Clinical and Neural Correlates of Alexithymia in Posttraumatic Stress Disorder

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Individuals with posttraumatic stress disorder (PTSD) often exhibit deficits in emotional experience and expression, which suggests that certain individuals with PTSD may be alexithymic. In this study, in a sample of 105 individuals with PTSD, clinical correlates of alexithymia included reexperiencing, hyperarousal, numbing, dissociative symptoms, and retrospectively reported experiences of childhood emotional neglect. In a subsample of 26 individuals with PTSD related to a motor vehicle accident, 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.

Keywords: alexithymia, posttraumatic stress disorder (PTSD), script-driven imagery, neuroimaging, functional magnetic resonance imaging (fMRI)

Under the current psychiatric nosological system, posttraumatic stress disorder (PTSD) is classified as an instance of the anxiety disorders (American Psychiatric Association, 1994) and therefore 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 have revealed 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, & Spinazzola, 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 have increasingly documented 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; S. Taylor, Kuch, Koch, Crockett, & Passey, 1998; Simms, Watson, & Doebbeling, 2002).
PTSD Hyperarousal, Emotional Numbing, and Alexithymia

Previous psychological theories have proposed a temporal-sequence model to account for the otherwise paradoxical co-occurrence 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, which, in turn, temporarily inhibits the ability to experience 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. For example, Litz et al. (2000; see also M. W. Miller & Litz, 2004) posited that the general “capacity to experience and express a variety of emotions is unaltered in PTSD” (p. 27).

Other evidence, however, attests that a primary disturbance in emotional experience and expression may characterize certain complex cases of PTSD. In particular, an emerging number of studies have identified a positive association between PTSD symptoms and alexithymia (e.g., Badura, 2003; Cloitre, Scaravalone, & Difede, 1997; Fukunishi, Sasaki, Chishima, Anze, & Saio, 1996; Hyer, Woods, Summers, Boudswyns, & Harrison, 1990; Monson, Price, Rodriguez, Ripley, & Warner, 2004; Søndergaard & 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 observed in psychosomatic patients whereby these individuals were often unable to identify and label their emotional feeling states (see also Nemiah, Freyberger, & Sifneos, 1973; Nemiah & Sifneos, 1970; Sifneos, 1967; G. J. 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. That is, alexithymia in individuals with PTSD may signify an uncoupling of cognitive and emotional processing through 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 (G. J. Taylor et al., 1997). Consistent with the blindfeel hypothesis (Lane, Ahern, Schwartz, & Kasznik, 1997), individuals with alexithymia and PTSD may manifest physiological and behavioral profiles indicative of hyperemotionality of which they may not be consciously aware, thereby reporting that they either feel nothing at all or do not know what they feel.

Although a cursory overview of Litz’s (1992; Litz & Gray, 2002; Litz et al., 2000; 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. That is, according to the network theory, for individuals with PTSD “the building blocks of emotional experience . . . that were available to the individual before [he or she was] traumatized are intact, as is pretraumatic, elaborated emotional knowledge or schemas” (Litz et al., 2000, p. 27). In other words, the explanatory scope of the network model may parsimoniously encompass a relationship between intact emotional functioning pretrauma and the presentation of seemingly expansive emotional-processing deficits posttrauma. 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 et al. (2001) found that alexithymia levels were distinctly associated with reported emotional and physical neglect during childhood in a heterogeneous sample of psychiatric patients that included individuals with PTSD. It is therefore possible that emotional neglect and/or maltreatment that occurs 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 and 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 an individual 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 Pole, 2007) and demonstrate less activation in the anterior cingulate cortex (ACC; Brodmann’s areas [BA] 24, 25, 32) and medial prefrontal cortex (mPFC; BA 9, 10; 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; Ochsnr & Gross, 2005; Seminowicz et al., 2004; Steele & Lawrie, 2004). Accordingly, diminished response in the ventral ACC in the PTSD population may 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 et al., 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; e.g., Fox, Corbetta, Snyder, Vincenr, & Raichle, 2006; Gusnard, Akbudak, Shulman, & Raichle, 2001). 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 as well as 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 known to be involved in emotion regulation (Ochsner & Gross, 2005). The direction of these differences, however, 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.

A 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 the 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 been implicated in the pathophysiology of PTSD, the functional brain alterations underlying symptoms of alexithymia and PTSD may be related.

The Present Study

Accordingly, the present study examines the clinical and neural correlates of alexithymia in PTSD. Psychometrically defined individual differences in alexithymia were predicted to correlate positively with PTSD symptomatology, in particular reexperiencing, avoidance, emotional numbing, hyperarousal, 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 MRI (fMRI), alexithymia symptoms were predicted to be associated with activity in brain regions associated with emotional processing and control—the ventral ACC, ventromedial prefrontal cortex (PFC), PCC, right inferior frontal cortex, and insula.

Method

Participants

One hundred five individuals with a principal diagnosis of PTSD participated in an 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 Axis I Disorders—Research Version (SCID–I; First, Gibbon, Spitzer, & Williams, 1996).

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, 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, Canada). Individuals with a positive history of 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 (Ruth A. Lanius). 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% employed full or part time vs. 74% unemployed), $\chi^2(2, N = 150) = 22.87, p < .001$.

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). The selection criteria used to form this subsample included the inclusion criterion that participants had experienced a traumatic event related to a motor vehicle accident (MVA) and were right-handed. In three cases, however, participants indicated their primary traumatic event to be an incident other than the MVA: sexual assault experienced in childhood ($n = 1$), physical assault during childhood ($n = 1$), and physical assault occurring in adulthood ($n = 1$). Exclusionary criteria included self-reported involvement in litigation related to the MVA; history of substance use disorder in remission for less than 6 months; and presence of significant medical conditions, presence of neurological illness, or history of a significant head injury with loss of consciousness, as assessed by interview. 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 2 weeks prior to fMRI scanning (note that no participant was receiving fluoxetine prior to the drug washout).

Measures

Twenty-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 G. J. 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. Coefficient alpha for the TAS–20 in the current sample was .93, and the intraclass correlation coefficient (ICC) was .38.

CAPS (Blake et al., 1995). The CAPS assesses both the frequency and the intensity of each of the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; American Psychiatric Association, 1994) PTSD symptoms and is widely considered to be the gold standard in semistructured diagnostic interviews for PTSD. High CAPS scores indicate greater severity of PTSD. Coefficient alpha for the CAPS items (with frequency and intensity items summed) overall in the current sample was .97, and the ICC was .66. The four symptom clusters of the CAPS identified by King, Leskin, King, and Weathers (1998) were also scored separately (Reexperiencing, Effortful Avoidance, Emotional Numbing, and Hyperarousal). Coefficient alphas and ICCs for each CAPS
subscale in the present sample were as follows: Reexperiencing (α = .91, ICC = .69), Effortful Avoidance (α = .82, ICC = .70), Emotional Numbing (α = .92, ICC = .76), and Hyperarousal (α = .92, ICC = .69).

Dissociative Experiences Scale (DES; E. M. Bernstein & Putnam, 1986). The DES is a well-recognized 28-item measure of trait dissociative experiences. Coefficient alpha for the DES in the current sample was .94, and the ICC was .36.

Childhood Trauma Questionnaire—Short Form (CTQ–SF; D. P. Bernstein et al., 2003). The CTQ–SF is a standardized measure of individuals’ exposure to traumatic events during childhood and adolescence. It has five subscales: Emotional Neglect, Emotional Abuse, Sexual Abuse, Physical Abuse, and Physical Neglect. Coefficient alphas and ICCs for these subscales in the present sample were as follows: Emotional Neglect (α = .93, ICC = .74), Emotional Abuse (α = .93, ICC = .73), Sexual Abuse (α = .97, ICC = .87), Physical Abuse (α = .92, ICC = .69), and Physical Neglect (α = .80, ICC = .45).

Procedure

This study was approved by the Health Sciences Research Ethics Board of the University of Western Ontario. 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 an fMRI trauma script imagery study, as discussed above. These participants, following administration of the CAPS, provided detailed descriptions of the traumatic MVA on which the CAPS assessment was based in addition to a detailed description of a neutral memory that occurred 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-s scripts that were then audio recorded via the Windows Sound Recorder tool. In keeping with 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 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 script as soon as it 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, throughout the duration of the script, and during the 30-s period of silence that immediately followed its presentation. An additional...
120 s 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 (RSDI), an 11-item measure of PTSD reexperiencing, avoidance, and dissociative symptoms prompted by trauma script-driven imagery (Hopper, Frewen, Sacks, Lanius, & van der Kolk, in press). Participants answered questions by giving a number from 0 (not at all) to 6 (a great deal). Note that the explicit neural correlates of this measure have been investigated and reported in another article (Hopper, Frewen, van der Kolk, & Lanius, in press).

All imaging data were acquired on a 4-Tesla Varian (Palo Alto, CA) UNITYINOVA whole-body MRI system equipped with Siemens Sonata (Erlangen, Germany) actively shielded gradient coils. A cylindrical transmit–receive hybrid birdcage radio frequency head coil was used for transmission and detection of signal. The participant's head was immobilized with foam padding and a Plexiglas head cradle within the head coil.

Preliminary T₁-weighted sagittal images were acquired via a fast low-angle shot inversion-recovery sequence (128 × 128 matrix size, field of view = 28 cm, inversion time = 750 ms, echo time = 3.5 ms, repetition time = 8 ms, tip angle = 11°), which provided excellent gray–white matter contrast. From these localizer images, 12 contiguous functional planes were prescribed with an axial orientation approximately parallel to the anterior commissure–posterior commissure line (centered on a plane level with the ACC) 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 via a navigator-corrected four-segment echo planar imaging sequence (128 × 128 matrix size, field of view = 22 cm, echo time = 10 ms, repetition time = 1,250 ms, flip angle = 40°, 108 volumes, volume collection time = 5 s). For registration of the BOLD-sensitive images, a high-resolution T₁-weighted anatomic reference volume was acquired with the same axial field of view via a three-dimensional fast low-angle shot sequence (256 × 256 × 64 matrix size, slice thickness = 3 mm, inversion time = 600 ms, echo time = 5.5 ms, repetition time = 10 ms, flip angle = 11°).

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 conducted according to the method of Meng, Rosenthal, and Rubin (1992).

Statistical analysis of the MRI data employed voxelwise general linear models with design matrices composed of epoch-related regressors. Baseline brain activation (i.e., the “implicit” baseline) was calculated on the basis of the average activation patterns 60 s 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 on the basis of average activation patterns that occurred during the silent 30 s 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 with Statistical Parametric Mapping (Wellcome Department of Imaging Neuroscience, 2007). 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 a cluster-size extent (referred to hereafter as k) of at least 50 voxels and two-tailed alpha less than or equal to .05, corresponding to a minimum absolute value correlation greater than or equal to .39 with 24 degrees of freedom (uncorrected for multiple comparisons). The voxel within each cluster that was observed to exhibit the strongest correlation with TAS–20 scores was then reported only if (a) the voxel survived correction for multiple comparisons (small volume corrected [SVC] with p < .05—referred to hereafter as pSVC—for false discovery rate) within a 5-mm spherical search volume centered at the identified voxel and (b) the cluster within which the voxel was identified had an extent threshold large enough to occur with p ≤ .05 within the search volume encompassed by the region of interest, as defined independently by the pick atlas provided by Statistical Parametric Mapping.

The three-dimensional (x, y, z) coordinates of these voxels are reported within Montreal Neurological Institute (MNI) space. In this system, the coordinate values refer to the location in millimeters of the voxel within the brain relative to the anterior commissure–posterior commissure line (increasing values indicative of increasing distance). The three-dimensional coordinates define the location of the voxel at the intersection of three planes: the x value refers to the location of the voxel at the midsagittal plane (left = negative, right = positive), the y value refers to the location at the transverse plane (superior = positive, inferior = negative), and the z value refers to the location at the coronal plane (anterior = positive, posterior = negative). Clusters of voxels that met the above criteria that were observed in the stated a priori regions of interest (ACC, PCC, mPFC, insula, inferior frontal cortex), as independently defined by the Statistical Parametric Mapping pick atlas and confirmed objectively against the atlas of Talairach and Tournoux (1988), were accepted as statistically significant. Note that the locations of peak activation reported in this article approximate those found to associate functional brain responses with PTSD or alexithymia in previous studies of the ventral ACC–mPFC (Lanius et al., 2001), right inferior frontal cortex (Lanius et al., 2002), ventral PCC (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 among alexithymia, PTSD, and dissociative symptoms.** The observed correlations between TAS–20 scores and the CAPS total and subscale scores in the PTSD sample were as follows: total ($r = .45$, $p < .001$), Reexperiencing ($r = .32$, $p < .001$), Effortful Avoidance ($r = .09$, $p = .19$), Emotional Numbing ($r = .43$, $p < .001$), and Hyperarousal ($r = .34$, $p < .001$). TAS–20 scores were less strongly 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 Reexperiencing ($z = 2.39$, $p < .05$). The observed correlation between TAS–20 and DES scores in the PTSD sample was .39 ($p < .001$).

**Association between alexithymia and childhood abuse and neglect.** TAS–20 scores were 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 strongly 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 were trends that 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 reexperiencing symptoms in response to the trauma script-driven imagery paradigm, as measured by the RSDI. For example, 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 item “Were you distressed?” and 45% indicated 3 or higher in response to the item “Were you emotionally upset?”

**Association between alexithymia and BOLD-fMRI response to trauma script imagery.** Two distinct clusters were observed in which a greater BOLD signal in the trauma script minus neutral script condition was associated with higher levels of alexithymia within the a priori regions of interest (please see Figure 1 for illustration): the right posterior insula ($k = 78$; maximum at MNI $176$ FREEWEN ET AL.}

**Figure 1.** Positive correlation between alexithymia and brain activation during trauma script-driven imagery ($p < .05$; $k \geq 50$; $n = 26$). The images are presented in neurological convention (left = left). Participants’ Twenty-Item Toronto Alexithymia Scale scores were regressed on their images of the contrast of trauma script imagery minus neutral script imagery in a random effects model ($df = 24$). The color bar denotes the $t$ statistic of the significance of the correlation; images present cluster activations with $t(24) \geq 2.06$ corresponding to $r(24) \geq .39$. STC = superior temporal cortex; BA = Brodmann’s area; vPCC = ventral posterior cingulate cortex.
46 to 22; \( r = .55, p_{SVC} = .027 \), and the right ventral PCC (\( k = 105 \); maximum at MNI 8 to 22; \( r = .54, p_{SVC} = .029 \)). Higher levels of alexithymia were also associated with a greater BOLD signal in a cluster within the left superior temporal cortex (\( k = 116 \); maximum at MNI -56 to 10; \( r = .55, p_{SVC} = .011 \)), although this was not predicted a priori.

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

**Discussion**

The present results indicate that certain traumatized individuals reported lacking the ability to reflect on, understand, and modulate their affective symptoms. Furthermore, these subjective reports were 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.), who was unaware of the specific hypotheses under investigation, gave several lucid descriptions of this phenomenon. In the context of the CAPS administration, when asked 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 those of W.H. help bring to life the clinical

![Figure 2](image-url)
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 results. It is interesting that alexithymia symptoms were associated most strongly with emotional numbing symptoms, whereas alexithymia was not associated with symptoms of effortful avoidance, which partially confirms 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 develop when a caretaker fails to teach a child how to differentiate among distinctive emotional states, regulate arousal, and respond adaptively to challenging life events.

Moreover, during traumatic memory recall imagery, relative 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 the right insular cortex might 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, Wiens, Rotchtein, Ohman, & Dolan, 2004; Ochsner & Gross, 2005). Additionally, left anterior insula activation was correlated negatively with alexithymia scores, which was predicted from Craig’s (2005) model, which proposes a hemispheric specificity in the functional activation of the 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, which suggests 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 G. J. 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, Vogt, and Laureys’s (2006) demonstration of the normally coordinated involvement of the ventral PCC and ACC in ongoing self-monitoring and the assessment of the emotional significance of events. 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 it is 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 occurrences (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 that employed in exposure-based therapies (Becker & Zayfert, 2001; Cloitre, Koenen, Cohen, & Han, 2002). In particular, 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 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 is that only a single self-report instrument of alexithymia was investigated, and therefore these results require replication with additional measures.

Moreover, the construct of alexithymia, including the TAS–20 measure of it, is currently not without controversy. For example, it has been argued that a valid self-report assessment of alexithymia (and of constructs related to emotional intelligence more generally) may require a level of emotional self-awareness that should not, by definition, be available to individuals who are alexithymic (e.g., Lane & Taitano, 2003; Mathews, Zeidner, & Roberts, 2002; Mayer, Curuso, & Salovey, 2000). Thus, a multimethod approach to the assessment of alexithymia is advised for future studies. Moreover, that alexithymia measures are often correlated with neuroticism and negative affect raises concern that associations with alexithymia may primarily reflect the effects of neuroticism and negative affect (Lane & Taitano, 2003). In addition, others have argued that increased negative affect in alexithymic individuals is a natural consequence of their inability to self-regulate emotion (Lumley, 2000). Accordingly, statistically controlling for negative affect in tests of the effects of alexithymia may render the latter uninterpretable. Continued research into the specificity, generalizability, and direction of causality of the various associations identified in this study is therefore crucial for a better understanding of the disturbances in emotional processing that accompany PTSD. 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, as well as the extent to which the present results relate specifically to alexithymia, rather than dimensions...
with which it is correlated, remain open theoretical questions that warrant further critical study.

In conclusion, the present studies reveal an association between perceived difficulty in identifying and describing emotional states, on the one hand, and severity of PTSD symptoms, dissociation, and retrospectively reported childhood emotional neglect, on the other. 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.

References


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