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Clinical and Neural Correlates of Alexithymia in PTSD:

Reexperiencing, Hyperarousal, Numbing, Dissociation, and Childhood Emotional Neglect

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Abstract

Individuals with PTSD often exhibit deficits in emotional experience and expression suggesting that certain individuals with PTSD may be alexithymic. In this study, psychological correlates of alexithymia included PTSD re-experiencing, hyperarousal, numbing, and dissociative symptoms and retrospectively-reported experiences of childhood emotional neglect. Functional neural responses to trauma-script imagery were associated with severity of alexithymia, including increased right posterior-insula and ventral posterior-cingulate activation, and decreased bilateral ventral anterior-cingulate, ventromedial prefrontal, anterior-insula, and right inferior frontal cortex activation. Clinical and theoretical implications and future research directions are discussed.

Clinical and Neural Correlates of Alexithymia in PTSD:

Reexperiencing, Hyperarousal, Numbing, Dissociation, and Childhood Emotional Neglect

Under the current psychiatric nosological system, posttraumatic stress disorder (PTSD) is classified as an instance of the anxiety disorders (American Psychiatric Association, 1994) and accordingly is chiefly construed as a condition involving elevated subjective anxiety. Accordingly, contemporary psychological models of PTSD predominantly aim to explain the information processing mechanisms underlying individuals' subjective anxiety (Brewin & Holmes, 2003; Dalgleish, 2004) and fear conditioning and extinction models represent the principal theoretical platform for current studies of the psychobiology of PTSD (see Yehuda, 2006, for recent reviews).

Notwithstanding the centrality of anxiety symptomatology to PTSD, clinical studies reveal that anxiety symptoms represent only a fraction of the psychopathological sequelae that may ensue as a consequence of prolonged exposure to traumatic stressors (e.g., van der Kolk, Roth, Pelcovitz, Sunday, & Spinnazzola, 2005). For example, individuals with PTSD may display extreme anger (Orth & Wieland, 2006), shame, guilt, dysphoria, and dissociation (e.g., Andrews, Brewin, Rose, & Kirk, 2000; Ehlers, Mayou, & Bryant, 1998; Feeny, Zoellner, Fitzgibbons, & Foa, 2000). Conversely, studies increasingly document the presence of 'emotional numbing' symptoms in the PTSD population which are characterized by a *restricted* range of affect (e.g., Kashdan, Elhai, & Frueh, 2006; Litz, Orsillo, Kaloupek, & Weathers, 2000). A key finding is that hyperarousal and emotional numbing symptoms are *positively* rather than negatively correlated in PTSD populations and may be functionally related (e.g., Buckley, Blanchard, & Hickling, 1998; Taylor, Kuch, Koch, Crockett, & Passey, 1998; Simms, Watson, & Doebbelling, 2002).

PTSD Hyperarousal, Emotional Numbing & Alexithymia

Previous psychological theories have proposed a temporal-sequential process in order to account for the otherwise paradoxical co-elevation of hyperarousal and emotional numbing symptoms in PTSD. For example, Litz (1992; Litz & Gray, 2002) advanced an influential network model in which trauma cues prime fear-related information processing that in turn temporarily inhibits access to emotions of an incompatible valence (i.e., positive affect). An important corollary of these models is that they account for emotional numbing symptoms principally as a secondary consequence of hyperarousal symptoms. To illustrate, Litz et al. (2000, p. 27; see also M. W. Miller & Litz, 2004) posited that the general "capacity to experience and express a variety of emotions is unaltered in PTSD".

Other evidence, however, contests that a primary disturbance in emotional experience and expression may characterize certain complex cases of PTSD. In particular, an emerging number of studies identify a positive association between PTSD symptoms and alexithymia (e.g., Badura, 2003; Cloitre, Scarvalone, & Difede, 1997; Fukunishi, Sasaki, Chishima, Anze, & Saijo, 1996; Hyer, Woods, Summers, Boudswyns, Harrison, & 1990; Monson, Price, Rodriguez, Ripley, & Warner, 2004; Söndergaard, & Theorell, 2004; Yehuda et al., 1997; Zlotnick, Mattia, & Zimmerman, 2001). The term 'alexithymia' was first defined by Sifneos (1973) to refer to a symptom constellation he observed in psychosomatic patients, specifically, that these individuals were often unable to identify and label their emotional feeling states (see also Nemiah, Freyberger, & Sifneos, 1973; Nemiah & Sifneos, 1970; Sifneos, 1967; Taylor, Bagby, & Parker, 1997). To the extent that PTSD emotional numbing symptoms reflect affective arousal or pain that has been 'anesthetized' in some way (Monson et al., 2004), one logical outcome may be a perceived difficulty in recognizing, describing, and regulating emotional responses (Krystal &

Krystal, 1988). Findings that link alexithymia with hyperarousal, however, suggest another conceptualization of the relationship between alexithymia and PTSD. Specifically, posttraumatic alexithymia may signify an uncoupling of cognitive and emotional processing within which intense emotional states become poorly integrated with verbal cognition. This construal of alexithymia is in better keeping with its contemporary conceptualization as a difficulty in identifying and labeling feelings (Taylor et al., 1997). Consistent with the 'blindfeel hypothesis' (Lane, Ahern, Schwartz, & Kaszniak, 1997), individuals with posttraumatic alexithymia may manifest physiological and behavioural profiles indicative of hyper-emotionality yet they may not be consciously aware of their feelings, causing them to report that they either feel nothing at all or do not know what they feel.

Although a cursory overview of Litz's (1992; Litz et al., 2000; Litz & Gray, 2002; M. W. Miller & Litz, 2004) network model might inaccurately suggest that the capacity for higher order emotional experience is wholly unperturbed in individuals with PTSD, closer examination suggests that the alexithymia and network models are *not* inherently incompatible. Specifically, according to the network theory, for individuals with PTSD "the building blocks of emotional experience… that were available to the individual *before* they were traumatized are intact, as is *pre*traumatic, elaborated emotional knowledge or schemas" (Litz et al., 2000, p. 27, italics added). In other words, the explanatory scope of the network model may parsimoniously encompass a relationship between intact emotional functioning pre-trauma and the presentation of seemingly expansive emotional processing deficits post-trauma. However, certain veritable cases of pervasive affective disturbance may result from deprived or absent exposure to adaptive emotional learning and attachment experiences early in development (e.g., Cloitre et al., 1997). In support of this hypothesis, Zlotnick, Mattia, and Zimmerman (2001) found that alexithymia

levels were distinctly associated with reported emotional and physical neglect during childhood in a heterogenous sample of psychiatric patients that included individuals with PTSD. It is therefore possible that emotional neglect and/or maltreatment occurring during childhood may obstruct the normal development of emotional processing skills, leading to alexithymia and a vulnerability to developing PTSD in adulthood.

Neuroimaging Studies of PTSD & Alexithymia

Further evidence in support of a possible intersection between PTSD and alexithymic clinical presentation comes from neuroimaging studies in which relationships have been found between the neural correlates of symptom provocation in PTSD and emotional processing paradigms in alexithymia. Of the PTSD symptom provocation paradigms, script-driven imagery has been the most extensively studied to date (reviewed by Lanius, Bluhm, Lanius, & Pain, 2006). This paradigm involves exposing individuals to an audio script that briefly recounts his or her traumatic life event. The participant is instructed to listen to the script and imagine the event happening. Compared with previously traumatized individuals who do not develop the disorder, individuals with PTSD typically exhibit greater psychophysiological reactivity (reviewed in Orr, McNally, Rosen, & Shalev, 2004) and demonstrate less activation in anterior cingulate cortex (ACC; Brodmann areas [BA] 24, 25, 32) and medial prefrontal cortex (mPFC, BA 9, 10) relative to trauma exposed nonpsychiatric controls (reviewed in Lanius et al., 2006; Yehuda, 2006). Specific subregions of the ACC have been associated with affective-, autonomic-, and attentional-control as well as mood and anxiety disorders (e.g., Critchley, 2005; Ochsner & Gross, 2005; Seminowicz et al., 2004; Steele & Lawrie, 2004). Diminished response in ventral-ACC in the PTSD population may therefore be consistent with clinical observations that these individuals are unable to modulate the intensity of their emotional reactions in the

presence of reminders of their traumatic experiences (Frewen & Lanius, 2006). The mPFC has been shown to activate during self-referential emotional processing tasks (Gilbert et al., 2006; Northoff, Heinzel, de Greck, Bermpohl, Dobrowolny, & Panksepp, 2005; Ochsner et al., 2004) and has also been found to be involved in inner- or 'self'-directed mental activity (i.e., during periods of cognitive processing characterized by a lack of externally-driven task-focused attention; Gusnard, Akbudak, Shulman, & Raichle, 2001; Fox, Corbetta, Snyder, Vincernt, & Raichle, 2006). Reduced activation of the mPFC in PTSD may therefore be consistent with a more pronounced deviation from baseline reflective/self-referential processing in individuals with PTSD and may also signify a deficiency in emotional self-awareness during traumatic memory recall (Frewen & Lanius, 2006). Studies have also revealed disturbances in activation of the posterior cingulate cortex (PCC, BA 23, 30, 31), which is known to be activated during episodic memory retrieval and pain processing (Nielsen, Balslev, & Hansen, 2005) as well as the assessment of emotional self-relevance (Northoff et al., 2005), and the right-inferior frontal cortex, which is know to be involved in emotion regulation (Ochsner & Gross, 2005). The direction of these differences has been less systematic across PTSD neuroimaging studies. Finally, Osuch et al. (2001) observed a positive correlation between activation in the left and right insula and self-reported flashback intensity during trauma script-driven imagery.

An emerging number of studies have also examined the functional neural correlates of alexithymia symptoms in the context of emotional processing paradigms. These studies have demonstrated alterations in the functional responsiveness of ventral and dorsal ACC (Berthoz et al., 2002, Kano et al., 2003; Lane et al., 1998), ventral and dorsal mPFC (Berthoz et al., 2002; Kano et al., 2003), right middle insula (Kano et al., 2003) and PCC (Mantani, Okamoto, Shirao, Okada, & Yamawaki, 2005) to be associated with alexithymia. Provided that these same

structures have implicated in the pathophysiology of PTSD, this suggests that the functional brain alterations underlying symptoms of alexithymia and PTSD may be related.

The Present Study

Accordingly, the present study examined the clinical and neural correlates of alexithymia in PTSD. Psychometrically-defined individual differences in alexithymia were predicted to correlate positively with PTSD symptomatology, specifically re-experiencing, avoidance, emotional numbing, hyper-arousal, and dissociative symptoms. In addition, alexithymic symptoms were predicted to correlate positively with severity of retrospectively-reported childhood abuse, particularly experiences of emotional neglect, as would be hypothesized by developmental models such as the levels of emotional awareness model (Lane & Schwartz, 1987) and on the basis of previous research (Zlotnick et al., 2001). Additionally, during a trauma script-driven imagery paradigm using functional magnetic resonance imaging (fMRI), alexithymia symptoms were predicted to be associated with activity in brain regions associated with emotional processing and control, specifically ventral ACC, ventromedial PFC, right inferior frontal cortex, and insula. This study was approved by the Health Sciences Research Ethics Board of the University of Western Ontario.

Method

Participants

One hundred five individuals with a principal diagnosis of PTSD participated in the interview and survey completion component of this study as did 45 nonpsychiatric controls. The control group met PTSD Criterion A but had no lifetime history of Axis I psychiatric disturbance as assessed by the *Structured Clinical Interview for DSM-IV (SCID-I)*.

Diagnosis of PTSD was established via the *Clinician Administered PTSD Scale* (*CAPS;* Blake et al., 1995) by clinicians with extensive hospital-based experience with the PTSD population who had received formal instruction in its administration by one of the lead developers of this instrument, Dr. F. Weathers, prior to the commencement of this study. Comorbid Axis I conditions were established via the SCID-I by the same clinicians who received formal instruction in the administration of this instrument at a distinguished psychiatric research institution prior to the commencement of the study (the *Centre for Addiction & Mental Health*, Toronto, Ontario). Individuals with a positive history for lifetime bipolar disorder, lifetime psychotic disorders, or current substance abuse as determined by the SCID-I were excluded from participation. All diagnoses were confirmed in consultation with the study psychiatrist (R.A.L.). Table 1 characterizes the PTSD sample in terms of demographics, clinical severity, and comorbidity. There were no statistically significant differences between the PTSD sample and the control group in ethnic or gender composition, marital status, mean age, or years of education, although the control group was approximately twice as likely to be employed (full or part time) at the time of the study (37% vs 74%, $\chi^2(2) = 22.87$, $p < .001$ [full/part time employment vs. unemployment]).

A subset of the PTSD sample ($n = 26$, 71% female, all right-handed) also participated in a trauma script-driven imagery fMRI study (the remaining PTSD participants also participated in other research studies following their psychological assessment that are not pertinent to the present investigation). All primary traumatic events for participants in the fMRI study were motor vehicle accidents (MVA). In this subsample there was no medical or neurological morbidity associated with head injury or with past drug or alcohol use as assessed by interview in participants with a history of substance abuse. All participants who were receiving

medications in this subsample prior to the script-driven imagery study ($n = 16$, 69%) had undergone a supervised drug washout for at least two weeks prior to fMRI scanning and none of the participants were receiving fluoxetine prior to the drug washout. Participants with a history of substance use disorder in remission for less than six months were excluded from this study, as were individuals with any significant medical conditions, neurological illness or history of a significant head injury with loss of consciousness as assessed by interview.

Measures

   20-item Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994). The TAS-20 is the most widely used and validated self-report measure of alexithymia (reviewed in Taylor et al., 1997). An example of a TAS-20 item is: "I am often confused about what emotion I am feeling". High TAS-20 scores indicate higher alexithymia. The internal consistency reliability for the TAS-20 in the current sample was .93 and the intraclass correlation coefficient was .38.

   Clinician Administered PTSD Scale (CAPS; Blake et al., 1995). The CAPS assesses both the frequency and intensity of each of the DSM-IV PTSD symptoms and is widely considered to be the gold standard in semi-structured diagnostic interviews for PTSD. High CAPS scores indicate greater severity of PTSD. The internal consistency reliability for the CAPS items (with frequency and intensity items summed) overall in the current sample was .97 and the intraclass correlation coefficient was .66. The four symptom clusters of the CAPS identified by King, Leskin, King, and Weathers (1998) were also scored separately (Re-experiencing, Effortful Avoidance, Emotional Numbing, and Hyperarousal). The internal consistency reliability and intraclass correlation coefficients for each CAPS subscale in the present sample were: Re-experiencing ($\alpha = .91$, $r = .69$), effortful avoidance ($\alpha = .82$, $r = .70$), emotional numbing ($\alpha = .92$, $r = .76$), and hyperarousal ($\alpha = .92$, $r = .69$).

Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986) is a well-recognized 28-item measure of trait dissociative experiences. The internal consistency reliability for the DES in the current sample was .94 and the intraclass correlation coefficient was .36.

Childhood Trauma Questionnaire – Short Form (CTQ-SF; Bernstein et al., 2003). The CTQ-SF is a standardized measure of individuals' exposure to traumatic events during childhood and adolescence. The CTQ-SF has five subscales: *Emotional Neglect*, *Emotional Abuse*, *Sexual Abuse*, *Physical Abuse*, and *Physical Neglect*. The internal consistency reliability of these subscales in the present sample was: Emotional Neglect ($\alpha = .93$, $r = .74$), Emotional Abuse ($\alpha = .93$, $r = .73$), Sexual Abuse ($\alpha = .97$, $r = .87$), Physical Abuse ($\alpha = .92$, $r = .69$), and Physical Neglect ($\alpha = .80$, $r = .45$).

Procedure

After thorough description of the study to the research participants, written informed consent was obtained. Diagnostic interviews (SCID-I and CAPS) were then conducted, followed by administration of the psychometric measures listed above, after which participants were debriefed.

A subset of the participants then completed a fMRI trauma-script imagery study as discussed above (the remaining participants variously took part in other additional psychological and neuroimaging studies conducted at our institution, as noted above). These participants, following administration of the CAPS, provided detailed descriptions of the traumatic MVA upon which the CAPS assessment was based in addition to a detailed description of a neutral memory occurring in close proximity to the traumatic event. This assessment took place approximately 1 week before fMRI scanning. Participants' descriptions of their traumatic and neutral memories were translated into 30-second scripts that were then audio-recorded using the

Windows© Sound Recorder tool. Following established methods (G. A. Miller et al., 1987; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987) the scripts were written in the third person.

MRI scanning of the neutral and traumatic imagery conditions was repeated for three trials within test-blocks (runs). Participants listened in blocked order to three repetitions of their neutral memory scripts followed by three repetitions of their traumatic memory scripts. Script order was not counterbalanced in order to prevent anxiety and other emotions elicited by the traumatic scripts from affecting the processing of neutral memories, consistent with previous studies (Bremner et al., 1999; Lanius et al., 2001, 2002, 2003). Participants were instructed to lie still and allow themselves to begin focusing on the scripts as soon as the script was read. Participants were further instructed to remember olfactory, auditory, somatosensory, and visual sensations that were associated with their traumatic event as soon as the script of their traumatic event began, and throughout the duration of the script and 30-second period of silence that immediately followed its presentation. One hundred and twenty additional seconds then passed before the script was repeated. During this time participants were asked to lie still, breathe through their nose, and attempt to "let go" of their traumatic memories. Following the three repetitions of each script type participants were interviewed regarding their response to the paradigm via the *Responses to Script-Driven Imagery Scale*, an 11-item measure of PTSD re-experiencing, avoidance, and dissociative symptoms prompted by trauma script-driven imagery. Participants answered questions by giving a number from 0 to 6 where 0 referred to "Not at all" and 6 referred to "A great deal".

All imaging data was acquired on a 4 Tesla Varian UNITY^{INVOA} whole body MRI system (Palo Alto, CA, USA) equipped with Siemens Sonata actively shielded gradient coils (Erlangen, Germany). A cylindrical transmit-receive hybrid birdcage radio frequency (RF) head coil was

used for transmission and detection of signal. The subject's head was immobilized with foam padding and a Plexiglas head cradle within the head coil.

Preliminary T_1-weighted sagittal images were acquired using a fast low-angle shot (FLASH) inversion-recovery sequence [128 x 128 matrix size, field-of-view (FOV) = 28 cm, inversion time (TI) = 750 ms, echo time (TE) = 3.5 ms, repetition time (TR) = 8 ms, tip-angle = 11°], which provided excellent grey/white matter contrast. From these localizer images, 12 contiguous functional planes were prescribed with an axial orientation approximately parallel to the anterior commisure (AC)-posterior commisure (PC) line (centered on a plane level with the anterior cingulate cortex) and a slice thickness of 6 mm. A constrained, three-dimensional phase shimming procedure (Klassen & Menon, 2004) was performed to optimize the magnetic field homogeneity over the prescribed functional volume. During each functional task, blood oxygenation level dependent (BOLD)-sensitive images were collected using a navigator-corrected four segment echo planar imaging (EPI) sequence (128 x 128 matrix size, FOV = 22 cm, TE = 10 ms, TR = 1250 ms, flip angle = 40º, 108 volumes, volume collection time = 5 s). For registration of the BOLD sensitive images, a high-resolution T_1-weighted anatomic reference volume was acquired with the same axial FOV using a three-dimensional FLASH sequence (256 x 256 x 64 matrix size, slice thickness = 3 mm, TI = 600 ms, TE = 5.5 ms, TR = 10 ms, flip angle = 11º).

Statistical Analyses

Standard pairwise correlation coefficients evaluated the significance of associations between TAS-20 scores and CAPS, DES, and CTQ-SF scores. Tests of the differential magnitude of correlations between TAS-20 scores and the various subscales of the CAPS and CTQ-SF were tested following the method of Meng et al. (1992).

Statistical analysis of the fMRI data employed voxel-wise general linear models with design matrices comprised of epoch-related regressors. Baseline brain activation (i.e., the 'implicit' baseline) was calculated based on the average activation patterns 60s prior to each audio presentation of the traumatic event script. Brain activation associated with the presentation of the neutral and traumatic event scripts was calculated based on average activation patterns occurring during the silent 30s that followed each script. Significant differences in location and intensity of BOLD response during the trauma script-driven imagery task, relative to the neutral script-driven imagery task, were ascertained by use of basic subtraction analyses using Statistical Parametric Mapping (SPM99; Wellcome Department of Neurology, London, UK: http://www.fil.ion.ucl.ac.uk/spm). These linear contrasts yielded statistical parametric maps of the t-statistic, referred to hereafter as *contrast images*. Participants' TAS-20 scores were then correlated with their individual contrast images to identify clusters of activation associated with alexithymia in a whole-brain random-effects model with $k \geq 50$ voxels and two-tailed $\alpha \leq .05$ corresponding to a minimum $|r| \geq .39$ with $df = 24$ (uncorrected for multiple comparions). The voxel within each cluster that was observed to exhibit the strongest correlation with TAS-20 scores within each cluster was then reported only if the voxel survived correction for multiple comparisons (small volume corrected [SVC] with a $p < .05$ [referred to as p_{SVC}] for false discovery rate) within a 5mm spherical search volume centered at the identified voxel. The coordinates of these voxels are reported within Montreal Neurological Institute space (x,y,z; referring to the location in mm of the activation within the brain relative to the AC/PC line in terms of the sagittal [mediolateral, left = negative, right = positive], transverse [superior = positive, inferior = negative], and coronal [anterior = positive, posterior = negative] planes, respectively). Clusters of voxels meeting the above criteria that were observed in the stated a

priori regions of interest (ACC, PCC, mPFC, Insula, inferior frontal cortex) as confirmed against the atlas of Taliarach and Tournoux (1988) were accepted as statistically significant. The locations of peak activation reported in this article approximate those found associating functional brain responses with PTSD or alexithymia in previous studies of the vACC-mPFC (Lanius et al., 2001), right inferior frontal cortex (Lanius et al., 2002), vPCC (Mantani et al., 2005), left and right anterior insula (Osuch et al., 2001), and left superior temporal cortex (Kano et al., 2003).

Results

Full Sample

Group Differences in Alexithymia

Consistent with previous research, individuals with PTSD reported higher TAS-20 symptoms of alexithymia ($M = 59.38$, $SD = 13.67$) than did the nonpsychiatric control group ($M = 35.39$, $SD = 8.86$), $t(148) = 12.85$, $p < .001$, $d = 2.71$ (large effect size).

Association between Alexithymia, PTSD, and Dissociative symptoms

The observed correlations between TAS-20 scores and the CAPS total and subscale scores in the PTSD sample were: Total, $r = .45$, $p < .001$, Re-experiencing, $r = .32$, $p < .001$, Effortful Avoidance, $r = .09$, $p = .19$, Emotional Numbing, $r = .43$, $p < .001$, and Hyperarousal, $r = .34$, $p < .001$. TAS-20 scores were less significantly correlated with Effortful Avoidance scores than with the other PTSD symptom constellations: Emotional Numbing, $Z = 3.63$, $p < .001$, Hyperarousal, $Z = 2.58$, $p < .01$, and Re-experiencing, $Z = 2.39$, $p < .05$. The observed correlation between TAS-20 and DES scores in the PTSD sample was $r = .39$, $p < .001$.

Association between Alexithymia and Childhood Abuse and Neglect

TAS-20 scores were significantly positively-correlated with the Emotional Neglect subscale of the CTQ-SF, $r = .29$, $p = .001$. In contrast, TAS-20 scores were not significantly correlated with the CTQ-SF Emotional Abuse, $r = .09$, Physical Neglect, $r = .15$, Physical Abuse, $r = .16$, or Sexual Abuse, $r = .09$ subscales. TAS-20 scores were more significantly correlated with Emotional Neglect scores than with the Emotional Abuse and Sexual Abuse subscale scores: Emotional Abuse, $Z = 2.84$, $p < .01$, and Sexual Abuse, $Z = 2.19$, $p < .05$. In contrast, comparisons with Physical Abuse and Physical Neglect scores failed to reach statistical significance: Physical Abuse, $Z = 1.46$, $p = .14$, and Physical Neglect, $Z = 1.75$, $p = .08$.

fMRI Participants

Phenomenological response

The majority of participants reported re-experiencing symptoms in response to the trauma script-driven imagery paradigm, as measured by the RSDI. To illustrate, 60% indicated 3 or higher on the 0 to 6 scale in response to the RSDI item "Did you feel as though the event was reoccurring, like you were reliving it?", 67% indicated 3 or higher in response to the RSDI item "Were you distressed?", and 45% indicated 3 or higher in response to the RSDI item "Were you emotionally upset?".

Association between Alexithymia and BOLD-fMRI Response to Trauma Script Imagery

Two distinct clusters were observed where greater BOLD signal in the trauma-script minus neutral-script condition was associated with *higher* levels of alexithymia within the a priori regions or interest: the right posterior insula ($k = 78$, maximum at MNI 46 -24 22, $r = .55$, $p_{SVC} = .027$) and the right vPCC ($k = 105$, maximum at 8 -50 22, $r = .54$, $p_{SVC} = .029$). Higher levels of alexithymia were also associated with greater BOLD signal in a cluster within the left

superior temporal cortex (k = 116, maximum at -56 -10 8, r = .55, p_{SVC} = .011) although this was not predicted a priori. Please see Figure 1 for illustration.

Conversely, three distinct clusters were observed where greater BOLD signal in the trauma-script minus neutral-script condition was associated with *lower* levels of alexithymia: bilateral ventral-ACC/mPFC (k = 295, maximum at 4 34 10, r = -.63, p_{SVC} < .003), the right inferior frontal cortex extending into the right anterior insula (k = 68, maximum at 50 22 -4, r = -.54, p_{SVC} = .026), and the left anterior insula (k = 56, maximum at -34 16 -6; r = -.45, p_{SVC} = .047). Please see Figure 2 for illustration.

Discussion

The present results indicate that certain traumatized individuals report lacking the ability to reflect on, understand, and modulate their affective symptoms. Furthermore, these subjective reports are predictive of neural responses to exposure to reminders of past traumatic experiences. Together these findings may exemplify a disintegration between these individuals' capacities for cognitive insight, conscious awareness, and focused attention (e.g., as indicated by correlations with activation of the ventral-ACC, ventromedial PFC, and ventral-PCC) and their ongoing affective- and arousal-related bodily experiences (e.g., as indicated by correlations with activation of the bilateral insula).

One of the individuals with PTSD who participated in these studies ("*W.H.*"), unaware of the specific hypotheses under investigation, gave several lucid descriptions of this phenomenon. In the context of the CAPS administration, when inquired about physiological reactivity in the presence of trauma reminders (PTSD Criterion B5), she replied "I don't know *what* I feel, it's like my head and body aren't connected." Later in the diagnostic interview, when asked about restricted range of affect (PTSD Criterion C6), she stated: "I'm living in a tunnel, a fog, no

matter what happens it's the same reaction – numbness, nothing. Having a bubble bath and being burned or raped is the same feeling. *My brain doesn't feel*." Qualitative descriptions such as *W.H.*'s help bring to life the clinical and psychological significance of the quantitative data reported above.

As predicted, the present results demonstrate a connection between the psychological construct of *alexithymia* and severity of PTSD and dissociative symptoms. The finding that alexithymia symptoms not only differentiated participants with PTSD from non-psychiatric controls but also distinguished levels of clinical severity within individuals with PTSD attests to the clinical significance of the findings. Interestingly, alexithymia symptoms were associated most strongly with emotional numbing symptoms, whereas alexithymia was not associated with symptoms of effortful avoidance, partially confirming the relevance of the distinction between these sets of PTSD Criterion C symptoms (e.g., King et al., 1998). Furthermore, the results confirm an association between alexithymia symptoms and self-reported history of childhood emotional neglect (Zlotnick et al., 2001). As cogently argued by Zlotnick et al. (2001), alexithymia in adulthood may procure when a caretaker fails to teach a child how to differentiate between distinctive emotional states, regulate arousal, and respond adaptively to challenging life events.

Moreover, during traumatic memory recall-imagery relative to recall-imagery of neutral events, increasing severity of alexithymia was associated with increasing activity in the right posterior insula, whereas reduced activity with increasing alexithymia was observed in the bilateral ventral-ACC, ventromedial PFC, left anterior insula, and right inferior frontal cortex (extending into the right anterior insula). Associations between individual differences in alexithymia and right insular cortex may have been functionally associated with centrally-

represented body-state mapping of sympathetic autonomic arousal, coupled with reduced executive-regulatory cognitive-affective control via the ventral-ACC, mPFC, and right inferior frontal cortex (Craig, 2002; Critchley, 2005; Critchley et al., 2004; Ochsner & Gross, 2005). Additionally, left anterior insula activation was correlated negatively with alexithymia scores which was predicted from Craig's (2005) model that proposes a hemispheric specificity in the functional activation of insular cortex, with left insula activation suggested to be distinctively involved in positive-affective and affiliative experiences associated with central representations of the activity of the parasympathetic nervous system. However, a negative correlation was also found in the right inferior frontal cortex that extended into the right anterior insula, suggesting that a complex relationship may exist between insular cortex activation and alexithymia in PTSD. It is of note that the right inferior frontal cortex responds to emotion regulation tasks, and therefore a relative absence of response in this area as a function of increasing alexithymia is consistent with a view of alexithymia as a disorder of affect regulation (Ochsner & Gross, 2005; see Taylor et al., 1997). Finally, findings of increasing ventral PCC activation coupled with decreasing ventral ACC activation as a function of increasing alexithymia are significant in light of Vogt and colleagues' demonstration of the normally coordinated involvement of ventral PCC and ACC in ongoing self-monitoring and the assessment of the emotional significance of events (Vogt et al., 2006). In other words, that alexithymia scores were positively associated with activation in the ventral PCC but negatively associated with activation in the ventral-ACC and ventromedial PFC may indicate that a decoupling of this normally integrated emotional-processing circuit during traumatic memory processing is related to alexithymia.

In summary, the present findings appear to be consistent with the hypothesis that the more an individual is capable of verbal awareness, interoceptive monitoring, and higher-order

insight regarding his or her bodily-emotional symptoms, the less likely that he or she will be overwhelmed by them, that is, experience a loss of executive control during reminders of past traumatic events and other stressful events (Frewen & Lanius, 2006). This may be clinically significant in that initially training more highly alexithymic PTSD patients in the ability to identify their affective feelings may lay the groundwork for more efficient trauma-memory focused treatment such as employed in exposure-based therapies (Becker & Zayfert, 2001; Cloitre, Koenen, Cohen, & Han, 2002). Specifically, the latter treatments may be less effective in the early stages of treatment for patients who are unable to cognitively make sense of let alone modulate and regulate the intense affective experiences that may be aroused by exposure procedures.

The present studies have both procedural strengths as well as limitations. The clinical scales administered evidenced good psychometric characteristics, the sample size was satisfactorily large, and the sample was well characterized diagnostically. In addition, participants' evidenced a range of scores on the clinical measures appropriating the continuous correlation analyses reported. These strengths notwithstanding, the present studies remain largely descriptive as all of the psychometric variables were studied via self-report instruments and not longitudinally. An additional limitation was that only a single self-report instrument of alexithymia was used and therefore these results require replication with additional measures. Future research should therefore elucidate the generalizability and direction of causality of the various associations identified in this study. For example, the extent to which alexithymic characteristics serve as vulnerability factors for the development of PTSD, as opposed to sequelae or concomitants of this disorder, remains an open theoretical question warranting further critical study.

In conclusion, the present studies reveal an association between perceived difficulty in identifying and describing emotional states and severity of PTSD symptoms, dissociation, and retrospectively-reported childhood emotional neglect. Additionally, symptoms of alexithymia predicted brain activation associated with exposure to reminders of traumatic memories in areas known to be involved in emotional processing. These findings bear on current theoretical and clinical conceptualizations of the nature of the human response to traumatic life experiences.

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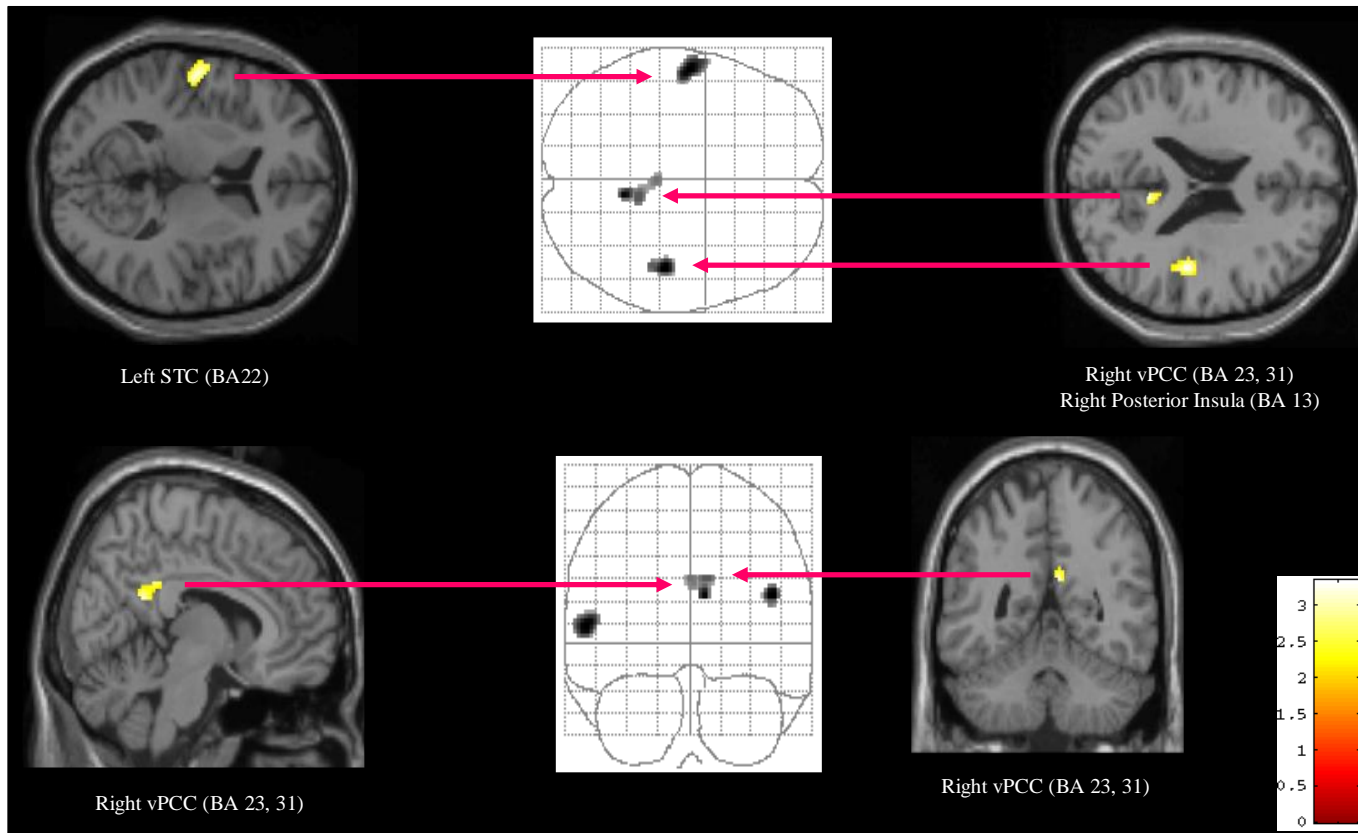
Table 1

Demographic and diagnostic information referring to PTSD participants

	Full Sample (*n* = 105)	fMRI Sample (*n* = 26/105)
Demographics		
% Female	88	71
Mean Age (SD)	38.70 (9.95)	35.95 (13.48)
% Employed (Full or part time)	57	69
Severity of PTSD, Current Treatment		
Mean CAPS (SD)	84.71 (26.03)	80.38 (23.81)
% Currently in Psychotherapy	50	39
% Currently on Psychotropic Medication	79	0
Comorbid Axis I Conditions		
% Major Depressive Disorder	44	54
% Dysthymia	17	4
% Panic Disorder w/wo Agoraphobia	19	12
% Agoraphobia wo Panic Disorder	10	0
% Social Phobia	28	0
% Specific Phobia	12	0
% Obsessive Compulsive Disorder	10	0
% Generalized Anxiety Disorder	33	12
% Body Dysmorphic Disorder	1	0
% Anorexia Nervosa	3	0
% Bulimia Nervosa	3	0
% Pain Disorder	4	0
% Hypochondriasis	2	0
Severity of Alexithymia		
Mean TAS-20 (SD)	59.38 (13.67)	57.77 (14.75)
% ≥ (TAS-20 = 61)	47	35

Note: DSM-IV Disorders not listed were not present in the sample. "SD" = Standard deviation. "CAPS" = Clinician Administered PTSD Scale, "TAS-20" = Toronto Alexithymia Scale – 20 item version.
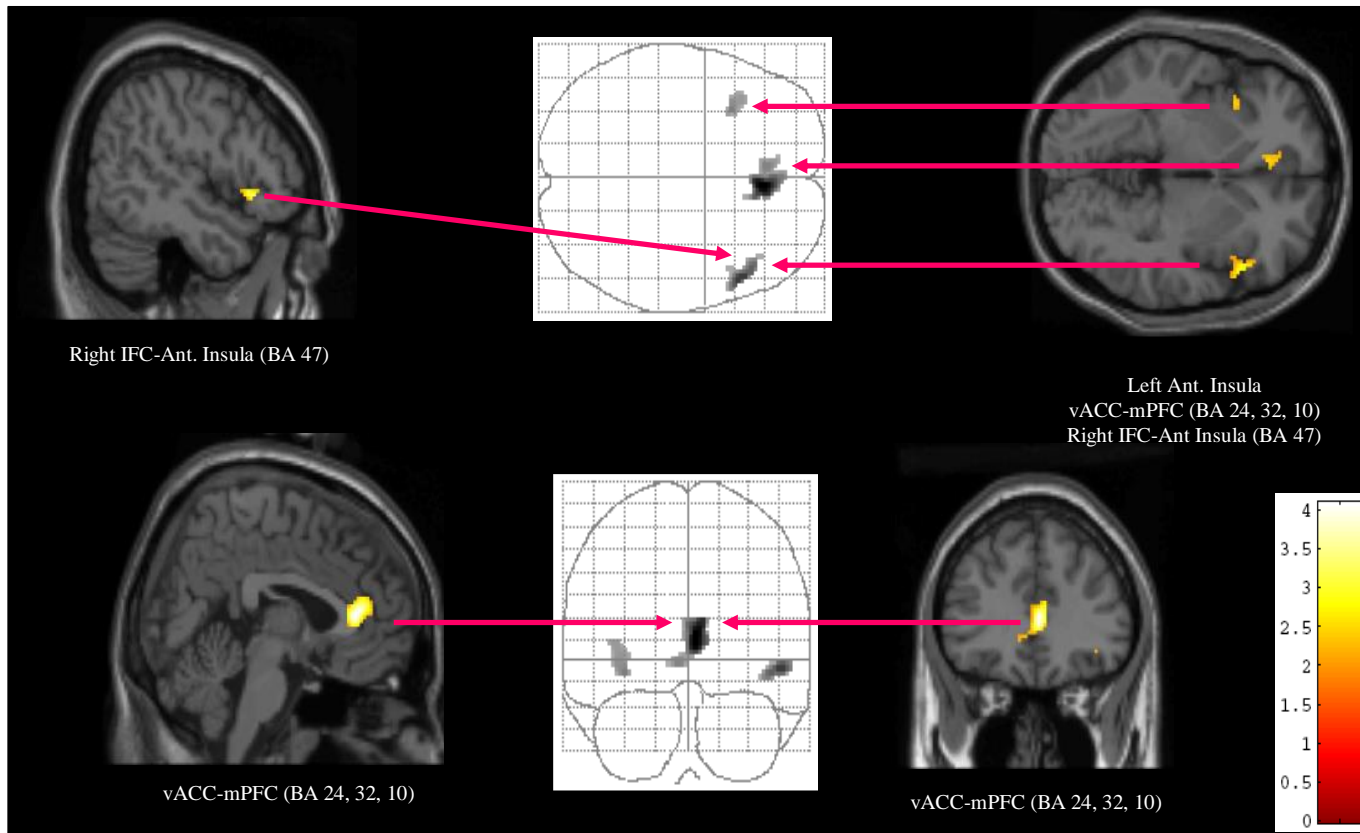
Figure 1

Positive correlation between Alexithymia and Brain Activation During Trauma Script-driven imagery

Left STC (BA22)

Right vPCC (BA 23, 31)
Right Posterior Insula (BA 13)

Right vPCC (BA 23, 31)

Right vPCC (BA 23, 31)

Notes: Presented in neurological convention (Left = Left), $p < 0.05$ (2-tailed), $k \geq 50$, $n = 26$. Participants' TAS-20 scores were regressed on their images of the contrast Trauma Script Imagery – Neutral Script Imagery in a random effects model *(df = 24)*. The color bar denotes the t-statistic of the significance of r (images present cluster activations with $t(24) \geq 2.06$ corresponding to $r(24) \geq .39$). BA = Brodmann Area, vPCC = ventral Posterior Cingulate Cortex, STC = Superior Temporal Cortex.

Figure 2

Negative correlation between Alexithymia and Brain Activation During Trauma Script-driven imagery

Right IFC-Ant. Insula (BA 47)

Left Ant. Insula
vACC-mPFC (BA 24, 32, 10)
Right IFC-Ant Insula (BA 47)

vACC-mPFC (BA 24, 32, 10)

vACC-mPFC (BA 24, 32, 10)

Notes: Presented in neurological convention (Left = Left), $p < 0.05$ (2-tailed), $k \geq 50$, $n = 26$. Participants' TAS-20 scores were regressed on their images of the contrast Trauma Script Imagery – Neutral Script Imagery in a random effects model *(df = 24)*. The color bar denotes the t-statistic of the significance of *r* (images present cluster activations with $t(24) \geq 2.06$ corresponding to $r(24) \geq .39$). BA = Brodmann Area, IFC = Inferior Frontal Cortex, Ant. = Anterior, vACC = ventral Anterior Cingulate Cortex, mPFC = Medial Prefrontal Cortex.