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Chantelle S. Lloyd

Andrew A. Nicholson

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Jean Théberge

Richard W. J. Neufeld

See next page for additional authors

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Authors

Chantelle S. Lloyd, Andrew A. Nicholson, Maria Densmore, Jean Théberge, Richard W. J. Neufeld, Rakesh Jetly, Margaret C. McKinnon, and Ruth A. Lanius

Shame on the brain: Neural correlates of moral injury event recall in posttraumatic stress disorder

Chantelle S. Lloyd^{1,2,3,4}  | Andrew A. Nicholson^{4,5} | Maria Densmore^{2,6} |
Jean Théberge^{2,6,7} | Richard W. J. Neufeld^{2,8,9} | Rakesh Jetly¹⁰ |
Margaret C. McKinnon^{3,4,5} | Ruth A. Lanius^{2,4,6,9}

¹Department of Psychology, Neuroscience, and Behaviour, McMaster University, Hamilton, Ontario, Canada

²Department of Psychiatry, Western University, London, Ontario, Canada

³Mood Disorders Program, St. Joseph's Healthcare Hamilton, Hamilton, Ontario, Canada

⁴Homewood Research Institute, Homewood Health, Guelph, Ontario, Canada

⁵Department of Psychiatry and Behavioural Neurosciences, McMaster University, Hamilton, Ontario, Canada

⁶Imaging Division, Lawson Health Research Institute, London, Ontario, Canada

⁷Department of Medical Biophysics, Western University, London, Ontario, Canada

⁸Department of Psychology, Western University, London, Ontario, Canada

⁹Department of Neuroscience, Western University, London, Ontario, Canada

¹⁰Canadian Forces, Health Services, Ottawa, Ontario, Canada

Correspondence

Ruth A. Lanius, University Hospital, 385 Windermere Rd, PO Box 5339, London, ON N6A 5A5, Canada.
Email: ruth.lanius@lhsc.on.ca

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Abstract

Background: Moral injury (MI) is consistently associated with adverse mental health outcomes, including the development of posttraumatic stress disorder (PTSD) and suicidality.

Methods: We investigated neural activation patterns associated with MI event recall using functional magnetic resonance imaging in participants with military and public safety-related PTSD, relative to civilian MI-exposed controls.

Results: MI recall in the PTSD as compared to control group was associated with increased neural activation among salience network nodes involved in viscerosensory processing and hyperarousal (right posterior insula, dorsal anterior cingulate cortex; dACC), regions involved in defensive responding (left postcentral gyrus), and areas responsible for top-down cognitive control of emotions (left dorsolateral prefrontal cortex; dlPFC). Within the PTSD group, measures of state and trait shame correlated negatively with activity among default mode network regions associated with self-related processing and moral cognition (dorsomedial prefrontal cortex; dmPFC) and salience network regions associated with viscerosensory processing (left posterior insula), respectively.

Conclusions: These findings suggest that MI event processing is altered in military and public safety-related PTSD, relative to MI-exposed controls. Here, it appears probable that as individuals with PTSD recall their MI event, they experience a surge of blame-related processing of bodily sensations within salience network regions, including the right posterior insula and the dACC, which in turn, prompt regulatory strategies at the level of the left dlPFC aimed at increasing cognitive control and inhibiting emotional affect. These results are consistent with previous findings showing enhanced sensory processing and altered top-down control in PTSD samples during autobiographical memory recall.

KEYWORDS

fMRI, moral injury, posterior insula, posttraumatic stress disorder, shame, viscerosensory

1 | INTRODUCTION

Military members and public safety personnel (PSP) are routinely confronted with ethical and moral challenges. Navigating such challenges successfully can generate a sense of purposefulness and coherence. Conversely, violating deeply held moral norms, or witnessing a trusted other make such transgressions, can result in a moral injury (Litz et al., 2009; Thompson, 2015). Moral injury (MI) is not classified as a mental disorder (APA, 2013), but rather a strong response to a morally noxious event (Griffin et al., 2019). Morally reprehensible action or inaction perpetrated by either oneself or another has the potential to provoke severe internal anguish (moral dissonance), blame-related emotion (e.g., guilt, shame, rage), negative thoughts and beliefs about self, loss of meaning, loss of trust, and profound social disconnection, contributing collectively to the development of moral injury (Frankfurt & Frazier, 2016; Jinkerson, 2016; Nazarov et al., 2015).

Previous research has focused heavily on the role of guilt and shame in MI (e.g., Aldridge et al., 2019; Farnsworth et al., 2014; Nazarov et al., 2015). Wrongdoing naturally elicits guilt and shame, intended to prompt self-reflection, correction, and learning (Damasio & Carvalho, 2013). Whereas guilt concerns one's behavior and is associated with empathy for the wronged other, shame disrupts empathetic connection altogether, diminishing positive affect, motivating withdrawal and re-focusing attention on self-criticism (Dorahy, 2010; Tangney et al., 2007). Thus, MI and related shame are thought to play a central role in the negative self-referential thoughts and other alterations in cognition and mood observed in posttraumatic stress disorder (PTSD; Litz & Kerig, 2019; Zalta & Held, 2020).

Critically, MI has been linked with the development of PTSD in military (Nazarov et al., 2018) and law enforcement samples (Papazoglou et al., 2020); where it has been further associated with the development of depression (Frankfurt et al., 2018). While MI and PTSD often co-occur, the two are proposed to be mechanistically distinct (Barnes et al., 2019; Bryan et al., 2018; Sun et al., 2019), with MI and PTSD proving to be a particularly deleterious combination, conferring an increased risk for both suicidal thoughts and attempts (Bryan et al., 2018).

Moral cognition is best understood as an integrative process that utilizes the entire neural axis (Greene, 2015), drawing upon a series of domain—general and domain-specific processes supported by the dynamic and interrelated functioning of multiple intrinsic connectivity networks (ICNs; Laird et al., 2011; Menon, 2011). Of these ICNs, the default-mode network (DMN) is critically involved in social cognition, self-referential processing, self-conscious emotion, autobiographical memory, future-oriented thinking, and the experience of self (e.g., Buckner & DiNicola, 2019; Frewen et al., 2020). As there is considerable overlap between social-cognitive and moral processes, DMN regions including the posterior cingulate cortex (PCC), precuneus, ventromedial (vmPFC), and dorsomedial prefrontal cortices (dmPFC) have been commonly cited as neural correlates of moral cognition (e.g., Bastin et al., 2016; Garrigan et al., 2017; Zinchenko & Arsalidou, 2018).

Social norm violation and negative self-conscious emotion are inherent to any MI. Norm violation has been associated with activity in the right insula, dorsolateral prefrontal (dlPFC), and dorsal cingulate cortices—regions responsible for error monitoring, affective processing, and regulation. Negative self-conscious emotion has been shown to preferentially activate regions within the DMN associated with introspective self-reflection (e.g., dmPFC), in addition to salience regions involved in environmental monitoring, appraisal, reward/punishment (dACC), and affective memory processing (e.g., anterior insula; Bastin et al., 2016; Gilead et al., 2016; Zhu et al., 2019). Here, the dlPFC is hypothesized to function as a part of the larger central executive network (CEN), serving as a cognitive-affective control and helping to regulate negative self-conscious emotions elicited in response to perceived norm violations (Zinchenko & Arsalidou, 2018). Critical alterations in ICN activity and connectivity have been noted in PTSD samples, reflecting the neurobiological underpinnings of this disorder and its sequelae (Akiki et al., 2018; Nicholson et al., 2020; Szeszko & Yehuda, 2019). DMN functional disruptions, namely aberrant dmPFC activity, are commonly reported in PTSD patients relative to controls and are hypothesized to mediate negative self-referential thoughts and beliefs, as well as altered social cognition, bodily self-consciousness, autobiographical memory, MI, and shame (Akiki et al., 2017; Bluhm et al., 2009; Cavanna & Trimble, 2006; Daniels et al., 2010; Fenster et al., 2018; Frewen et al., 2020; Tursich et al., 2015). Altered social-cognition in PTSD, including Theory of Mind (ToM; McKinnon et al., 2016; Nazarov et al., 2014), is thought to result from increased connectivity with subcortical structures involved in the innate alarm system (e.g., superior colliculus, amygdala, and postcentral gyrus among other regions; Lanius et al., 2016; Steuwe et al., 2014). Moreover, increased connectivity between DMN and salience network (SN) nodes, and hyperactivity within key salience regions (e.g., dACC, anterior insula), are thought to underpin enhanced sensory processing in PTSD. Hyperactivity within these salience regions appears to contribute toward altered autobiographical memory recall in PTSD, resulting in a pattern of re-experiencing rather than remembering—heightening arousal and challenging memory integration (Thome et al., 2020). Finally, significant functional disruption of CEN regions (e.g., dlPFC) has been observed in PTSD samples relative to controls, revealing a bias toward either under- or over-modulation of limbic activity (Lanius et al., 2010, 2015; Nicholson et al., 2020). Taken together, these patterns of altered neural activity and connectivity disrupt the integrity of primary neural networks in PTSD, corrupting the underlying processes they support, and presumably, the higher-order moral cognition they enable.

To date, only one uncontrolled functional magnetic resonance imaging (fMRI) study of 26 U.S. military veterans has investigated the neural correlates of MI through a resting-state analysis, revealing left inferior parietal lobule (L-IPL) activity as unique to MI but not to PTSD (Sun et al., 2019). Given the paucity of knowledge surrounding the neural correlates of active MI event recall and the relevance of MI to front-line treatments (Held et al., 2018), we sought to fill this evidence gap. We predicted that neural activation patterns during MI

event processing would differ significantly between military and public safety-related PTSD as compared to civilian MI-exposed controls. We included Canadian Armed Forces (CAF) and PSP members with PTSD, given that both populations are considered at heightened risk for MI (Griffin et al., 2019). Consistent with PTSD-related alterations, we expected to observe hypoactivity in prefrontal cortex regions functionally associated with inhibitory control of negative self-conscious emotions (dIPFC), alongside increased activation within areas associated with salience and viscosensory processing (e.g., dACC, and anterior/posterior insula). In addition, we predicted aberrant activation within areas associated with the DMN (e.g., dmPFC), reflecting disrupted self-related processing and maladaptive moral cognition in PTSD during MI event recall.

2 | METHODS

2.1 | Participants

Our sample consisted of 46 participants (military and public safety-related PTSD [$n = 28$]; civilian MI-exposed controls [$n = 18$]). The PTSD group consisted of 16 CAF members (active or retired) and 12 PSP (police or corrections officer), all with a primary diagnosis of PTSD. Four participants were receiving inpatient residential treatment at the time of the study. Group demographic and clinical characteristics are presented in Table 1. Participants were recruited via advertisements posted within the London Ontario community and local mental health treatment centers. Study procedures were approved by the Health Sciences Research Ethics Board of Western

TABLE 1 Demographic and clinical information

	PTSD Group	Control Group
N	28	18
Sex	25 males, 3 females	7 males, 11 females
Age	48.5 ± 8.3	33.1 ± 10.9
CAPS-Total	40.89 ± 7.94*	0 ± 0
CAPS-D	15.39 ± 3.53*	0 ± 0
CTQ-Total	50.4 ± 22.6*	30.3 ± 8.4
MDI-Total	60.3 ± 16.3*	35.6 ± 5.2
MDD Recurrent	Current = 9, past = 0	Current = 0, past = 0
MDD Single Episode	Current = 1, past = 1	Current = 0, past = 0
Psychotropic medication	23	0

Abbreviations: CAPS, Clinician-Administered PTSD Scale for DSM-5; CTQ, Childhood Trauma Questionnaire; MDD, major depressive disorder; MDI, Multiscale Dissociation Inventory; PTSD, posttraumatic stress disorder.

*Significantly higher clinical symptom values in the trauma-exposed control group.

University. Consenting participants received financial compensation for participation.

Participants with a lifetime diagnosis of bipolar or psychotic disorder, or, with current alcohol or substance use disorder were excluded. Sustained full remission from substance use was required for a minimum of 3 months before study involvement. For control participants, lifetime psychiatric illness or psychotropic medication served as an additional exclusion criteria. The Moral Injury Events Scale (MIES; Nash et al., 2013) was used to confirm exposure to potentially morally injurious events (items 8–9 omitted for non-military participants). Exclusion criteria for all participants included noncompliance with 3 Tesla fMRI safety standards, significant untreated medical illness, pregnancy, a neurological or pervasive developmental disorder, or a head injury with loss of consciousness.

2.2 | Clinical interviews and memory scripts

The Structured Clinical Interview for DSM-IV Axis-I Disorders—Research Version (First et al., 2002) was administered to ascertain psychiatric history for study inclusion and determine comorbidities. The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5; Weathers, Blake, et al., 2013; Weathers, Litz, et al., 2013) was used to assess total PTSD symptom severity as well as Criterion D symptom severity, pertaining to alterations in cognition and mood (i.e., the inability to recall event details, exaggerated negative beliefs/expectations, distorted cognitions leading to blame, persistent negative state, apathy, interpersonal detachment, and diminished positive emotion). The Multiscale Dissociation Inventory (MDI; Briere et al., 2005) and Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994) were used to assess group differences in dissociative symptoms and childhood maltreatment and neglect, respectively.

Two personalized memory scripts were developed during the clinical interview; each included eight short sentences describing a discrete event. The first script described a neutral event (e.g., a trip to the store) and the second described a morally injurious event. Memories were prompted during the clinical interview by procedures outlined in past autobiographical memory studies of trauma (McKinnon et al., 2015; Palombo et al., 2016) and reformatted to resemble widely used script-driven imagery procedures suitable to fMRI (Lanius et al., 2010).

2.3 | Experimental setup and fMRI protocol

Lying supine on the MR scanner bed, participants were exposed first to their neutral memory script, and following a 2-min break to their MI memory script. Scripts were presented sentence by sentence. Sentence 1 was visually displayed for 5 s and read concurrently in a neutral affective tone, before transitioning to a blank screen wherein participants were instructed to recall that part of the memory for 25 s. Following recall, a virtual avatar was presented, followed by a fixation cross (data relating to the avatar will be presented

elsewhere). This procedure was repeated for the next sentence. Visual stimuli were projected onto a screen, visible to the participant through a mirror attached to the head coil. Participants wore MR-compatible headphones to reduce the scanner noise and deliver audio stimuli. Stimulus presentation was controlled by a PC running E-Prime 3.0 software (Psychology Software Tools, Inc., 2016). After each script, participants rated the degree to which each memory induced (state) shame using a MR-compatible button press (1 = *not at all* and 4 = *very much so*).

2.4 | fMRI image acquisition and preprocessing

We utilized a 3 Tesla MRI Scanner (Biograph mMR; Siemens Medical Solutions) for brain imaging with a Siemens 32-channel head coil locally adapted to this scanner's 4-plug interface. Orthogonal scout images were collected and used to prescribe a tri-dimensional T1-weighted anatomical image of the whole head with 1-mm isotropic resolution (Magnetization Prepared Rapid Gradient Echo (MP-RAGE). Functional whole-brain images with blood-oxygen-level-dependent (BOLD) contrast were acquired transversely with the manufacturer's gradient echo, T2*-weighted blipped-echo-planar sequence (TE = 20 ms, TR = 3000 ms, FOV = 256 × 256 mm, flip angle = 90°, in-plane resolution = 2 × 2 mm), parallel imaging acceleration factor = 4). Each volume included 60 ascending interleaved slices with a thickness of 2 mm. Participants' heads were stabilized with foam padding and the experimental runs each consisted of 118 volumes.

Preprocessing of the functional images was conducted with SPM12 (Wellcome Department of Cognitive Neurology) within MATLAB R2019. Our standard preprocessing routine included discarding four initial volumes, reorientation to the AC-PC axis, spatial alignment to the mean image using a rigid body transformation, re-slicing, and coregistration of the functional mean image to the subject's anatomical image. Co-registered images were segmented using the "New Segment" method in SPM12. The functional images were normalized to MNI space (Montréal Neurological Institute) and were smoothed with a FWHM Gaussian kernel of 6 mm. Additional correction for motion was implemented using the ART software package (Gabrieli Lab, McGovern Institute for Brain Research), which computes regressors that account for outlier volumes.

2.5 | Statistical analyses

2.5.1 | First-level analysis

For each participant, all events (rest, instructions, fixation, and conditions) were modeled as blocks of brain activation and convolved with the hemodynamic response function. We used 117 volumes; the last volume was excluded because stimuli presentation ended partway through its acquisition. At this stage, functional data were high-pass filtered and serial correlations were accounted for using an autoregressive AR(1) model; ART software regressors were included

as nuisance variables to account for any additional movement artifacts. The two experimental conditions (*neutral/MI event recall*) were modeled separately on the first level for each participant.

2.5.2 | Second-level analysis

Contrast images for neutral and MI event recall were entered into second-level analyses in SPM12 to examine between group differences in neural activation during MI processing. We first conducted a 2 (group) × 2 (condition) full-factorial split-plot analysis of variance (ANOVA) to examine interaction effects between participant group (PTSD, controls) and conditions (MI and neutral event recall). We then tested specifically our a priori defined hypotheses regarding between group differences in terms of neural activation during MI event recall. We evaluated between group differences in functional activation at the whole-brain level, with separate two-sample *t* tests for the two event conditions. All analyses were evaluated at the conservative threshold of $pFDR < .05$, $k = 10$, observed at the cluster-corrected level to control for multiple comparisons (see Eklund et al., 2016) with the initial uncorrected cluster-forming threshold in statistical parametric mapping (SPM) set at $p < .001$, $k = 20$.

3 | RESULTS

3.1 | Demographic and clinical variables

No statistically significant group differences emerged with respect to biological sex (Table 1). Significant differences were found between groups for age; however, a closer examination of the influence of age on the BOLD signal was found to reveal nonsignificant differences across conditions. While the PTSD group reported higher scores on the CTQ, the entire sample endorsed exposure to childhood maltreatment or neglect (as measured by any subscale >5, or minimization scale >0). As expected, the PTSD group reported significantly higher dissociation scores on the MDI.

3.2 | Neuroimaging results

Our full factorial split-plot ANOVA revealed a trending group by condition interaction in the left posterior cingulate cortex (PCC; MNI = -2, -40, 22; $F = 30.55$; $Z = 4.97$; $pFDR$ cluster corrected = .055; $k = 71$) and the left dIPFC (MNI = -32, 46, 8; $F = 21.38$; $Z = 4.21$; $pFDR$ cluster corrected = .055; $k = 68$), in the absence of both a significant main effect of group and main effect of condition. Nevertheless, direct group comparisons revealed that the PTSD group had significantly greater activation in the dACC, left dIPFC, right posterior insula, and left postcentral gyrus during MI memory recall, as compared to controls (see Table 2 and Figure 1). By contrast, the control group did not display significantly greater activation as compared to the PTSD group during MI recall conditions. Strikingly,

TABLE 2 Independent samples *t* tests

Condition	Comparison	Brain region	H	Cluster size	BA	MNI coordinate			<i>t</i> stat.	Z score	pFDR cluster level
						x	y	z			
Moral injury	PTSD > Control	Dorsal anterior cingulate cortex		175	32	-2	32	14	5.56	5.13	.001
			L	133	8, 9, 10	-38	46	10	4.46	4.23	.005
			R	106		38	-14	-6	4.37	4.15	.011
			L	69	40	-64	-22	18	4.02	3.84	.049
	Control > PTSD	ns									
Neutral	PTSD > Control	ns									
	Control > PTSD	ns									

Note: Direct group comparisons for the moral injury and neutral memory recall conditions. Independent samples *t* tests were evaluated at the FDR-cluster corrected threshold for multiple comparisons ($p < .05$, $k = 10$).

Abbreviations: BA, Brodmann area; FDR, false discovery rate correction; H, hemisphere; PFC, prefrontal cortex; PTSD, posttraumatic stress disorder.

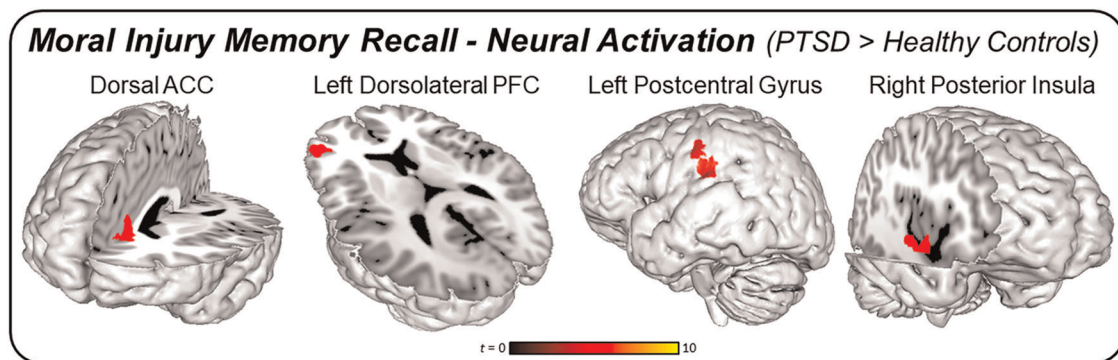


FIGURE 1 Brain areas showing greater activation in the PTSD group as compared to MI-exposed controls during moral injury memory recall. Neural activation results are reported at the FDR-cluster corrected threshold ($p < .05$, $k = 10$). ACC, anterior cingulate cortex; FDR, false discovery rate correction; PTSD, posttraumatic stress disorder

we found no significant differences between groups in terms of neural activation during neutral memory event recall conditions (see Table 2).

3.3 | Clinical correlations

Within the PTSD group, we found a negative correlation between memory-related shame scores and activity in the right superior frontal gyrus (SFG) during MI recall conditions. We also found that symptoms of altered cognitions and mood (CAPS-5 Criterion D) correlated negatively with left posterior insula activation during MI recall (see Table 3 and Figure 2).

4 | DISCUSSION

Our findings point toward important differences with respect to how morally injurious events are recalled and processed among individuals with military and public safety-related PTSD, relative to

civilian MI-exposed controls. During MI event processing, the PTSD group displayed increased activation within key salience network hubs, including the right posterior insula and the dACC, as well as in regions involved in defensive responding (left postcentral gyrus). Contrary to our prediction, dlPFC activity increased rather than decreased in the PTSD group, relative to controls, in response to MI recall. Interestingly, higher ratings of state shame during the fMRI scan were associated with dmPFC hypoactivity, a key node within the DMN. Additionally, alterations in cognitions and mood (Criterion D) were related to hypoactivity in the left posterior insula involved in the posterior SN. Interestingly, there were no statistically significant differences between the PTSD and control group when recalling neutral events, suggesting this disrupted processing was unique to morally injurious events.

4.1 | Neural correlates of MI event recall

In PTSD, MI event recall prompted increased activation in key SN nodes (right posterior insula, dACC), regions involved in defensive

TABLE 3 Clinical correlation analysis

Measure	Group	Condition	Correlation	Brain region	H	Cluster size	BA	MNI Coordinate			t stat.	Z score	pFDR cluster level
								x	y	z			
Shame	PTSD	Moral injury recall	Negative	Superior frontal gyrus	R	102	8	26	12	48	5.66	4.50	.021
Alterations in cognitions and mood	PTSD	Moral injury recall	Negative	Posterior insula	L	92	-40	-18	12	6.45	4.94	4.94	.033

Note: Correlations between neural activation during moral injury recall among PTSD patients and (i) alterations in cognitions and mood (CAPS cluster D severity score), and (ii) state measures of shame induced by event recall in the fMRI scanner, evaluated at the FDR-cluster corrected error protection rate ($p < .05$, $k = 10$).

Abbreviations: BA, Brodmann area; CAPS, Clinician-Administered PTSD Scale for DSM-5; FDR, false discovery rate correction; fMRI, functional magnetic resonance imaging; H, hemisphere; PTSD, posttraumatic stress disorder.

responding (left postcentral gyrus), as well as hyperactivity in brain areas responsible for top-down cognitive control of emotions (left dIPFC). The dACC contributes centrally to moral processing, activating in response to social norm violations (Zinchenko & Arsalidou, 2018), error monitoring, future planning (Heilbronner & Hayden, 2016), shame and guilt (Bastin et al., 2016), and social pain (Eisenberger & Lieberman, 2004; Masten et al., 2011). Indeed, the dACC appears to be hyperactive among patients with PTSD, resulting in greater salience interference within DMN regions and reflecting a neural correlate of chronic fight-or-flight states and hyperarousal (Akiki et al., 2017; Koch et al., 2016).

Whereas the anterior insula is linked with conscious awareness of feeling states, the posterior insula has long been conceptualized as a somatosensory integration hub of viscerosensory information (Craig, 2002), receiving raw interoceptive information from the brainstem via thalamic projections and connecting, in turn, with posterior, temporal, parietal, and sensorimotor areas (Craig, 2009). The insula itself supports a wide variety of functions, including multisensory integration within and between cognitive, affective, and sensorimotor networks (Namkung et al., 2017; Uddin et al., 2017). Notably, increased posterior insula activation has been associated with the perception of primary visceral sensations, including unpleasant feelings of gastrointestinal constriction and distress (e.g., a pit in the stomach or vomiting; Stephani et al., 2011) and moral disgust (Ying et al., 2018). Similar to the dACC, the posterior insula has also been implicated in the perception of both painful (e.g., pinprick, burning; Pavuluri & May, 2015) and non-painful tactile sensations (Jensen et al., 2016).

Consistent with increased posterior insula/dACC function, almost uniformly, our PTSD participants described the experience of MI recall to be both nauseating and painful, like an internal gnawing sensation. One participant reported feeling like something was “*eating [him] alive inside.*” Here, we speculate that MI recall in PTSD heightens posterior insula activation, heightening its connections to the viscera, prompting a raw viscerosensory experience of blame-related emotion that occurs alongside activation of defensive responding (postcentral gyrus/innate alarm system). The postcentral gyrus, another somatosensory processing region, has been shown to activate during the induction of basic negative emotions such as anger (Gilead et al., 2016) and during defensive responding more generally (Lanius et al., 2016). We speculate that SN hyperactivity and exacerbated somatosensory processing may be related to unpleasant visceral sensations associated with morally injurious events. In turn, this may prompt increased dIPFC/CEN activity in an attempt to modulate excessive bottom-up affect—a neural activation pattern that has also been linked with dissociation (Lanius et al., 2018).

4.2 | Neural correlates of shame

In PTSD participants, activity in the right SFG decreased as MI-related shame ratings during the fMRI scan increased. Contained within the dmPFC, the right SFG is involved in self-reflective

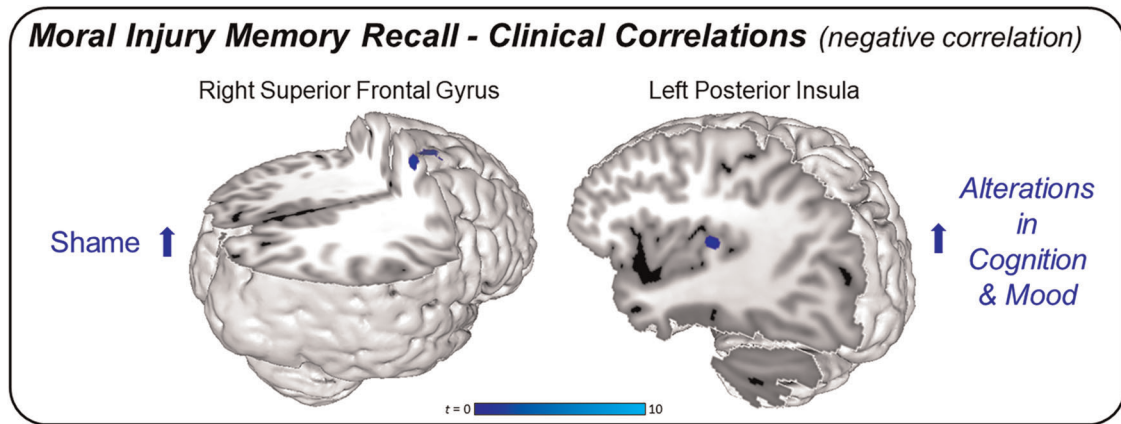


FIGURE 2 Brain areas showing significant correlations to clinical PTSD symptoms in the PTSD group during moral injury memory recall. Trait measures of PTSD clinical symptoms: symptoms of altered cognitions and mood (CAPS cluster D), were negatively correlated to the left posterior insula during moral injury recall. Paradigm induction (state) symptoms: shame scores were negatively correlated to the right superior frontal gyrus during moral injury recall conditions. Correlation results are reported at the FDR-cluster corrected threshold ($p < .05$, $k = 10$). CAPS, clinician-administered PTSD scale; PTSD, posttraumatic stress disorder

processes, including the processing of negative self-conscious emotions, self-evaluation, self-judgment, and autobiographical memory (Gilead et al., 2016; Frewen et al., 2020; Goldberg et al., 2006; Göttlich et al., 2020; Thome et al., 2020). The dmPFC, as well as other DMN hubs, are thought to be critical to moral cognition (e.g., Bastin et al., 2016; Garrigan et al., 2017; Zinchenko & Arsalidou, 2018). In the current study, high experiences of shame co-occurring with decreased SFG activation may reflect disrupted self-related processing, maladaptive moral cognition, and altered self-reflection among PTSD patients during MI event recall.

Finally, self-reports of diminished positive affect, persistent self-blame, lack of interest, and social disengagement (PTSD Cluster D; negative alterations in cognition and mood) among PTSD participants were associated with reduced activity in the left posterior insula, which may suggest viscerosensory dampening within this group. Here, we hypothesize that left posterior insula activation may be inhibited in participants with more severe alterations in cognition and mood in an attempt to facilitate emotional numbing (detachment from bodily sensations). These findings converge with prior research in PTSD showing that hypoactivity in the posterior insula is associated with altered socio-emotional processing (Akiki et al., 2017; Koch et al., 2016).

4.3 | Limitations and future directions

The COVID-19 pandemic limited group sizes unexpectedly. Controlled replication studies are required before results are considered reliable (Yarkoni, 2009), including studies that examine subjective measures of visceral sensations directly. Our findings point toward important group differences that may be further analyzed in larger samples, including comparisons between participants with and without the PTSD dissociative subtype. Network analyses will be needed to explore changes in ICN activity and connectivity during MI

recall. The examination of this visceral somatosensory phenomena, as mediated by the posterior insula, will be important for the understanding of psychopathology, including within anxiety and somatic symptom and related disorders as well as somatization more broadly.

5 | CONCLUSION

Our results suggest that MI event processing is altered among individuals with military and public safety-related PTSD as compared to MI-exposed controls. MI event recall in the PTSD as compared to the control group was associated with increased neural activation among salience network nodes involved in viscerosensory processing and hyperarousal (right posterior insula, dACC), regions involved in defensive responding (left postcentral gyrus), and areas responsible for top-down cognitive control of emotions (left dlPFC). We hypothesize that as individuals with PTSD recall their MI event, they experience a surge of blame-related processing of bodily sensations within salience network regions, which in turn, prompt prefrontal regulatory strategies aimed at increasing cognitive control and inhibiting emotional affect. High levels of shame during the fMRI scan were associated with aberrant activation within the dmPFC, which may reflect disrupted DMN processing, maladaptive moral cognition, and altered self-reflection among PTSD patients. Finally, excessive left posterior insula activation may be inhibited in participants with more severe alterations in cognition and mood in an attempt to facilitate emotional numbing (detachment from bodily sensations), thus challenging the integration of morally injurious events.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

ORCID

Chantelle S. Lloyd  <https://orcid.org/0000-0003-2326-7851>

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