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Review article

Alexithymia in post-traumatic stress disorder is not just emotion numbing: Systematic review of neural evidence and clinical implications

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1. Introduction

Posttraumatic stress disorder (PTSD) is a heterogeneous disorder that develops after exposure to a traumatic event such as threatened death, injury or sexual assault (American Psychiatric Association, 2013). According to the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5; American Psychiatric Association, 2013), the signs and symptoms of PTSD span four symptom clusters: (1) intrusive symptoms or re-experiencing of the traumatic event, (2) avoidance of reminders that are associated with the traumatic event, (3) hyperarousal and (4) negative cognitions about oneself, the world and others.

A prevailing model of PTSD is that the disorder results and is perpetuated by impaired fear extinction (Mineka and Oehlberg, 2008; Pitman et al., 2012; Shin and Liberzon, 2010). This is based on Pavlov's (1927) work in classical conditioning, where a learned association between a previously neutral stimulus (conditioned stimulus; CS) and an aversive stimulus (unconditioned stimulus; US), leads to heightened US-expectancy of the CS. After learning, the CS alone generates a conditioned fear response in absence of the US. This principle operates when people experience a traumatic event. For example, an individual involved in an armed robbery (US) is likely to experience a natural response of fear and heightened physiological arousal (the unconditioned response; UCR). Following the robbery, aspects of the trauma that were not associated with the robbery before (e.g., a black hooded sweatshirt), may act as a trigger to activate the trauma memory, eliciting the CR. This has subsequently led to the development of trauma-focused treatments aimed at exposing patients to benign trauma cues and memories in order to facilitate extinction learning via emotional processing of conditioned stimuli and responses. Critical to the success of these interventions are emotion engagement and processing of the traumatic event (Foa and Kozak, 1986).

Emotional processing theory developed by Foa and Kozak (1986) has informed the conceptualization and development of Prolonged Exposure (PE) therapy, the gold standard, trauma-focused treatment for PTSD. The theory is based on Lang's bio-informational theory of fear (1977; 1979) where fear is represented in memory as structures that are made up of related: stimulus, response and meaning elements designed as a program to escape danger. Engagement with the trauma memory is indexed by increased objective and subjective distress. The aim is for 'optimal' engagement with the trauma memory (as indexed by change in physiological arousal), there should be enough engagement for the individual to 'recreate' the triggering event but not too much so that new learning cannot occur (Rauch and Foa, 2006). Given the noted efficacy of trauma-focused treatments (Bisson et al., 2019), it is curious that so many individuals continue to regularly experience symptoms following evidenced based treatments (Santiago et al., 2013). One potential reason for the variation in responses to trauma-focused treatment could be the high comorbidity with alexithymia. Alexithymia, or the lack of emotion awareness (Taylor et al., 1997), may interfere with the mechanisms at play in trauma-focused therapy, rendering them less effective. However, it is unclear whether alexithymia is a unique condition or is merely an expression of the emotional numbing commonly observed in PTSD (Declerq, Vanheule and Deheegher, 2010;

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Eichhorn et al., 2014; Sopp et al., 2018). By distinguishing between alexithymia and emotional numbing, it may help to increase specificity of clinical formulation and subsequent treatment planning.

1.1. Relation of alexithymia to ptsd

Alexithymia is a sub-clinical, dimensional construct involving deficits in emotion engagement, defined as (1) a difficulty in identifying feelings; (2) a difficulty in describing feelings to other people and (3) externally orientated thinking (Bagby & Taylor, 1997). These aspects of deficient emotion processing vary considerably between individuals but remain stable across time (Bagby et al., 1994; Larsson et al., 2010). Alexithymia may be a vulnerability to trauma-focused treatment resistance as those with alexithymia showing hypo-arousal to emotional stimuli, coupled with a poorer habituation to emotional stimuli (Panayiotou & Constantinou, 2017). Therefore not only potentially impeding, engagement and recognition of trauma-memory activation, but also impeding the process of habituation/ fear extinction to these stimuli.

One meta-analysis found that 16% of individuals with PTSD experience symptoms of alexithymia (Frewen et al., 2008). Other work suggests that these rates are even higher in combat veterans: 43.4% of veterans with PTSD exhibited high alexithymia (Becirovic et al., al.,2017), compared to 10.9% of veterans without PTSD (rates comparable to those of the general population; Franz et al., 2008; Mattila et al., 2006). Alexithymia has also been found to be a predictor of PTSD subsequent to a traumatic event (Zahradnik et al., 2009; McCaslin et al., 2006). Higher levels of alexithymia are also linked to greater PTSD severity (Frewen et al., 2008).

Measuring alexithymia is inherently difficult with common approaches requiring individuals with deficits in emotional awareness to rate their lack of awareness. Such meta-cognitive skills can be elusive and those individuals with the greatest levels of alexithymia are potentially the least aware of the nature of the problem. Measures such as the Toronto Alexithymia Scale (TAS-20; Bagby et al., 1994) have shown good psychometric properties and clinical utility for over two decades (Bagby et al., 2020). However, it is difficult to examine subjective experience objectively (Barrett, 2012). Despite the fact that different cognitive and affective states can evoke activity in the same brain areas and that the same emotion can evoke activity in different brain areas (Lindquist et al., 2012), there is evidence of some network-level localization of emotion in the brain (Wager et al., 2015). As such, it would seem that exploring brain activation is one useful way of trying to disentangle different emotional states with the explicit recognition of the aforementioned limitations. One might reasonably anticipate differential patterns of brain activity associated with alterations to the emotional experiences (emotional numbing) and the awareness of those emotional experiences (alexithymia), if they are distinguishable.

One critical question is whether alexithymia is distinct from psychopathology or is rather a characteristic of certain types of psychopathology (cf. Bagby et al., 2020). A number of studies have examined the functional neural correlates of alexithymia in the context of emotion-processing paradigms and suggest some overlap with PTSD-related emotional numbing. These studies have demonstrated that the presentation of salient emotional stimuli to those with alexithymia resulted in a decreased responsiveness of: (1) the anterior cingulate cortex (ACC; Kano et al., 2003), (2) the ventral medial prefrontal cortex (vmPFC) and dorsal medial prefrontal cortex (dmPFC; Berthoz et al., 2002; Kano et al., 2003), (3) the right insula (Kano et al., 2003), and (4) the posterior cingulate cortex (PCC; Mantani, Okamoto, Shirao, Okada & Yamawaki, 2005). The ACC has been associated with affective, autonomic and attentional control (Critchley et al., 2005). Altered activation patterns in the ACC associated with alexithymia may also explain why individuals with PTSD are unable to modulate the intensity of their emotional reactions in the presence of trauma reminders (Frewen and Lanius, 2006). The vMPFC and dmPFC have been implicated in self-referential emotion-processing (Gilbert et al., 2006) and may explain exaggerated deviations from baseline self-referential processing observed in those with PTSD (American Psychiatric Association, 2013). The right insula is associated with awareness of self and bodily sensations (Craig, 2009); increased activation has been found to be associated with higher self-reported flashback intensity during script-driven imagery in PTSD (Ousch et al., 2001). The PCC is a major cortical hub, reflecting activity of the default mode network but also seems to play an important role in shifting attention internally vs. externally (Leech and Sharp, 2014). The PCC exhibits increased activation during episodic memory retrieval (Nielsen et al., 2005), which may explain abnormal processes in PTSD such as flashbacks, intrusive thoughts and nightmares.

1.2. Emotion numbing and alexithymia in ptsd

A number of individuals contend that the presentation of alexithymia among individuals with PTSD is best explained by emotion numbing (Declerq, Vanheule and Deheegher, 2010; Eichhorn et al., 2014; Sopp et al., 2018). Although PTSD is most commonly associated with heightened fear reactions (corresponding to a hypoactive vmPFC and hyperactive amygdala, Hayes et al., 2012; Rauch et al., 2006), some individuals with PTSD also report restrictions in their capacity to feel, also known as emotion numbing (American Psychiatric Association).

Neuroimaging studies show that PTSD-related emotion numbing symptoms have been associated with less arousal as measured by self-report (Spahic-Mihajlovic et al., 2005) as well as attenuated activation in temporal and parahippocampal brain regions when exposed to pleasant images (Jatzko et al., 2006). Relatively less activation in ventral striatum to positive feedback in decision-making tasks has also been observed (Sailer et al., 2008) among those with emotional numbing symptomology. Furthermore, there is evidence that individuals with PTSD who disengage from the trauma memory during trauma script recall display increased activation in the inferior frontal gyrus, mPFC and ACC relative to controls. These neural correlates are consistent with an over-suppression of emotional arousal (Frewen and Lanius, 2006) and are posited to be linked to PTSD-related emotion numbing experiences (Lanius et al., 2002), as emotional numbing is a key symptom within dissociative PTSD. Taken together, these findings suggest that extreme responses to emotional stimuli (both exaggerated/ PTSD-related hypervigilence and attenuated/ PTSD-related emotion numbing) are common among PTSD. However, ongoing debate exists among clinicians and within the literature as to whether alexithymia and emotion numbing are fundamentally separate constructs or manifestations of the same posttraumatic emotion-processing deficits (see e.g., Declerq, Vanheule and Deheegher, 2010; Eichhorn et al., 2014; Sopp et al., 2018).

1.3. Rationale and objectives

Given a general lack of emotional awareness and insight that defines alexithymia, it may be contraindicated to rely on self-reports measures to assess emotional processes among those with elevated alexithymia. Furthermore, some have posited that the TAS-20 may also be significantly correlated to negative affective states (depression and anxiety) due to the presence of methodological artefacts as a result of the self-report nature of the TAS-20 or a general susceptibility of the TAS-20 to a negativity response bias (Marchesiet al., 2013). Despite difficulties in assessment, there is compelling evidence of clinical overlap between emotion numbing and alexithymia as well as substantial brain imaging literature in both these conditions. To our knowledge there is no previous review contrasting the neural correlates of these two constructs. Given the need to identify via objective measurement whether alexithymia and emotional numbing are overlapping or different experiences, a review of neural circuitry underlying affective processing

involved in these conditions is warranted to guide selection of appropriate intervention. This review examined the neural correlates of PTSD with alexithymia and PTSD with emotion numbing symptoms. Specifically, the aim was to explore the spatial neural correlates of PTSD with alexithymia and PTSD with emotion numbing while participants were engaged in affective tasks.

2. Method

2.1. Study identification

This review followed PRISMA guidelines (Moher et al., 2009) and a step-wise procedure was used to identify neuroimaging studies of: (1) alexithymia and PTSD, and (2) PTSD with emotion numbing symptoms. Articles were searched on PubMed and Web of Science published before the 9th of February 2020. Identified articles were also scanned for additionally relevant studies. Two sets of search terms were included in the first component of the review: (1) ["alex*"] AND [PTSD OR posttraum*] AND [" PET" OR "BOLD-fMRI" OR " fMRI" OR "fPET" OR " functional AND imaging" OR "positron" OR "neuro*" OR "imaging" OR "magnetic resonance imaging" OR "MRI" OR "cortical thickness" OR "volume" OR "morphometry" OR "VBM"]; and (2) [PTSD OR posttraum*] AND [emo* numb*] AND [" PET" OR "BOLD-fMRI" OR " fMRI" OR "fPET" OR " functional AND imaging" OR "positron" OR "neuro*" OR "imaging" OR "magnetic resonance imaging" OR "MRI" OR "cortical thickness" OR "volume" OR "morphometry" OR "VBM"]. A total of 442 (132 of alexithymia and PTSD and 310 PTSD and emotion numbing) potential articles of interest were identified. After removing duplicates between the databases, articles were assessed by reviewing their titles and abstracts for the following inclusion criteria: (1) written in the English language; (2) reported empirical results; (3) made use of functional neuroimaging; and (4) included human subjects. Studies meeting these criteria were selected for full-text review and were included in the review if they also met the following criteria: (5) investigated functional neural activation or connectivity among participants with PTSD and alexithymia or emotion numbing symptomology specifically; and (6) assessed alexithymia using the Toronto Alexithymia Scale (TAS-20) or the cognitive dimension of the Bermond-Vorst Alexithymia Questionnaire (BVAQ; Shields et al., 1989) or specifically assessed emotion numbing symptoms of PTSD.

Fig. 1,2

2.2. Data extraction

For each study, we extracted the following data: (1) study ID (first author and publication year); (2) sample size; (3) mean age of subjects; (4) whether a control group was utilized; (5) imaging method; (6) PTSD/ emotion numbing/ alexithymia measures; (7) in-scanner task; and (8) Results: Main effect between or within group-activation differences. A schematic representation of reported MNI coordinates is presented I Fig. 3.

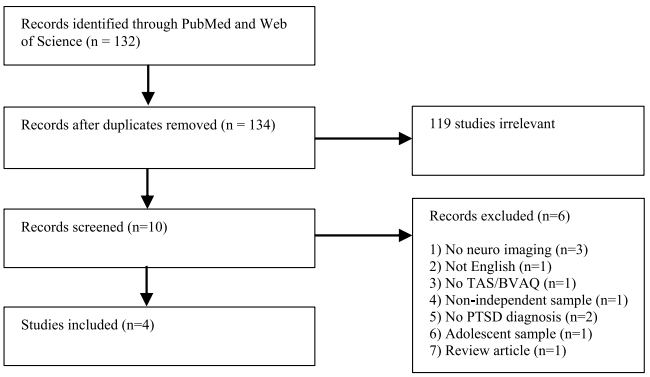

Fig. 1. Data extraction process for Alexithymia and PTSD.

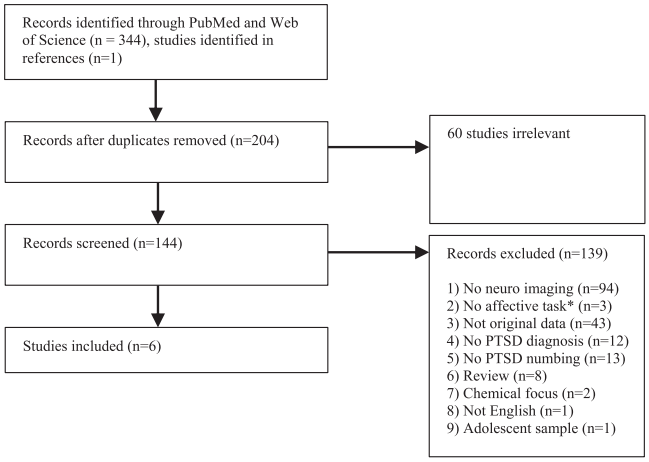

Fig. 2. Data extraction process for PTSD with emotion numbing.

3. Results

Ten studies were included in this review (four featuring alexithymia; six featuring emotion numbing) spanning the years 2005 – 2015. Tables 1 and 2 summarize the main characteristics of each study in terms of methodology, sample and results.

3.1. Methodology

Studies differed substantially in terms of the methodology and imaging analysis method used. Most studies (8/10) presented findings from whole brain analyses, but two focused on specific regions of interest (Elman et al., 2009; Felmingham et al., 2014). The majority of the fMRI/PET studies examining alexithymia correlates employed script-driven tasks (3/4), studies examining emotion numbing utilized affective priming tasks (2/6), passive viewing tasks (2/6), emotion imagery tasks (1/6) and a wheel-of-fortune game (1/6). All but one study (Frewen et al., 2008) included control comparisons.

3.2. The functional neural circuitry of alexithymia

Three of the four studies utilized healthy control comparisons, with Britton et al. (2005) also including combat veterans without PTSD and age-matched non-combat controls. Reviewed studies showed increased activations in the pregenual and subgenual ACC, Mid-Cingulate and supplementary motor area (SMA), and thalamus. This suggests that alexithymia is predominately associated with alterations in (cortical-only) Frontoparietal Control-, Ventral Attention- and to a lesser extent the Default Mode-Network(s) (FCN; VAN; DMN; Yeo, 2011). Fig. 3 presents a schematic summary of the neural activation results of the reviewed studies.

3.3. The functional neural circuitry of emotion numbing

All of the extracted studies included healthy control comparisons. Reviewed studies show altered activations in the mPFC and key limbic and brainstem regions (e.g. striatum, amygdala and culmen). This suggests the PTSD-related emotion numbing is primarily associated with alterations in the salience network (specifically the fear circuitry and reward networks; as per Yeo, 2011; cortical-only).

3.4. Similarities and differences in alexithymia and emotion numbing

Like PTSD with alexithymia, PTSD-related emotion numbing was

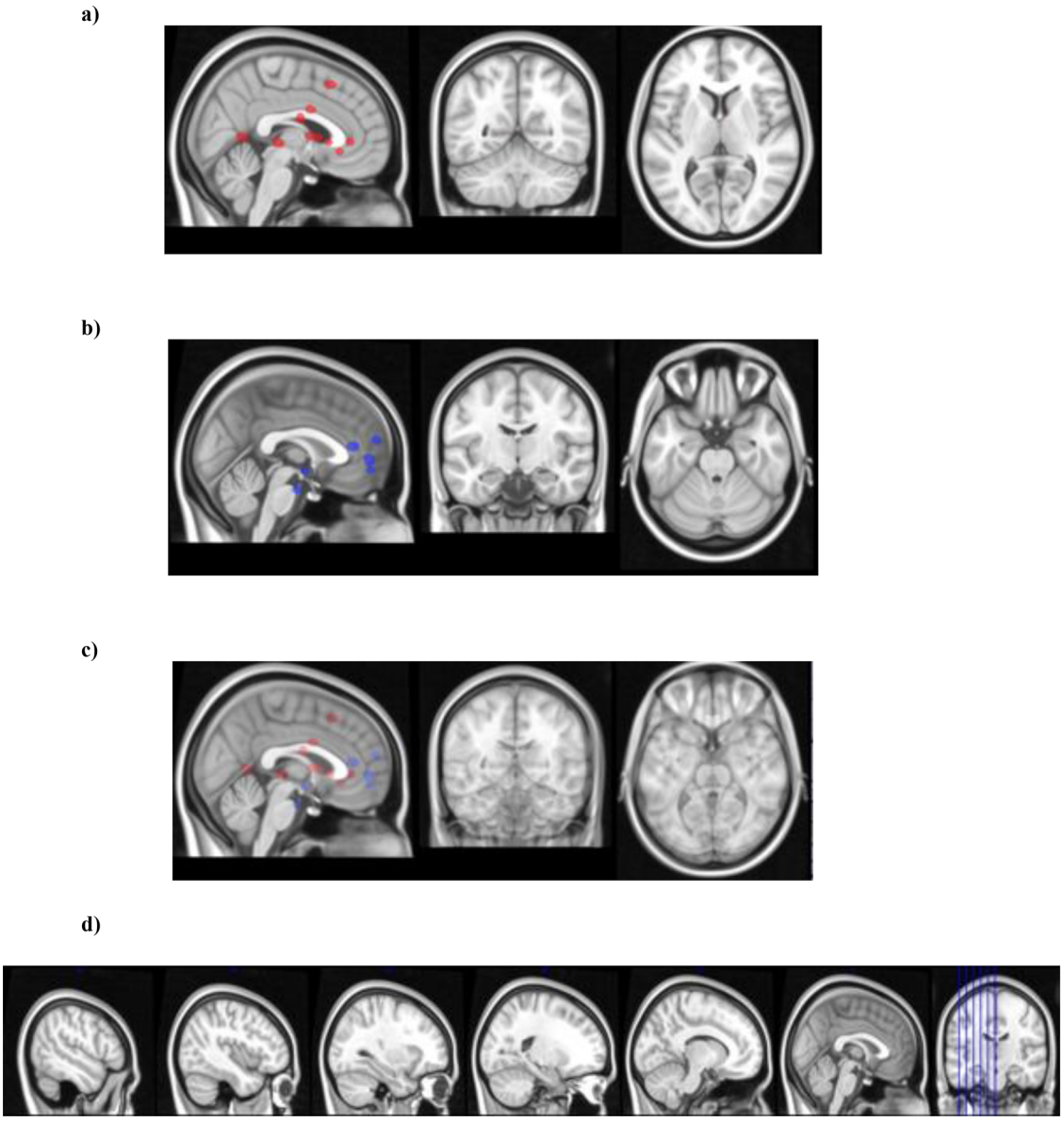

Fig. 3. (a) Brain regions reported to be involved in processing of salient emotional stimuli in PTSD and alexithymia. (b) Brain regions reported to be involved in processing of salient emotional stimuli in PTSD related emotion-numbing symptomology. (c) Overlay of (a) and (b) to display differentiation and overlap between regions. (d) multi slice representation of (c). Note: Laterality of brain regions was not taken into account to illustrate results. Peak activation reported by studies has been shifted in some cases to enable illustration of all results.

found to be associated with increased activations of the insula and inferior frontal gyrus when processing salient emotional stimuli. This suggests some slight overlap in activation of the DMN and VAN. However the main differences between alexithymia and emotion numbing were found in alterations in reward network regions (e.g. striatum and culmen), with decreased activation in these areas found among those with PTSD-related emotion numbing. Furthermore PTSD with alexithymia appears to display unique increased activations in the FCN.

4. Discussion

This review examined the neural correlates of PTSD with alexithymia and PTSD with emotion numbing symptoms. Specifically, the aim was to explore the spatial neural correlates of PTSD with alexithymia and PTSD with emotion numbing while participants were engaged in affective tasks. Our results suggest that alexithymia is linked to alterations in the Frontoparietal Control Network (FCN), Ventral Attention Network (VAN) and Default Mode Network (DMN) while PTSD-related emotion numbing appears to be primarily linked to alterations in fear and reward pathways of the salience network with some dysregulation in the DMN. Conversely, our results suggest that emotion numbing may be linked to disorder-level negative affect while alexithymia is linked to alterations in emotional meta-cognition and attentional control, providing evidence that these constructs are at least partially distinct in the brain.

4.1. Common and distinct neural circuitry underlying ptsd with alexithymia

During script imagery tasks, individuals with PTSD and alexithymia demonstrated increased activation in the insula while demonstrating a diminished activation of the mPFC and ACC. The insula cortex is known to contribute to diverse mental functions such as motor control, interoception awareness and emotion. The role of the insula in emotion is broad, and the function of the insula have been linked to the experience of a number of positive and negative emotions. The insula is known to

Table 1
Alexithymia Studies Included in Review.

Study	Population Mean (SD)	Imaging Method	Analysis	Type of Paradigm	Results	Scales
Britton et al., 2005	16 combat veterans with PTSD age: 49.4 (7.9) 15 combat veterans without PTSD age: 56.5 (4.9) 14 age-matched noncombat controls age: 53.8 (4.2)	PET	**Whole Brain**	Script driven imagery task and neutral autobiographic events	Traumatic/ stressful versus neutral comparison - **PTSD:** Increased activation in the insula, less activation in the medial frontal cortex (mPFC) and rostral anterior cingulate cortex (rACC). **Combat controls:** less activation in left amygdala, increased activity in the insula, less activity in the mPFC **Controls:** increased activation in the left amygdala, increased activation in the insula and mPFC	CAPS TAS-20
Mazza et al., 2015	7 PTSD post L'Aquila earthquake age: 34 (9.4) 10 healthy controls who experienced L'Aquila earthquake age: 30.00 (8.2)	fMRI	**Whole Brain**	Multifaceted Empathy Test	**PTSD versus controls**: **Implicit emotional empathy task**- greater activation in the left palladium and right insula (contrast: implicit dimension – neutral B vs cognitive – neutral A); control versus PTSD: greater activation in the inferior frontal gyrus (contrast: implicit emotional – neutral B vs explicit emotional – neutral C) **Explicit emotional empathy task -** less activation in the left insula (contrast: explicit emotional – neutral C vs cognitive – neutral A) and left inferior frontal gyrus. **Cognitive empathy task** - greater activation of the right medial frontal gyrus (contrast: cognitive -neutral A vs implicit emotional – neutral B) and left inferior frontal gyrus (contrast: cognitive – neutral A vs neutral C).	CAPS; TAS-20
Frewen et al., 2006	26 people with PTSD age: not reported 16 healthy controls age: not reported	fMRI	**ROI**	Trauma script imagery task	TAS Scores correlated with - **Controls:** increased bilateral mPFC activation and negative left thalamus and bilateral anterior cingulate cortex (ACC) activation. **PTSD:** increased activation of the bilateral thalamus, insula and posterior cingulate cortex (PCC) and decreased activation of the ACC.	CAPS TAS-20
Frewen et al., 2008	26 PTSD post motor vehicle accident age: 35.95 (13.5)	fMRI	**Whole Brain**	Trauma-Script Imagery Task	Trauma script v neutral – **High alexithymia**: increased activation in the right posterior insula; right ventral PCC; left superior temporal cortex. **Low alexithymia:** increased activation of the bilateral ventral ACC and mPFC; right inferior frontal cortex extending into the right anterior insula and left anterior insula.	CAPS; TAS-20

Table 2
Emotion Numbing Studies Included in Review.

Study	PopulationMean (SD)	Imaging Method	Analysis	Type of Paradigm	Results	Scales
Elman et al., 2009	20 people with PTSD age: 33.0 (10.5) 26 healthy controls 28 (8.2)	fMRI	**ROI**	Wheel-of-fortune-type game	**Gains contrasted to losses:** PTSD subjects showed less bilateral striatal activation compared to controls.	CAPS
Jatzko et al., 2006	10 people with PTSD age: 50 (13) 10 healthy controls	fMRI	**Whole Brain**	Viewing a sequence of well-known Walt Disney cartoons known to elicit positively valanced emotions	**PTSD** > **controls** – increased activity in right precentral, right superior frontal and right posterior middle temporal regions; Controls > PTSD – increased activity in left parahippocampal/fusiform gyrus, right temporal pole/ right superior temporal region and left temporal pole/ superior frontal regions.	CAPS
Catalucci et al., 2011	10 people with PTSD age: 33.9 (14.6) 10 healthy controls age: 27.1 (10.4)	fMRI	**Whole Brain**	Affective priming task	**PTSD** > **Controls** left inferior frontal gyrus and right insula; **Controls** > **PTSD** left middle frontal gyrus and right superior frontal gyrus.	CAPS
Felmingham et al., 2014	23 people with PTSD age: 38.5 (11.5) 20 trauma exposed controls age: 30.5 (12)	fMRI	**ROI**	Passive viewing task – rating happy faces	**PTSD** (severity of emotion numbing symptoms) was associated with decreased activity in the right ventral striatum and culmen (controlling for depression and PTSD severity)	CAPS
Frewen et al., 2012	14 people with PTSD age: 37.2 (7) 10 healthy controls	fMRI	**Whole Brain**	Emotion imagery task	**PTSD (emotion numbing)** was associated with decreased BOLD response in the dmPFC during imagery of positive social script and negative social scripts;	CAPS TAS-20
Mazza et al., 2012	10 people with PTSD age: 27.1 (20.4) 10 healthy controls matched for age, sex and education age: 33.9 (14.6)	fMRI	**Whole Brain**	Affective priming task	**PTSD** > **Controls** showed grater activation in the left amygdala and right insula; **Control** > **PTSD** showed greater activation in the left lingual gyrus Whole sample (emotion numbing symptoms) greater activation in the amygdala and insula activation while observing negative pictures; and less activation in the lingual gyrus.	CAP Empathy Quotients

play a role in representing emotional states, especially in relation to processing subjective feelings, empathy and uncertainty (Singer et al., 2009). Furthermore, the insular cortex has been shown to be involved in the experience of interoceptive awareness (Craig, 2009). This may explain the findings that those with alexithymia demonstrate atypical interoceptive ability (Longarzo et al., 2015; Nakao et al., 2002).

Interestingly, findings that those with PTSD and alexithymia demonstrated increased activation of the PCC is somewhat contrary to established literature (Mantani et a., 2005). Although shown to be highly heterogenous, the posterior cingulate cortex has a central role in supporting internally-directed cognition, regulating the focus of attention. It is a key node in the default mode network and shows increased activity when individuals retrieve autobiographical memories or plan for the future, as well as when activity in the brain is not engaged in goal-directed behavior. In addition, its activity varies with arousal state and its interactions with other brain networks may be important for conscious awareness (Leech and Sharp, 2014). The PCC also has a strong reciprocal connection to the ACC (Vogt et al., 2006). However Frewen et al. (2008) found that alexithymia scores were positively associated with activation in the PCC but negatively associated with the activation in the ACC and vmPFC indicating a decoupling of the normally integrated emotion processing circuit linked to self-monitoring and the assessment of the emotion significance of events. Taken together this suggests that alexithymia may be a result of alterations in FCN, VAN and DMN which are linked to meta-cognition, executive control (guiding goal-driven behavior) and orienting attention towards processing of unexpected salient stimuli (Dixon et al., 2018; Jia et al., 2019). This may explain dysfunctions in emotion processing which can lead to aberrant allocation of attentional resources and consequently to diminish goal-relevant cognitive capabilities e.g. limited perspective taking and an externally oriented thinking style.

4.2. Common and distinct neural circuitry underlying emotion numbing

Like PTSD with alexithymia, PTSD-related emotion numbing was found to be associated with increased activations of the insula when processing salient emotional stimuli. However, PTSD-related emotion numbing appears to be primarily linked to alterations in the salience/reward network. Specifically, PTSD-related emotion numbing was found to be uniquely associated with an increased activation of the amygdala when processing negatively valanced stimuli and reward correlates (striatum and culmen) when presented with positive stimuli. This is consistent with numerous studies implicating amygdala hyperactivity in PTSD using a variety of trauma-related stimuli (Felmingham et al., 2010; Protopopescu et al., 2005; Shin et al., 2005) and non-trauma related stimuli (Felmingham et al., 2010). This is consistent with the “fear circuitry” model of PTSD which posits that the amygdala and insula are hyperresponsive thereby increasing fear and anxiety responses. Interestingly findings by Felmingham and colleagues found decreased activation of amygdala when viewing happy faces. This suggests that those with PTSD-related emotion numbing may have a higher response threshold to positively valanced stimuli and higher threshold when responding to positive stimuli. This could account for the increased negative affect and anhedonia correlated to PTSD-related emotion-numbing (Eskelund et al., 2018; Pietrzak et al., 2015).

4.3. Clinical implications

Although similar in clinical presentations, the nuances in underlying mechanisms and deficits between PTSD with alexithymia vs PTSD with emotion numbing may call for differential formulation and subsequent treatment planning. Emotion numbing patients may be able to identify and communicate negative affective experiences, therefore PTSD-related emotion numbing may be best conceptualised as an avoidance response ‘protecting’ patients from overwhelming negative affect. These patients may therefore benefit from some preparatory work to enable them to tolerate overwhelming affect to limit numbing during trauma-focused interventions. One such program, the Skills Training in Affective and Interpersonal Regulation (STAIR; Cloitre, Heffernan, Cohen & Alexander, 2001) protocol, implements a phase-based, sequential approach which aims to first build emotion regulation skills as a method of improving engagement with and response to trauma-focused treatment.

On the other hand, those with alexithymia may present as generally detached, cold or aloof and may even be judged as being avoidant during treatment, eliciting negative evaluations from their therapist (Ogrodniczuk et al., 2011). Furthermore, those with alexithymia show hypo arousal and poor habituation to fear cues (Panayiotou & Constantinou, 2017), suggesting that they may be likely to underengage with- or not be able to index when a trauma-memory has been activated. Therefore, PTSD patients with alexithymia may not be able to adequately engage in trauma-focused PTSD treatments such as PE which calls for optimal exposure with emotion-related stimuli, and it is consequently unsurprising that alexithymia has been implicated as a predictor of PTSD treatment resistance (Kosten et al., 1992). Individuals with PTSD and alexithymia may therefore benefit from transdiagnostic interventions primarily targeting emotional clarity deficits, such as the Unified Protocol for Transdiagnostic Treatment of Emotion Disorders (UP; Barlow et al., 2018). Specifically, UP targets emotion awareness, identification, tracking and builds up to patients engaging in exposures to intense and/or unwanted emotion experiences. Therefore, it is paramount in the clinical context to assess for and differentiate between emotion numbing and alexithymia. This may be achieved by clinical interviewing of peri-traumatic emotion processing abilities, collateral information from significant others and/or self-reports such as the TAS-20.

5. Limitations

The impact of the review may be limited due to cross-study variations in control groups, different trauma histories, individual differences and emotion processing paradigms utilised - these introduce unavoidable heterogeneity which makes it difficult to draw valid conclusions regarding subgroup and overall results. Specifically, we cannot rule out that PTSD presentations had varying levels of emotion numbing symptomology or alexithymia which were not explicitly controlled for. However, included studies were screened for potential indicators of the presence of these covariates via available correlated measures (e.g. Empathy Quotient) or for neural profiles indicative of these presentations. In order to enhance the potential differentiation in functional correlates, future studies should include larger samples exploring these two presentations explicitly.

Similarly, only 40% and 60% of the reviewed studies explicitly considered psychiatric comorbidities and concomitant psychopharmacological therapies respectively within their study populations – these largely formed part of study exclusion criteria rather than being formally considered in analyses. Of the remaining studies it was not reported whether these variables were considered in the neuroimaging analyses. However, the consistency of the findings outlined in the present review nonetheless indicates the validity of results. Future studies would benefit from explicitly controlling for these confounds as they can significantly impact on alexithymia and emotion numbing symptomology in PTSD.

Further, while a more in-depth critical appraisal of methodological aspects of studies such as sample size, sampling biases, data processing, etc., would be useful, the limited number of studies and varied methodologies used to date makes this difficult. Such in-depth appraisal should, however, be made in future when there are more studies available. Finally, we did not perform a meta-analysis of research findings, primarily because of the relatively small number of studies performed to date, in addition to the wide variation of methodologies employed. A reliable meta-analysis should be performed in future with

the availability of further research.

6. Conclusions

Our findings suggest that the neural underpinnings of PTSD-related emotion numbing symptomatology differ from PTSD with high trait alexithymia, evidencing that these are distinct constructs. This has important clinical implications which warrant consideration from clinicians when developing individualised treatment programs. The reviewed studies suggest that PTSD-related emotional numbing may be a result of neural hyper-reactivity in the salience network (e.g. in fear and reward circuitry) when presented with negative stimuli, and neural hypo-reactivity when presented with positive/reward stimuli. PTSD-related emotion numbing may therefore be linked to the disorder-level negative affect often reported in PTSD. Conversely, those with PTSD with PTSD and alexithymia showed alterations in DMN (mPFC and posterior cingulate). At a treatment level it suggests that emotion numbing symptomology should remit with adequate treatment of PTSD, however those high in alexithymia may not be adequately able to tend to or report their emotional states making it difficult to access fear memories during trauma-focused psychotherapies. Therefore, it is paramount to identify PTSD patients with high levels of alexithymia as these individuals may not be able to optimally engage in trauma-focused treatments without these emotion deficits being addressed.

Contributors

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Declaration of competing interest

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