in PTSD towards the larger goal of elucidating core dysfunctional processes mediating PTSD..

The Emotional Stroop task

The classic Stroop task (Stroop, 1935) displays color words (e.g., blue) in varying colors of ink (e.g., red). The participant is asked to ignore the semantic content of the word and name the color in which the word is displayed. The critical trials are when the color is incongruent with the semantic word (e.g., the word ‘blue’ displayed in green ink). This manipulation induces conflict between task-relevant stimuli (the color) and distracting stimuli (the semantic content). The Stroop task can also be manipulated to examine how emotional stimuli disrupt color-naming performance. The emotional Stroop task (EST) is similar to the original task, but the semantic content of the word is manipulated: the word is neutral in valence (e.g., ‘pencil’) on some trials, whereas the word is threatening in valence (e.g., ‘attack’) on other trials. Thus, the EST can detect the influence of emotional processes on the traditionally cognitively-based task by manipulating the emotional content of the to-be-ignored word and testing for any changes in performance. Numerous studies demonstrate that anxious individuals, including individuals with PTSD (Buckley et al., 2000; Foa et al., 1991; McNally, 1990), have longer color naming times for emotional words compared to neutral words (Bar-Haim et al., 2007; Williams et al., 1996).

The extent to which data from emotional Stroop studies inform a better understanding of the mechanisms underlying PTSD is dependent on the extent to which the mechanisms underlying the emotional Stroop task are understood. Thus, a discussion of the mechanisms mediating the classic and emotional Stroop task is necessary prior to discussing findings among individuals with PTSD specifically. Previous models explaining the mechanisms mediating the EST are not entirely consistent (e.g., Algom et al., 2004; McKenna & Sharma, 2004; Williams et al., 1996; Wyble et al., 2008), but the models can likely be integrated to suggest two key complementary mechanisms that govern performance in the EST. First, a heightened threat detection mechanism1 that causes a greater ‘bottom-up’ disrupting influence in response to emotionally salient stimuli would explain the heightened EST effect in randomized designs (i.e., where neutral and threatening trials are intermixed randomly) found only among high-trait anxious individuals (cf., Bar-Haim et al., 2007). This threat

[1]Some researchers link enhanced threat detection with heightened fear responding (e.g., Ohman, 2005; Ohman & Mineka, 2001). Indeed, in the present manuscript, these two phrases could be used almost interchangeably. However, we chose the term enhanced threat detection because 1) it is more consistent with the broader attentional bias towards threat literature (Bar-Haim et al., 2007; Cisler & Koster, 2010), 2) it specifies a more specific mechanism relative to broadly specifying an emotional response, and 3) it emphasizes that cognitive and emotional processes interact (Pessoa, 2008), which is not as clearly emphasized by referring to this process as heightened fear responding/reactivity.

detection mechanism is consistent with Williams and colleagues' (1996) and Wyble and colleagues' (2008) suggestion of heightened output tendencies associated with emotional stimuli among anxious individuals. It is also consistent with other lines of research suggesting a threat detection mechanism responsible for rapidly detecting potential sources of threat (Davis & Whalen, 2001; Ohman, 2005; Ohman & Mineka, 2001; Ohman & Soares, 1993; Whalen, 1998; 2004), and that this threat detection mechanism is heightened among anxious individuals (Bishop, Duncan, & Lawrence, 2004; Etkin & Wager, 2007). Second, an attentional control mechanism responsible for orienting, maintaining, and shifting attention (cf. Posner, 1980; 1990) may modulate the degree to which emotional stimuli withdraw attention from task demands (Derryberry & Reed, 2002; Eysenck et al., 2007; Reinholdt-Dunne et al., 2009; Wyble et al., 2008). Disruption of this mechanism would explain the delayed EST effect (i.e., impaired performance on the neutral trial *after* the threat word) found by McKenna and Sharma (2004) as well as the heightened EST effect in blocked design (i.e., where neutral trials all occur together, then threatening trials all occur together) studies (Bar-Haim et al., 2007; Phaf & Kan, 2008). Consistent with this view, recent research suggests that heightened anxiety is associated with poor recruitment of neural networks involved in attentional control, including the anterior cingulated cortex (ACC) and dorsal lateral prefrontal cortex (dlPFC) (Bishop et al., 2004; Bishop, 2008; 2009).

In tandem, these two mechanisms are consistent with each of the previous accounts of the EST effect (Algom et al., 2004; Williams et al., 1996; Wyble et al., 2008) and explain 1) the distracting influence of emotional stimuli, 2) the heightened EST effect in randomized Stroop formats among high anxious individuals, 3) the heightened EST effect in blocked designs found among all individuals, and 4) the exaggerated delayed EST effect found among high anxious individuals. The hypothesis of complementary threat detection and attentional control mechanisms is consistent with emerging research in the broader attentional bias towards threat literature (Bishop, 2007, 2008; Cisler & Koster, 2010; Pessoa, 2009).

PTSD and the Emotional Stroop Task

Despite the wealth of research conducted on the EST effect in PTSD over the past 20 years, there remain two gaps in the literature. First, whether an EST effect exists at all in PTSD has recently been questioned (Kimble et al., 2009) due to the inconsistencies in demonstrating the general effect (i.e., slowed RTs for disorder-relevant words relative to neutral words; e.g., Devineni et al., 2004). Kimble and colleagues (2009) argued that dissertations tend to produce less reliable EST effects in PTSD, which introduces a publication bias. When accounting for the possible publication bias, they concluded from their qualitative review: "our data suggest that the [EST effect] is not a reliable finding in persons with PTSD" (Kimble et al., 2009, pg. 653) and that "the [EST effect] in PTSD seems to be an issue that warrants reconsideration" (pg. 654). Given that this conclusion differs from prior qualitative reviews (Buckley et al., 2000; McNally, 1998), it seems that a quantitative review is necessary. Indeed, given that the EST effect in PTSD has been used to inform both cognitive (e.g., McNally, 2006) and neural (Rauch et al., 2006) models of the core deficits in PTSD, it seems essential to quantitatively clarify whether there is EST impairment in PTSD. Further, an important consideration when interpreting published articles versus dissertations is whether the groups were diagnosed with a structured interview or not, which presumably could influence results. Yet another difference across studies that might contribute to inconsistencies is the type of traumatic event to which participants were exposed. Emerging research suggests that assaultive events (i.e., physical assault, sexual assault) are a more potent risk factor for psychopathology relative to other types (e.g., motor vehicle accidents, natural disasters) (Breslau et al., 1999; Cougle et al., 2009; Resnick et al., 1993). To address these possibilities, the present meta-analysis included both published articles and

dissertations, and also coded method of diagnosis (structured interview versus self-report questionnaire) and trauma type (assaultive versus non-assaultive).

Second, assuming there may be a significant EST effect in PTSD, the extant research has not clearly elucidated whether the mechanism producing the EST in PTSD is a hypoactive attentional control mechanism, hyperactive threat detection mechanism, or both mechanisms operating in tandem. One manner to tease apart these competing possibilities is to examine the various procedural manipulations of the EST that may differentially isolate the candidate mechanisms mediating the EST effect. An enhanced EST effect in PTSD was first identified by McNally and colleagues (1990), who compared Stroop response times towards PTSD-related words, Obsessive-Compulsive Disorder (OCD)-related words, positive words, and neutral words between Vietnam combat veterans with and without PTSD. The results revealed that participants with PTSD had longer response times towards PTSD-related words relative to OCD-related, positive, and neutral words. Participants with PTSD had longer response times towards PTSD words compared to participants without PTSD, and the response times of participants without PTSD did not differ across word types. This initial study is an excellent point of departure from which to evaluate how the EST may inform the mechanisms underlying PTSD.

McNally and colleagues (1990) compared performance between several types of word types: positive, neutral, general threat (i.e., OCD-related words), and disorder-relevant words. Disorder-relevant words may differ from other word categories in either threat intensity or personal relevance. This is an important distinction, such that research has shown that personally-relevant words can also elicit longer RTs in a Stroop task (see Williams et al., 1996). As such, slower RTs toward disorder-relevant words could be due to an enhanced threat-detection mechanism or to mere personal relevance. Generally threatening words, which should be equally relevant to PTSD, trauma-control, and no-trauma control groups and only differ from neutral words in threat intensity, is one means of controlling personal relevance in order to study the EST effect. Another means of addressing the limitation of greater personal relevance of trauma-related words is to use a trauma-exposed control group without PTSD that is matched to the PTSD groups' trauma type (e.g., combat veterans with and without PTSD). This control procedure presumably equates the groups in personal relevance of the trauma-related words; however, it still remains possible that trauma-related words are more personally relevant to individuals with PTSD because of PTSD-related symptoms (e.g., re-experiencing symptoms). Accordingly, comparisons between generally-threatening words and neutral words may be the most stringent test of a threat-detection mechanism. Studies have yielded mixed results regarding an enhanced EST effect towards generally-threatening words among PTSD groups (Buckley et al., 2002; Foa et al., 1991; McNally et al., 1990), though there appears to be a trend suggesting greater latencies towards generally-threatening words. A meta-analysis of existing studies may be a more powerful means of examining whether PTSD individuals display slower RTs towards general threat words compared to neutral words, which would allow for a test of whether PTSD is characterized by an enhanced threat detection mechanism.

McNally and colleagues (1990) also investigated RTs towards positive words and found no difference between groups. This comparison is also important because positive words may have higher emotionality (i.e., arousal) value than neutral words but not threat words, and positive words may only differ from threat words in valence. For example, previous research has found that the arousal value of a stimulus, and not the valence, may be more important in the disruption of attentional processes (Anderson, 2005; Vogt et al., 2008). As such, comparisons between positive words, threat words, and neutral words allow for a test of whether emotionality *per se* slows RTs, or whether *threat* specifically slows RTs. As with

general threat words, meta-analysis may be a more powerful means of ruling out the hypothesis that emotionality slows RTs instead of enhanced threat detection specifically.

McNally and colleagues (1990) used a card format of the Stroop task, in which word types occurred in blocked format printed on a poster. As noted by McNally and colleagues, this blocked format precludes a full assessment of automatic processing, in which case the degree to which the EST effect reflects automatic or strategic processing was unclear. Automatic processing generally refers to processing that is capacity-free and occurs without intent, control, or awareness, whereas strategic processing generally refers to processing that is intentional, controllable, capacity-limited, and dependent on awareness (McNally, 1995; 1996; Moors & de Houwer, 2006; Schiffrin & Schneider, 1977). One means of manipulating stage of processing is to mask the presentation of words in the EST. In masked presentations, the word is displayed briefly (e.g., 17 ms) followed by a mask (e.g., 'XXXX' displayed in orange), which precludes conscious identification of the initial word. Of the two candidate processes underlying the EST effect, it is likely threat detection that is able to operate automatically. For example, neuroimaging studies among non-clinical (Morris et al., 1998; Whalen et al., 1998; 2004) and PTSD samples (Bryant et al. 2008; Felmingham et al., 2010; Hendler et al., 2003; Rauch et al., 2000) have demonstrated that the amygdala responds to masked stimuli (Morris et al., 1998; Whalen et al., 1998; 2004). By contrast, attentional control is an executive function that may necessitate more time and processing resources to fully engage (Pessoa, 2009). Masked presentations may therefore be one means of teasing apart the influence of the threat detection and attentional control mechanisms. Research using masked presentations has yielded mixed results (Buckley et al., 2000; Buckley et al., 2002; Harvey et al., 1996; McNally et al., 1996; Paunovic et al., 2002). Meta-analysis may be a more powerful means of examining automaticity in the EST among individuals with PTSD that may help resolve the inconsistencies.

In a related vein, manipulating blocked versus randomized formats is another means of manipulating automaticity, such that if the effect is more dependent on effortful/controlled processing, then the effect should be larger in blocked compared to randomized formats (cf. McKenna & Sharma, 2004; Phaf & Kan, 2008; see above discussion). Attentional control resources may deplete across repeated presentations of threat during blocked format, which would result in less ability to perform task demands and consequently a greater EST effect compared to randomized format. As such, greater effects in the blocked format may indicate greater disruptions in attentional control (Wyble et al., 2008). By contrast, a significant EST effect in randomized designs would be consistent with the 'fast' EST effect (Wyble et al., 2008) and suggest enhanced threat detection. Direct comparisons of blocked versus randomized presentations in a single study are rare (e.g., Dalgleish, 1995; Kindt et al., 1996). Meta-analysis seems like an appropriate way of testing this effect across studies. It is important to note, however, that blocked presentations may also be viewed as a mood induction procedure which could influence performance; thus, interpretation of blocked versus randomized comparisons are not unambiguous.

In sum, there are two main purposes of the present meta-analysis. Our first goal is to synthesize the EST research in PTSD over the past 20 years and quantitatively examine whether the EST effect exists in PTSD (cf. Kimble et al., 2009). Second, many manipulations of the EST may shed light on the mechanisms underlying PTSD, but studies do not always provide consistent results. Thus, our second goal is to quantitatively synthesize this research paying careful attention to these manipulations in order to test whether PTSD is characterized by a hyperactive threat detection mechanism, a hypoactive attentional control mechanism, or both. Greater effect sizes in blocked versus randomized designs and unmasked versus masked designs would provide support for hypoactive attentional control mechanisms. Significant effect sizes in masked designs, randomized

designs, and towards generally threatening stimuli would provide support for hyperactive threat detection mechanisms.

Methods

Selection of Studies

We identified appropriate studies by conducting searches in the PsychINFO database and searching the reference sections of review articles on this topic (Bar-Haim et al., 2007; Buckley et al., 2000). To address the possible publication bias suggested by Kimble and colleagues (2009), we also included the dissertations pertaining to PTSD and the EST that these authors located as well as additional dissertations identified through searches of the PsychINFO database. We conducted searches using a PTSD term (e.g., 'PTSD' 'posttraumatic stress disorder' 'posttraumatic' 'trauma') and task term (e.g., 'Stroop' 'attention' 'attentional bias'). These searches identified 42 unique (i.e., articles that did not appear in multiple searches) articles for possible inclusion.

Articles were included if they met the following criteria: 1) report original results (i.e., not a literature review; not results from a previously published study), 2) used an EST, 3) included a PTSD group, and 4) reported relevant statistics for calculating effect sizes in the journal article2. Twenty-six articles met all of these criteria and were included in the present study (see Table 1 for description of studies). Articles were excluded for not providing relevant statistics (n = 7), not using an EST or not recording RT data (n = 3), not including a PTSD group (n = 3), using a left versus right fixation manipulation that could confound results (n = 1), and the data from three published articles were identical to data from three dissertations (total articles excluded n = 16).

Diagnosis of PTSD

Seventeen articles used a structured diagnostic inventory (Clinical Administered PTSD Scale, Blake et al., 1995; Anxiety Disorder Interview Schedule, Brown et al., 1994; Structured Clinical Interview for Axis I Disorders, First et al., 1995), three articles used a non-specified clinical interview, two articles used a non-specified clinical interview and a self-report questionnaire, and four articles used only the cut score of a self-report questionnaire.

Coding Relevant Variables

We coded articles for the following EST variables: 1) presentation (masked versus unmasked), 2) design (blocked versus randomized), 3) publication type (journal article versus dissertation), 4) word type (neutral, PTSD-relevant, general threat, positive), 5) assessment procedure (structured interview versus self-report questionnaire), and 6) trauma type of the sample (assaultive trauma versus non-assaultive trauma versus a mixed trauma sample). We also coded a study's PTSD, trauma-control, and no-trauma control N, age, and gender.

Statistical Analysis

Database—A database was created using Comprehensive Meta-analysis Program (CMA) Version 2 (Biostat; Borenstein, Hedges, Higgins & Rothstein, 2005). CMA has been used for the analyses of several published meta-analyses (e.g., van IJzendoorn, Juffer, & Poelhuis, 2005; Cepeda-Benito, Reynoso & Erath, 2004; Prochaska, Delucchi, & Hall, 2004;

[2]Eleven authors were contacted to obtain relevant statistics to compute effect sizes. Of these, four were able to provide the relevant information.

Wolitzky, Horowitz, Powers & Telch, 2008). For each study, means, standard deviations, sample size, and pre-post correlations were entered into the database for the within-group effect analyses. In order to examine whether these effects were significantly different between groups (e.g., difference in effect size between the PTSD group and no trauma control group with regard to differences in reaction time between PTSD-related words and neutral words), the effect sizes obtained through CMA were directly compared (as opposed to only including studies that included both groups and both word types in question, thus allowing for inclusion of all studies).

Effect size calculation—For each study, we computed effect sizes for one or more comparisons of interest. When studies included multiple groups that were suitable for comparison, multiple effect sizes were obtained. For example, for a study comparing Stroop RT between a PTSD group and trauma control group using PTSD-relevant, neutral, and positive words, within-group effect sizes were obtained for the PTSD group and then the trauma control group for "PTSD-relevant v. neutral," "PTSD-relevant v. positive," and "positive v. neutral," thus yielding six total within-group effect sizes. Effect size estimates derived from repeated-measure designs must account for the correlation between measures or the resulting effect size will be inflated (Dunlap, Cortina, Vaslow, & Burke, 1996). The present study used the correlation between emotional and neutral words of .90 reported in the EST psychometric study by Eide and colleagues (2002). Between-group differences were assessed by examining whether group (PTSD versus non-trauma control versus trauma-exposed control) moderated the within-subject comparisons (e.g., does group moderate the difference between PTSD and neutral RTs?).

In order to ensure that the comparisons reported were based on a meaningful number of studies and that fail-safe N analyses could be conducted, comparisons were not made when only one or two studies were available for a particular comparison. In addition, this commonly practiced, conservative approach (e.g., Bar-Haim, Lamy, Pergamin, Bakermans-Kraneburg, & van Ijzendoorn, 2007) also limits the likelihood of a given ES being driven by a study that may be an outlier (McKay, 2008). For all comparisons, Cohen's *d* was selected as the index of effect size, with *d*-values of 0.2, 0.5, and 0.8 representing small, medium, and large effect sizes, respectively. Average effect sizes for each outcome were weighted in each relevant comparison using the inverse variance estimate. Weighting of sample size was done in order to minimize the risk that a small, outlying sample would exert a disproportionate influence over the final effect size for a comparison (Rosenthal, 1991). The use of random-effects models also helped to weight the studies appropriately, and were used to increase the generalizability of findings beyond the studies that were able to be included in the meta-analysis. In addition, in order to account for the bias that may be present by including only published studies (i.e., file drawer effect), a fail-safe N (FSN) analysis was conducted for each comparison. Because meta-analyses are largely limited to published studies, the fail-safe N statistic accounts for the number of non-significant findings (that presumably may not have been published) needed to bring a significant effect size to non-significance. Thus, a large fail-safe N statistic indicates that the significant effect is not likely to be due to the bias of published studies having larger effects than non-published studies.

Given the large number of coded variables (3 groups × 6 word type comparisons × 5 moderators = 90 possible statistical tests), alpha inflation needed to be addressed. To control for alpha inflation resulting from the large number of possible comparisons, we 1) set the alpha level to $p < .01$, 2) controlled for the number of comparisons by only testing for moderation when there was a significant overall effect size, and 3) only made group comparisons based on the initial omnibus word-type comparison effect sizes. Following between-group analyses of the omnibus word-type comparison effect sizes, moderator

analyses were conducted separately within-each group. Higher-order interactions (e.g., group × word type × moderator interactions) were not conducted because of the low number of studies for most of these comparisons and lack of power in detecting significant higher-order interactions. For each within-group comparison, we calculated the statistical significance (*p*-value) of the effect size, the within-comparison heterogeneity index (Q), and the *p*-value for the heterogeneity index. We also calculated the: (a) standard error (SE*d*), (b) variance, and (c) 95% confidence intervals (CI) for the effect sizes. These statistics provide information on the stability, significance, and range of the true effect size.

Moderator Analyses—Moderator analyses were also conducted within the CMA program using the moderator platform (mixed-effects models). Q-values (between-group heterogeneity) are reported with their *p*-values for categorical moderators. Studies were appropriately dummy coded for each putative moderator whenever the relevant data was available. Moderator analyses were conducted for comparisons in which the heterogeneity index was statistically significant. The moderators examined included: (a) Stroop presentation (dichotomized as masked v. all other types); (b) use of blocked design (yes v. no); (c) publication status (published in journal v. dissertation); (d) trauma type (categorized as assault v. non-assault v. mixed samples), (e) group (PTSD versus no trauma control versus trauma-exposed control), and (f) assessment procedure (structured interview versus self-report questionnaire). Trauma type was not examined as a moderating variable for the within-groups effects in the no-trauma control group.

Results

Basic Study Information

The meta-analysis included 26 studies. The studies were published between 1990 and 2007, and included a total of 538 PTSD participants, 254 no trauma control participants, and 276 trauma control participants. Mean age for the PTSD, no trauma control, and trauma control samples were 36.3 (*SD* = 10.8), 28.1 (*SD* = 8.5), and 35.4 (*SD* = 11.5), respectively. The groups were predominantly female, with percentages of 51, 79, and 58, respectively, for PTSD, no trauma control, and trauma control groups. While the rates of women in the samples collectively appeared to be higher for no trauma control groups, among the individual studies there were no differences between groups in gender distributions (i.e., in samples with no trauma controls, the PTSD groups also had higher proportion of women), all *t*'s < .60. Table 1 reports basic study characteristics for all studies included in the meta-analysis. Tables 2 and 3 report all outcome data. Table 4 summarizes the within-group moderator findings.

Differences between PTSD, Trauma-Exposed Control, and No Trauma Control Groups in Overall Word Type Comparisons

Summaries of the relevant statistics are displayed in Tables 2 and 3.

General threat v. neutral words—The effect sizes for this comparison did not differ between the PTSD (*d* = .28, *p* < .01), trauma control (*d* = .17, n.s.), or no trauma control (*d* = .14, n.s.) groups; Q(2) = 3.09, n.s.

General threat v. positive words—The effect sizes for this comparison did not differ between the PTSD (*d* = .13, *p* < .01), trauma control (*d* = .15, n.s.), or no trauma control (*d* = .01, n.s.) groups; Q(2) = 2.73, n.s.

Positive v. neutral words—The effect sizes for this comparison did not differ between the PTSD (d = .07, p < .01), trauma control (d = .02, n.s.), or no trauma control (d = −.03, n.s.) groups; Q(2) = 2.29, n.s.

PTSD-relevant v. general threat words—The effect sizes for this comparison did not differ between the PTSD (d = .29, p < .01), trauma control (d = .22, p < .01), or no trauma control (d = .11, n.s.) groups; Q(2) = 2.93, n.s.

PTSD-relevant v. neutral words—The effect sizes for this comparison significantly differed between the three groups, Q(2) = 9.08, p < .01. The PTSD (d = .39, p < .01) group demonstrated significantly greater effect sizes compared to the no trauma control group (d = .14, n.s.); Q(1) = 9.1, p < .01. The trauma control group (d = .24, p < .01) also demonstrated significantly greater effect sizes compared to the no trauma control group; Q(1) = 4.08, p < .01. There was no difference between the PTSD and trauma control groups; Q(1) = .92, n.s.

PTSD-relevant v. positive words—The effect sizes for this comparison significantly differed between the three groups, Q(2) = 9.18, p < .01. The PTSD (d = .29, p < .01) group demonstrated significantly greater effect sizes compared to the no trauma control group (d = .04, n.s.); Q(1) = 8.7, p < .01. The trauma control group (d = .22, p < .01) did not differ from the no trauma control group; Q(1) = 1.15, n.s. There was no difference between the PTSD and trauma control groups; Q(1) = 2.85, n.s.

Within-group Effects and Moderator findings: PTSD Group

Summaries of these effects are displayed in Tables 2 and 4.

General threat v. neutral words—Nineteen outcomes were included in this comparison. A small but statistically significant effect was observed, d = 0.28, p < .001, favoring general threat words. The FSN analysis indicated that the findings were robust with regard to publication bias (FSN= 600). The heterogeneity index was statistically significant, Q(18) = 71.21, p < .001, suggesting that the studies varied with respect to the magnitude of this effect. None of the candidate moderators were significant.

General threat v. positive words—Eleven outcomes were included in this comparison, yielding a small but statistically significant effect size favoring general threat words d = .13, p > .01. FSN analysis indicated that the effect was *not* robust with regard to publication bias (FSN = 58). The heterogeneity index was statistically significant, Q(10) = 25.15, p < .05. However, none of the putative moderators significantly impacted the difference between reaction times for general threat and positive words in the PTSD group.

Positive v. neutral words—Nineteen outcomes were included in this comparison, yielding a very small and non-significant effect size favoring positive words, d = .07, p > .05. The heterogeneity index was statistically significant, Q(18) = 35.92, p < .05. Given the null overall effect, moderator analyses were not conducted.

PTSD-relevant words v. general threat words—Eighteen outcomes were included in this comparison. A small, statistically significant effect size favoring PTSD-relevant words was observed, d = .29, p < .001. FSN analysis indicated that the effect was robust (FSN = 588). The heterogeneity index was statistically significant, Q(17) = 83.89, p < .001. Assault-exposed (d = .34) and mixed trauma (d = .38) samples had larger effects sizes relative to non-assault trauma-exposed samples (d = −.04). Task design moderated the findings, with blocked designs (d = .40) demonstrating greater effects than mixed designs (d = .21).

PTSD-relevant words v. neutral words—Thirty-two outcomes were included in this comparison, yielding a moderate effect favoring PTSD words, $d = .39$, $p < .001$. This finding was robust with regard to publication bias (FSN = 2628) and the heterogeneity index was statistically significant, Q(31) = 217.40, $p < .001$. Studies with blocked designs had significantly larger effects than studies with non-blocked designs ($d = .57$ v. $d = .26$), Q (1) = 8.64, $p < .01$. Trauma type moderated the effects, Q (2) = 7.53, $p < .05$, with larger effects observed for assault samples ($d = .48$) compared to non-assault ($d = .23$) and mixed trauma ($d = .34$) samples. Studies using unmasked Stroop tasks showed larger differences in reaction time between word types compared to masked tasks ($d = .44$ v. $d = .09$), Q (1) = 12.91, $p < .001$.

PTSD-relevant v. positive words—Nineteen outcomes were included in this comparison and yielded a small effect size favoring PTSD-relevant words, $d = .29$, $p < .001$. This finding was robust with regard to publication bias (FSN = 611) and the heterogeneity index was statistically significant, Q(18) = 90.74, $p < .001$, suggesting the studies in this comparison significantly varied with respect to the magnitude of the effects. Moderator analyses revealed that studies using blocked designs had larger effects than those using non-blocked designs ($d = .44$ v. $d = .24$), Q (1) = 4.2, $p < .05$. In addition, the magnitude of the difference in reaction time between PTSD-relevant words and positive words was significantly impacted by the type of Stroop task (unmasked v. masked), with significantly larger effects for studies using unmasked paradigms ($d = .33$) compared to masked ($d = -.03$), Q (1) = 18.83, $p < .001$.

Within-group effects and moderator findings: Trauma control group

General threat v. neutral words—Eleven outcomes were included in this comparison. A small and non-significant effect size was observed favoring general threat words, $d = .17$, $p > .01$. This effect was significantly heterogeneous, Q(10) = 48.80, $p < .001$, but given the null overall effect moderator analyses were not conducted.

General threat v. positive words—Seven outcomes were included in this comparison. The effect size was small and statistically significant, $d = .15$, $p < .01$. The effect was not, however, robust (FSN = 29). The heterogeneity index was not statistically significant, Q (6) = 11.23, $p = .09$.

Positive v. neutral words—Twelve outcomes were included in this comparison. The effect size was small and not statistically significant, $d = .02$, $p > .01$. The heterogeneity index was statistically significant, Q (11) = 31.42, $p < .001$, but given the null overall effect moderator analyses were not conducted.

PTSD-relevant words v. general threat words—Eleven outcomes were included in this comparison and yielded a small, statistically significant effect favoring PTSD-relevant words, $d = .22$, $p = .002$. The heterogeneity index was significant, Q(10) = 55.05, $p < .001$ and the effect was robust (FSN = 136). Only trauma type moderated the effect, Q(1) = 3.87, $p < .05$, with assaultive trauma-exposed samples ($d = .28$) demonstrating a greater effect than non-assault trauma-exposed samples ($d = -.01$).

PTSD-relevant v. neutral words—Eighteen outcomes were included in the comparison. A small, statistically significant effect was observed favoring PTSD-relevant words, $d = .24$, $p < .001$. The heterogeneity index was significant, Q(17) = 117.55, $p < .001$ and the effect was robust (FSN = 333). Studies using blocked designs showed significantly greater differences in reaction time between PTSD-relevant and neutral words in the trauma control

group (i.e., larger effects) than those using non-blocked designs (d = .39 v. d = .11), Q (1) = 3.87, p < .05.

PTSD-relevant words v. positive words—Twelve outcomes were included in this comparison, yielding a small, statistically significant effect size favoring PTSD-relevant words, d = .22, p < .001. The heterogeneity index was significant, Q(11) = 39.18, p < .001 and the effect was robust (FSN = 134), but no putative moderators were significant.

Within-group effects and moderator findings: No trauma control group

General threat words v. neutral words—Six outcomes were included in this comparison. The heterogeneity index was statistically significant, Q (5) = 19.13, p < 01, but because the overall effect was non-significant, d = .14, p > .05, moderator analyses were not conducted.

General versus positive words—Five outcomes were included in this comparison and produced a small, non-significant effect size, d = .01, p > .05. The heterogeneity index was statistically significant, Q (4) = 15.77, p < .01. As there is a non-significant effect, no moderator analyses were conducted.

Positive words v. neutral words—Ten outcomes were included in this comparison, yielding a non-significant effect size of d = −.01. The heterogeneity index was not statistically significant, Q (9) = 5.25, p = .81.

PTSD-relevant words v. general threat words—*S*even outcomes were included for this comparison, yielding a non-significant effect size of d = .11. The heterogeneity index was significant, Q (6) = 29.64, p < .001. However, none of the putative moderators significantly impacted the effect.

PTSD-relevant v. neutral words—Fifteen outcomes were included in this comparison, yielding a small and statistically non-significant effect size favoring PTSD-relevant words, d = .14, p > .01. This finding was not robust to publication bias, FSN = 55. The heterogeneity index was statistically significant, Q(14) = 76.07, p < .001.

PTSD-relevant v. positive words—Ten outcomes were included in this comparison. No significant effect was observed, d = .04, p > .01. The heterogeneity index was statistically significant, Q (9) = 33.50, p < .001.

Discussion

PTSD is associated with low quality of life (Olatunji, Cisler, & Tolin, 2007) and significant psychosocial impairment (Amaya-Jackson et al., 1999; Breslau, 2001; Kessler, 2000). Research has used the EST as one means of elucidating some of the core deficits associated with PTSD (Buckley et al., 2000; McNally et al., 1990). Whether an EST effect exists at all in PTSD has been questioned (Kimble et al., 2009), necessitating a quantitative review to examine the EST effect in PTSD. Further, two complimentary mechanisms are theorized to mediate performance on the EST, a threat detection mechanism and attentional control mechanism, but key EST manipulations that facilitate insights into the mechanisms mediating PTSD have yielded inconsistent results. The present study synthesized this body of literature via meta-analysis as a means of better understanding the core deficits in PTSD.

In regards to the first goal, our quantitative results suggest significantly impaired EST performance in the PTSD group towards PTSD-relevant and generally threatening stimuli,

and the EST impairment in the PTSD group is significantly greater than the NTC group. The TC group demonstrated impaired performance towards only PTSD-relevant words, and this impairment was also significantly greater compared to the NTC group. The PTSD and TC groups did not significantly differ in the direct comparisons, though effect sizes consistently favored the PTSD group. On the one hand, the results suggesting that groups only differed in comparisons involving PTSD-relevant words when compared to the NTC group could be interpreted to suggest a lack of an EST effect. That is, trauma-related words are personally relevant to the trauma exposed groups (both TC and PTSD groups) and not the NTC group, so the finding that between group differences are only found towards these words may reflect either an artifact (i.e., personal relevance) or the more general negative consequences of trauma exposure per se (i.e., not PTSD specifically). On the other hand, if it were the case that the observed effects were an artifact of personal relevance, then there should not have been a significant difference between generally threatening words and neutral words (and positive words) found within the PTSD and TC groups. While these effect sizes did not differ significantly between the groups, if it were the case that the observed effects were only due to personal relevance, then there should not have been effects towards non-personally relevant words found only among the clinical groups. Accordingly, the negative consequence of trauma exposure is the most likely interpretation of the present results.

On the one hand, the pattern of results suggesting the negative consequences of trauma exposure generally, and not PTSD specifically, may contradict the hypothesis of an enhanced EST effect in PTSD. That is, if it were the case that PTSD specifically is associated with a pronounced EST effect, then it would be expected that the effects should be greater in PTSD relative to trauma control groups. On the other hand, it is important to consider why comparisons to a trauma control group are important. One factor this comparison controls is personal relevance of the PTSD-words (though not in studies using mixed trauma samples). If personal relevance is held constant between the groups, then differences in performance disruption are likely due to other factors (e.g., attentional control, threat detection). From this perspective, the current results are not consistent with hypotheses of disrupted attentional control or threat detection mechanisms in PTSD. The more obvious factor that a trauma control group comparison controls is trauma exposure, which presumably can address issues of specificity. For example, is psychopathology following trauma exposure uniquely associated with impaired EST performance? From this perspective, the results are again not consistent with the hypothesis that PTSD is uniquely associated with impaired EST performance. However, it is interesting to note that the trauma control group, but not the no trauma control group, had similar within-subject evidence for impaired EST performance. The pattern of within-subject effects and between-group differences suggests that individuals exposed to traumatic events, regardless of PTSD, display impaired EST performance. These findings raise the possibility that the EST effect in PTSD does not necessarily reflect specific characteristics of PTSD; rather, it reflects the consequences of exposure to traumatic events. In a related vein, a growing body of research suggests that assaultive traumas (e.g., combat, physical assault, sexual assault) are a more potent risk factor for PTSD relative to other traumatic event types (e.g., motor vehicle accidents, natural disasters) (Cougle et al., 2009; Resnick et al., 1993). Consistent with the view of impaired EST performance resulting from trauma exposure per se, the present results found that trauma type moderated some of the EST effects in both PTSD and TC groups, such that samples exposed to assault had greater effect sizes relative to other trauma types.

There is a growing body of evidence indicating the negative consequences of traumatic event exposure on both physical health (Sledjeski, Speisman, & Dierker, 2008) and mental health outcomes (Breslau et al., 1991; Kilpatrick et al., 2003; Resnick et al., 1993). Relevant to the present topic, emerging research suggests that traumatic event exposure has a

cumulative ‘allostatic’ (McEwen, 2004) effect (Kollassa, Kolassa, Ertl, Papassotiropoulos, & De Quervain, 2010; Neuner et al., 2004; Steel et al., 2009). One study conducted among a sample of over 3000 refugees found that the rate of PTSD was about 23% among individuals exposed to 1–3 traumatic events, and this rate increased up to 100% among individuals exposed to 28 or more traumatic events (Neuner et al., 2004). Neurobiological research is clarifying the mechanisms by which this allostatic effect may occur (Arnsten, 2009; Izquierdo et al., 2006; Liston et al., 2006; Mitra et al., 2005; Radley et al., 2006; Vyas et al., 2002; 2004). This research suggests that chronic stress increases dendritic branching in the amygdala and increases reactivity towards motivationally salient cues (e.g., threat cues), and also decreases dendritic spine density and length in key structures of the prefrontal cortex necessary for cognitive, emotional, and behavioral control. Thus, emotional reactivity increases while the ability to regulate emotion simultaneously decreases. Behavioral genetic studies have demonstrated that genes affecting dopamine and serotonin transmission moderate the dose-response relationship between cumulative traumatic event exposure and PTSD vulnerability (Grabe et al., 2009; Kolassa, Kolassa et al., 2010; Kolassa et al., in press; Xie et al., 2009), and these same genes also affect emotion processing (Hariri et al., 2002; 2005), cognitive flexibility (Krugel, Biele, Mohr, Li, & Heekeren, 2009), and cognitive control of attention to threat (Beevers et al., 2007; 2009; Bishop, Cohen, Fosella, Casey, & Farah, 2006). This body of research implicates the key roles that cumulative traumatic event exposure and its neurobiological consequences play in the development of PTSD. From this perspective, it would be expected that individuals exposed to traumatic events, regardless of PTSD, would display disrupted attention towards threat. Further, a dose-response relationship between traumatic event exposure frequency and disrupted attentional control of threat would be expected. There is only limited evidence testing a relationship between trauma exposure frequency/severity and EST performance (McNally et al., 1990). Future research along these lines will help clarify the source of impaired EST performance in PTSD, which will help clarify the dysfunctional processes characterizing PTSD.

If this interpretation were valid and these results do suggest that traumatic event exposure, regardless of PTSD, impairs attentional control and enhances threat processing, then the results are also inconsistent with the view that these processes are specifically linked with psychopathology. Instead, it may be the case that these processes are risk factors that confer vulnerability for psychopathology, or that they are correlated with some other processes that confer vulnerability for psychopathology. This may be consistent with research showing that 1) genes affecting serotonin transmission confer vulnerability to psychopathology following stressful events (Caspi et al., 2003; Grabe et al., 2009; Xie et al., 2009), 2) these same genes modulate difficulty disengaging attention from threat among samples screened to be free from psychopathology (Beevers et al., 2009), and 3) impaired performance in the EST has been found to predict distress to subsequent stressors (MacLeod & Hagan, 1992). The fact that attentional biases towards threat are found in vulnerable, but psychopathology free, individuals is consistent with the view of these processes as risk factors for psychopathology instead of the view of these processes as markers of current psychopathology. One means of testing this conceptualization as it relates to PTSD specifically would be to measure prospectively EST performance, exposure to traumatic events, and symptoms of PTSD, which would allow for delineation of the temporal relationships between these processes.

In regards to the second purpose of the meta-analysis, the overall pattern of results provide support for hypoactive attentional control mechanisms in PTSD, but only weak support for a hyperactive threat detection mechanism in PTSD. When examining moderators of within-subjects effects, two key observations provide support for deficits in attentional control: 1) threat words only impaired performance in unmasked, but not masked, Stroop presentations, and 2) threat words impaired performance in blocked designs significantly more so than in

randomized designs. The former effect demonstrates that performance impairment is only observed when some degree of elaborate and/or effortful processing is possible, which is consistent with the hypothesis of impaired attentional control. The latter effect demonstrates that the 'slow' (i.e., inter-trial) EST effect, which is dependent on attentional control (McKenna & Sharma, 2004; Wyble et al., 2008), is particularly pronounced among PTSD groups. Again, however, the larger effect in blocked designs may result from mood induction stemming from repeated presentations of threat words; thus, conclusions based solely on the block design manipulation must be tempered.

There were two key pieces of evidence for a hyperactive threat detection mechanism when examining within-subject comparisons. First, both PTSD-relevant and generally threatening words, but not positive words, significantly slowed RTs relative to neutral words among PTSD groups. Given that PTSD-relevant words may be more personally relevant for PTSD groups, performance disruptions from these words may not necessarily reflect threat processing. The finding that generally threatening words, but not positive words, also impair performance is consistent with the view that individuals with PTSD are more reactive to threat stimuli and not emotionality per se. Second, the EST effect size for PTSD-relevant and generally threatening words remained in the small range in randomized designs. As discussed above, randomized designs should reflect the 'immediate' (i.e., intra-trial) EST effect, hypothesized to be more dependent on emotional reactivity and less so on attentional control (Wyble and colleagues, 2008); thus, meaningful effect sizes in these designs provides some support for enhanced threat detection. However, the key prediction from the hypothesis of enhanced threat detection was not supported: EST effect sizes in masked Stroop designs were generally of a trivial magnitude. Given that masking is a procedure commonly used to assess enhanced threat detection (Ohman & Soares, 1993; 1994), the present failure to find strong evidence of an EST effect towards masked words notably tempers conclusions about enhanced threat detection in PTSD.

The only evidence from between-group comparisons for enhanced threat detection in PTSD is the fact that PTSD groups (and TC groups) differed from the NTC groups in attention towards threat above and beyond attention towards neutral stimuli. The fact that threat disrupted attentional performance above and beyond general attentional control impairment (i.e., longer reaction times towards threat words compared to neutral words) implicates some degree of enhanced threat processing. However, the fact that PTSD was not associated with greater EST effect sizes in masked or randomized designs is inconsistent with the enhanced threat detection hypothesis and qualifies strength of support for enhanced threat detection. The pattern of findings regarding hyperactive threat detection, then, suggests that enhanced threat detection may potentiate general attentional control deficits (i.e., an interacting system hypothesis). Enhanced threat detection may heighten prepotent responses towards threat stimuli (Williams et al., 1996; Wyble et al., 2008), which creates even greater demands on an already weakened attentional control system in PTSD. The key prediction from this hypothesis is that enhanced threat detection will only affect performance when attentional control demands are high and resources are low. By contrast, threat detection does not appear to be enhanced in PTSD to the degree that it impairs performance independent of attentional control functioning (i.e., does not facilitate detection of masked stimuli).

While the present data cannot directly speak to clinical implications, some tentative speculation might be warranted. There has been growing recent interest in attention training as an intervention for anxiety disorders, with preliminary reports suggesting significant reductions in anxiety symptoms (Amir et al., 2008Amir et al., 2009; Hazen et al., 2009; Schmidt et al., 2009). Attention training procedures generally present two stimuli, one threatening and one neutral, on a computer screen, and the participant's task is to detect a subsequent probe that appears in a location previously occupied by either the threat or

neutral stimulus. In the training condition, the probe appears most frequently in the location opposite to the threat stimuli; thus, participants are trained to disengage attention from threat stimuli. Indeed, the only study that has investigated which attentional bias components are affected by attention retraining (Amir et al., 2009) found improvements in disengagement from threat on a separate task. The current finding that enhanced threat processing disrupts attentional control in PTSD may implicate the need for such training procedures. Given that a sizeable percentage of PTSD patients receiving cognitive-behavioral treatments continue to exhibit significant symptoms post-treatment (Foa et al., 1991; 1999; Schnurr et al., 2007), it will be interesting to test whether adding attention retraining procedures can improve the efficacy of these treatments. Also, if impaired EST performance is indeed a risk factor for the subsequent development of PTSD, then attention retraining following traumatic event exposure regardless of PTSD could operate as a preventative intervention. Future research along these lines is necessary for both clinical and theoretical reasons.

It will also be important for future research to investigate how processes presumably measured by the EST (attentional control, threat detection) relate to the other mechanisms implicated in PTSD, such as memory dysfunction (Brewin et al., 2007; McNally et al., 1995), anxiety sensitivity (Marshall, Miles, & Stewart, 2010), and emotion regulation difficulty (Tull et al., 2007). The current conceptualization of these processes is that they represent relatively basic cognitive and emotional functions, which presumably makes them superordinate to some of the other processes implicated in PTSD (e.g., emotion regulation, anxiety sensitivity, avoidance). For example, given the developmental link between attentional control and self-regulation (Posner & Rothbart, 2000), it might be expected that emotion regulation difficulties observed in PTSD are due to causally prior attentional control disruptions. Similarly, preoccupation with unpleasant interoceptive sensations could be due to enhanced detection of these danger signals and decreased ability to disengage attention from them. However, future research is clearly necessary to test this conceptualization. It will also be interesting to test how impaired attentional control and threat processing relate to memory dysfunction implicated in PTSD (Brewin et al., 2007; McNally et al., 1995). It could be the case that memory dysfunction and the current processes are either overlapping or distinct impairments. For example, perhaps overgeneral autobiographical memories (McNally et al., 1995) and poor attentional control in the context of threat both reflect deficits in a shared higher-order cognitive control process. Future research is necessary to clarify the relationships between different cognitive dysfunctions identified in PTSD.

Several limitations must qualify conclusions from this study. First, the only mechanisms the current data address are those presumably measured by the EST. Discussion was accordingly focused on threat processing and attentional control, but it is important to note that the current results cannot speak to the relative centrality of attentional control and threat detection to understanding PTSD. Second, most of the studies using a blocked design also used a card format, as opposed to computerized format. This means that design is somewhat confounded with other experimental procedures and conclusions based on this manipulation must be tempered accordingly. Third, the EST is just one of many tasks used to assess attentional and emotional processes. It will be important for future research to continue to examine these processes using novel methodological approaches. Fourth, between-group differences were tested by comparing the within-group differences between the PTSD and comparison groups. This was done because of the significant group differences in performance on neutral trials, but this statistical approach is less powerful because the groups are not compared on a study by study basis, which would hold extraneous study factors (e.g., stimulus words, diagnostic procedures, data cleaning, etc.) constant. Fifth, it is uncommon for emotional words in EST research to be matched in arousal, and emerging research suggests that arousal, not valence, is more important for disrupting attentional

processes (Anderson, 2005). As such, it remains possible that any differences observed between emotional words (PTSD-threat versus general-threat versus positive) are due to differences in arousal and not due to valence or semantic category. While conclusions from this meta-analysis must accordingly be tempered, this meta-analysis represents the first attempt to quantitatively synthesize results from the EST research in PTSD and provides initial evidence for the negative consequences of trauma exposure on attention towards threat and for disrupted attentional control and enhanced threat detection.

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*indicates an article included in the meta-analysis.

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Table 1

Study Characteristics

Study Name	Assessment Procedure	Publication	*PTSD N*	*Trauma Control N*	*No Trauma Control N*	*% Female*	*Stimulus Presentation*	*Trial Design*	*Trauma Type*	*Stimulus Words*
Beck et al., 2001	Structured Interview	J	28	26	-	75	U	n/a	MVA	PT, GT, P, N
Beckham et al., 1996	Structured Interview	J	25	-	-	0	U	B	Combat	PT, N
Bryant & Harvey, 1995	Questionnaire	J	15	15	-	47	U	R	MVA	PT, P, N
Buckley et al., 2002	Structured Interview	J	30	-	30	73	U, M	R	MVA	PT, GT, N
Cassiday, 1991	Structured Interview	D	5	5	5	88	U	R	SA	PT, P, N
Cassiday, 1991	Structured Interview	D	7	7	7	100	U	B	SA	PT, P, N
Constans et al., 2006	Structured Interview	J	15	-	-	0	U	R	Combat	PT, GT, N
Devineni et al., 2004	Structured Interview	J	23	-	-	95	U, M	R	MVA	PT, N
Foa et al., 1991	Structured Interview	J	15	13	16	100	U	R	Rape	PT, GT, N
Freeman & Beck, 2000	Structured Interview	J	20	13	20	100	U	R	SA	PT, GT, P, N
Garcia, 2007	Questionnaire	D	13	24	21	100	U	R	SA	PT, GT, P, N
Harvey et al., 1996	Questionnaire	J	20	20	20	73	U, M	R	MVA	PT, N
Kaspi et al., 1995	Structured Interview	J	30	30		0	U	B, R	Combat	PT, GT, P, N
Litz et al., 1996	Structured Interview	J	24	15		0	U	B	Combat	PT, GT, N
McNally et al., 1990[a]	Structured Interview	J	15			0	U	B	Combat	PT, GT, P, N
McNally et al., 1993	Structured Interview	J	24	-	-	0	U	B	Combat	PT, GT, P, N
McNally et al., 1996	Structured Interview	J	14	14		0	U, M	B	Combat	PT, P, N
McNeil et al., 1999	Structured Interview	J	17			47	U	R	Other	GT, N
Metzger et al., 1997	Structured Interview	J	9		10	40–67	U	B	Other	PT, P, N

Study Name	**Assessment Procedure**	**Publication**	*PTSD N*	*Trauma Control N*	*No Trauma Control N*	*% Female*	*Stimulus Presentation*	*Trial Design*	*Trauma Type*	*Stimulus Words*
Moradi et al., 1999	Structured Interview	J	23		23	54	U	R	Other	PT, GT, P, N
Paunovic et al., 2002	Structured Interview	J	39		39	59	U, M	-	Mixed	PT, P, N
Rampersaud, 2006	Questionnaire	D	30	30	30	100	U	R	SA	PT, GT, P, N
Sawhney, 2002	Structured Interview	D	23	21	21	100	U	B	Rape	PT, GT, N
Shin et al., 2001	Structured Interview	J	8	8		0	U	B	Combat	PT, GT, N
Tayler et al., 2006	Structured Interview	J	11	-	-	73	U	B	Other	PT, GT, P, N
Thrasher et al., 1994	Structured Interview	J	13	20	12	25–50	U	B	MVA	PT, GT, P, N
Vrana et al., 1995	Structured Interview	J	42	15	-	0	U	B	Combat	PT, GT, N

Note. J = journal article, D = dissertation. PT = PTSD-relevant threat, GT = general threat, P = positive, and N = neutral. U = unmasked presentation, M = masked presentation. R = randomized design, B = blocked design.

[a]This study included a combat-exposed group without PTSD, but the raw data relevant for the meta-analysis were only available for the PTSD group.

Table 2

Summary statistics of overall word type comparisons by group.

Group	Comparison	*K*	*d*	*Sed*	Var.	*95% C.I.*	*Q (df)*
PTSD							
	GT vs N	19	.28*	.05	.002	.18 – .37	(18) 71.21*
	GT vs P	11	.13*	.05	.002	.04 – .23	(10) 25.15*
	P vs N	19	.07	.07	.001	.01 – .14	(18) 35.92*
	PT vs GT	18	.29*	.06	.003	.18 – .40	(17) 83.89*
	PT vs N	32	.39*	.05	.003	.29 – .49	(31) 217.40*
	PT vs P	19	.29*	.06	.003	.19 – .40	(18) 90.74*
Trauma Control							
	GT vs N	11	.17	.07	.005	.04 – .30	(10) 48.80*
	GT vs P	7	.15*	.05	.002	.06 – .25	(6) 11.23
	P vs N	12	.02	.05	.003	−.08 — .13	(11) 31.42*
	PT vs GT	11	.22*	.07	.005	.08 – .36	(10) 55.05*
	PT vs N	18	.24*	.07	.005	.11 – .38	(17) 117.55*
	PT vs P	12	.22*	.06	.003	.10 – .33	(11) 39.18*
No Trauma Control							
	GT vs N	6	.14	.08	.01	−.02 — .29	(5) 19.13*
	GT vs P	5	.01	.09	.01	−.17 — .18	(4) 15.77*
	P vs N	10	−.01	.03	.001	−.07 — .06	(9) 5.25
	PT vs GT	7	.11	.09	.01	−.07 — .29	(6) 29.64*
	PT vs N	15	.14	.07	.004	.01 – .27	(14) 76.07*
	PT vs P	10	.04	.06	.004	−.09 — .17	(9) 33.50*

Note. PT = PTSD-relevant threat, GT = general threat, P = positive, and N = neutral.

* indicates significant at $p < .01$. *Sed* = standard error, Var = variance.

Table 3

Group moderator analyses of overall comparisons.

Comparison	PTSD *d*	Trauma Control *d*	No Trauma Control *d*	Group Moderator Effect	PTSD vs NTC	PTSD vs TC	TC vs NTC
GT vs N	.28*	.17	.14	Q = 3.09	-	-	-
GT vs P	.13*	.15*	.01	Q = 2.29	-	-	-
P vs N	.07	.02	−.03	Q = 2.73	-	-	-
PT vs GT	.29*	.22*	.11	Q = 2.93	-	-	-
PT vs N	.39*	.24*	.14	Q = 9.08*	Q = 9.01*	Q = .92	Q = 4.08*
PT vs P	.29*	.22*	.04	Q = 9.18*	Q = 8.7*	Q = 2.85	Q = 1.15

Note. Specific comparisons were made between the three groups only when there was evidence that the 'group' variable significantly moderated the effect. NTC = no trauma control. TC = trauma control.

* indicates a significant effect.

Table 4

Summary of moderator findings for the PTSD and Trauma-Exposed control groups.

		Stroop Type		Publication Type		Trial Design		Assessment		Trauma Type		
Group	**Comparison**	**Unmasked**	**Masked**	**Journal**	**Dissertation**	**Blocked**	**Random**	**Structured Interview**	**Self-report**	**Assault**	**Non-Assault**	**Mixed**
PTSD												
	GT vs N	n/a	n/a	-	-	-	-	-	-	-	-	-
	GT vs P	n/a	n/a	-	-	-	-	-	-	-	-	-
	P vs N	-	-	-	-	-	-	-	-	-	-	-
	PT vs GT	n/a	n/a	-	-	.40	.21	-	-	.34	−.04	.38
	PT vs N	.44	.09	-	-	.57	.26	.42	.25	.48	.23	.34
	PT vs P	.33	−.03	-	-	.44	.24	-	-	-	-	-
TC												
	GT vs P	n/a	n/a	-	-	-	-	-	-	-	-	-
	PT vs GT	n/a	n/a	-	-	-	-	-	-	.28	−.01	n/a
	PT vs N	-	-	-	-	.39	.11	.34	.02	-	-	-
	PT vs P	-	-	-	-	-	-	-	-	-	-	-

Note. PT = PTSD-relevant threat, GT = general threat, P = positive, and N = neutral. Moderator analyses were only computed when there was evidence of a significant overall effect within a group. Effect sizes are only displayed where there was evidence of a significant moderator. n/a = analysis not computed due to low number of studies available. TC = Trauma Control. No effect sizes displayed for no trauma control group, as there was no evidence of significant overall effect sizes.