NEUROIMAGING AND BEHAVIOURAL INVESTIGATION INTO THE EXPERIENCE OF MORAL INJURY

BEHAVIOURAL AND NEUROIMAGING INVESTIGATION INTO THE EXPERIENCE OF MORAL INJURY

By

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Lay Abstract

Moral injury (MI) is a psychosocial-spiritual injury that can occur when deeply held values are violated either by oneself or a trusted other; it produces considerable pain and social alienation. MI has been linked to suicide and the development of posttraumatic stress disorder (PTSD), a mental health condition associated with distressing symptoms and reduced functioning in important areas of life, including social relationships. This thesis focuses on how MI events are processed by military members and public safety personnel, who are both at risk for MI and PTSD. We investigate how shame is experienced in the brain and body, and explore how intensified visceral sensations may become overwhelming (e.g., *pit in stomach*, *vomiting*) prompting emotional numbing or difficulties remaining embodied in the present moment (e.g., *zoning out*, *freezing up*). By understanding MI event processing when PTSD is present, we hope to gain insight into more effective treatments for these individuals.

Abstract

Moral injury (MI) is associated with severe blame-related emotion and the development of psychopathology including posttraumatic stress disorder (PTSD). Little is known about how MI events are neurally processed when PTSD is comorbid, limiting the development of tailored interventions. Thus, this thesis sought to provide a novel, multi-method examination of the biological underpinnings of moral injury and relevant behavioural correlates. Study one provides the first investigation into the neural activation patterns elicited during MI event recall in military members and public safety personnel with PTSD, relative to MI-exposed civilian controls. In PTSD, emotional processing is challenged by heightened sensory information. Here, we provide evidence of heightened viscerosensory information processing (i.e. *internal gnawing* or gastrointestinal constriction related to blame-based emotion) during MI event recall, which appears to exert a strong influence over cortical regions facilitating moral cognitive processes including emotion regulation, autobiographical memory integration, and social cognition. Overwhelming visceral sensations can elicit defensive behaviour including tonic immobility (TI), a defensive response that facilitates viscerosensory dampening. Interestingly, more severe negative alterations in cognition and mood were associated with viscerosensory dampening in our PTSD group, pointing towards a compensatory pattern of emotional numbing. Studies two and three explore two posttraumatic symptoms consistent with emotional numbing: alexithymia and posttraumatic TI. In study two, we explore posttraumatic TI as a survival-based dissociative response and test a new measure of posttraumatic TI. In study three, we provide evidence that alexithymia is associated

with an altered (muted) pattern of emotion-specific bodily sensation. This thesis provides a framework for embodied MI event processing in PTSD and highlights the importance of assessing the somatic experience of MI and screening for TI responses and emotional numbing as part of PTSD symptomatology. The evidence presented here suggests sensorimotor-based approaches and bottom-up regulatory strategies may be useful adjuncts to MI event processing.

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You must have sufficient joy in order to be moral

- John Stewart Mills

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List of Abbreviations

ACE	Adverse Childhood Experience
ACT	Acceptance and Commitment Therapy
ANOVA	Analysis of Variance
AR	Auto Regressive
BOLD	Blood-Oxygen-Level-Dependent
CAF	Canadian Armed Forces
CAPS-5	Clinician Administered PTSD Scale for DSM 5
CBT	Cognitive Behavioural Therapy
CEN	Central Executive Network
CFA	Confirmatory Factor Analyses
CFI	Comparative Fit Index
CI	Confidence Interval
CPT	Cognitive Processing Therapy
CTQ	Childhood Trauma Questionnaire
dACC	Dorsal Anterior Cingulate Cortex
DBT	Dialectical Behavioural Therapy
dmPFC	Dorsal Medial Prefrontal Cortex
DSS	Dissociative Symptoms Scale
DMN	Default Mode Network
DSM-5	Diagnostic and Statistical Manual, Fifth Edition
dlPFC	Dorsolateral Prefrontal Cortex
EFA	Exploratory Factor Analysis
EM	Expectation Maximization
ESEM	Exploratory Structural Equation Models
FDR	False Discovery Rate
fMRI	Functional Magnetic Resonance Imaging
ICN	Intrinsic Connectivity Network
IRRT	Imagery Rescripting and Reprocessing Therapy
MCAR	Missing Completely at Random
MDD	Major Depressive Disorder
MDI	Multiscale Dissociation Inventory
MI	Moral Injury
MIES	Moral Injury Events Scale
MNI	Montréal Neurological Institute
mPFC	Medial Prefrontal Cortex
MTurk	Amazon's Mechanical Turk
PCC	Posterior Cingulate Cortex
PCL-5	PTSD Checklist for DSM 5
PCL-5+	PTSD Checklist for DSM 5 plus ten TRASC items
PDEQ	Peri-traumatic Dissociative Experiences Questionnaire
PE	Prolonged Exposure

PFC	Prefrontal Cortex
PHQ-4	Patient Health Questionnaire, four item version
PMIE	Potentially Morally Injurious Event
PSP	Public Safety Personnel
PTSD	Posttraumatic Stress Disorder
RMSEA	Root Mean Square Error of Approximation
SFG	Superior Frontal Gyrus
Shut-D	The Shutdown Dissociation Scale
SI	Supplemental Information
SN	Salience Network
SRMR	Standardized Root Mean Residual
STOP	Scale for Tonic immobility Occurring Post-trauma
TAS	Toronto Alexithymia Scale
TI	Tonic Immobility
TLI	Tucker-Lewis Index
ToM	Theory of Mind
TRASC	Trauma Related Altered States of Consciousness

Declaration of Academic Achievement

This thesis contains a total of five chapters: Chapter 1 provides a background to the material, Chapters 2 through 4 are empirical articles, and Chapter 5 discusses the main conclusions, limitations, and future directions.

Drs. McKinnon and Lanius conceived and developed the study presented in Chapter 2. This research was supported by the Lawson Health Research Institute and the Homewood Research Institute. C. Lloyd assisted Dr. Théberge to refine the final fMRI protocol and conduct pilot testing, prior to data collection. Dr. Théberge oversaw the implementation of brain imaging acquisition methods used to collect neuroimaging data. C. Lloyd conducted all clinical interviews and scans under the guidance of Drs. McKinnon, Lanius. and Théberge. C. Lloyd conducted all data analyses under the guidance of M. Densmore. Drs. McKinnon, Lanius, Neufeld, Théberge, Nicholson and M. Densmore provided critical revisions to the final article, which was submitted to *Depression & Anxiety* in September 2020 (presently under review).

The research presented in Chapter 3 was collaboratively conceptualized by C. Lloyd, M. Brown and Drs. McKinnon and Lanius. C. Lloyd developed and evaluated the scale, under the guidance of Drs. McKinnon, Lanius, Neufeld and Frewen. C. Lloyd and M. Brown developed and administered the online survey battery, and C. Lloyd cleaned and prepared the final dataset. Dr. Frewen and M. Brown consulted on the plan for data analysis, and C. Lloyd carried out data analysis and interpretation under their guidance. Drs. Lanius, Neufeld, Frewen, & McKinnon, and M. Brown provided critical revisions to the final article, which was submitted to and published in *Chronic Stress* in January 2019.

The research presented in Chapter 4 was collected by E. Stafford as part of a larger cross-sectional study conceptualized by her supervisor, Dr. D'Andrea. Dr. Thome assisted with the plan for data analysis and adapted the statistical code. Drs. McKinnon, Lanius, Thome, and Neufeld were consulted about the inferences made from the experimental findings. E. Stafford, M. Densmore, and Drs. McKinnon, D'Andrea, Thome, Rabellino, Neufeld, and Lanius provided critical revisions to the final article, which was submitted for review to *Child Abuse & Neglect* in September 2020 (presently under review).

Dr. Neufeld was consulted about the statistical analysis used across Chapters 2-4, and M. Densmore supervised the statistical analysis used to investigate neuroimaging data (Chapter 2) as well as the topographical data presented in Chapter 4. Drs. McKinnon and Lanius supervised all stages of this thesis.

Chapter 1: General Introduction

Moral injury (MI) has gained considerable attention as a psychological construct over the past decade, motivating questions about the boundaries of therapeutic practice, the applicability of current front-line treatments for posttraumatic stress disorder (PTSD) for treating MI symptoms, and the role of morality in recovery from posttraumatic stress. Despite a burgeoning literature base, little evidence has described the neurobiological correlates of MI. This thesis examines the neural correlates of MI event recall, when military-related PTSD is present compared to when it is absent. Behavioural and emotional responses relevant to MI are also explored.

1.1 What is Moral Injury?

The term *moral injury* was coined in 1992 by Jonathan Shay, and has evolved as a construct for understanding what happens to humans when deeply held moral beliefs are transgressed (Litz et al., 2009). As a consensus definition has not been reached (Griffin et al., 2019; Litz & Kerig, 2019), we adopt a broad definition of MI as the psychosocial-spiritual injury that occurs after perpetrating, failing to prevent, bearing witness to, or, learning about acts that transgress deeply held moral beliefs and expectations (Litz et al., 2009). This may also relate to a betrayal by a leader, a trusted other (Shay, 2014), or an institution (Jakupcak, 2013). Exposure to such MI events may lead to reduced confidence, inner turmoil and negatively altered thoughts and expectations about one's own or others' motivation or capacity to behave in a just and ethical manner (Drescher et al., 2011).

Morality is a code of conduct based upon a set of customs or values, referred to as norms, that set the standards of right and wrong within a culture or group (Tangney et al., 2007). From an evolutionary standpoint, possessing a shared capacity for morality enables social learning and promotes socially favorable behaviour that maximizes the cohesion and cooperation necessary for survival (Moll et al., 2003). Accordingly, the social and moral brain are highly interconnected (e.g., Bastin et al., 2016; Garrigan et al., 2017; Yoger et al., 2018; Zinchenko & Arsalidou, 2018). Social-moral emotions (hereafter referred to as moral emotions) allow an individual to learn the rules, or norms, of a group and act within the boundaries of that group's moral system (Giner-Sorolla & Espinosa, 2011; Goetz et al.,2010). Negative moral emotions (e.g., guilt, shame, anger-rage, betrayal) serve to punish behaviour and prompt behavioural change, whereas positive moral emotions (e.g., pride, compassion, love, gratitude), serve to reinforce and celebrate prosocial behaviour (Tangney et al., 2007). By prompting reflection and evaluation by self and other, moral emotions collectively serve to regulate and strengthen one's reputation and behaviour within a group (Mendez, 2009; Tangney et al., 2007). Trust is born out of shared norms, representing a social contract (Fiske, 2002; Tangney et al., 2007). MI invariably breaches this social contract, motivating social disconnection.

MI began as a heterogeneous construct, which referred to both i) exposure to potentially morally injurious events (PMIEs), and/or ii) the symptom expression resulting from exposure (Griffin et al., 2019; Litz & Kerig, 2019).

Critically, MI exposure and symptom expression are distinct, where PMIE exposure is a necessary precipitant of MI, but PMIE exposure does not always result in MI (Frankfurt & Frazier, 2016; Griffin et al., 2019). Attempts have been made to differentiate between perpetration-based and betrayal-based moral injuries, made by self and other, respectively (Jordan et al., 2017). Similar distinctions have been made between individual- and institutional-based moral injuries (Birrell et al., 2017; Jakupcak, 2013).

1.2 Models of Moral Injury

Current models of MI fall into two categories: stress injury models (e.g., Nash et al., 2007) and stress-appraisal and coping models (e.g., Lazarus & Folkman, 1984), where the former emphasizes a literal biomedical injury as the generating source of distress, whereas the latter emphasizes the role of perspective and thinking (Nash, 2019). Both model types note the role of negative moral emotions such as guilt, shame, and anger, in generating symptom expression. More recently, shame, not guilt, has been proposed to distinguish MI from less severe forms of moral distress (Litz & Kerig, 2019; Zalta & Held, 2020). Shame, like guilt, is an important adaptive response that facilitates social-moral learning (de Hooge et al., 2008). Evolutionary models posit that shame results when a moral transgression is attributed to an internal, stable, and global sense of self (Tangney & Dearing, 2002; Tangney & Dearing, 2004), providing a powerful conduit to self-condemnation - a framework echoed by Litz and colleagues (2009) in their preliminary model. Farnsworth et al (2019) has recently proposed a stress-

appraisal framework wherein descriptive cognitions about the MI event and prescriptive cognitions about what the event means, are used to differentiate PTSD and MI, helping to identify unique treatment needs and target mechanisms of change. Importantly, a model of MI when PTSD is also present has not been produced. Thus, evidence from this thesis will be used to inform a framework of MI when PTSD is comorbid.

1.3 Who is affected by Moral Injury?

MI can occur following exposure to PMIEs (Williamson et al., 2018) and is more likely to occur in those exposed to high levels of moral stress and ambiguity (Farnsworth et al., 2014; Harris et al., 2015). To date, the majority of research has focused on military-related MI (e.g., Bryan, 2015; Bryan et al., 2016; Gray et al., 2012; Williamson et al., 2018; Yeterian et al., 2017) and a growing body of research has investigated MI among police officers (Komarovskaya et al., 2011; Papazoglou et al., 2019; 2020). However, research examining MI in other public safety personnel (PSP) or first responders (e.g., corrections officers, paramedics, firefighters, 911 dispatch operators, etc.) is relatively scant (Dentry et al., 2017; Murray, 2019). MI also has been examined in a wide-range of civilian populations including refugees (Nickerson et al., 2018, 2015), teachers in violent settings (Currier et al., 2015), child protection personnel (Haight et al, 2017), and parents involved with child protective services (Haight et al., 2017). MI can also commonly occur among health care clinicians (Dean et al., 2019), including physicians (Førde & Aasland, 2013), nurses (Stovall et al., 2020), and medical

students (Murray et al., 2018). In fact, over the last six months, the COIVD-19 pandemic has motivated dozens of articles examining MI among various front-line workers who are operating in settings with extreme moral stress (e.g., Haller et al., 2020).

Importantly, the experience of MI has yet to be compared between occupations, or between groups with and without comorbid PTSD. In the next chapter, we begin to address this evidence gap by examining the neural correlates of MI event recall in MI-exposed military members and PSP with PTSD, as compared to civilian MI-exposed controls (without PTSD).

1.4 Core Symptom Expression of Moral Injury

Clinicians and researchers have generally cautioned against overpathologizing the expression of MI, suggesting its "symptoms" (e.g., self- and other-condemning cognitions, emotions, and behaviours) represent understandable, albeit passionate, responses to severe ethical transgressions (e.g., Farnsworth et al., 2017; Litz et al., 2009). Accordingly, MI is most often associated with blame-related emotion (e.g., guilt, shame, anger), loss of trust, anhedonia, social alienation, negative changes in the perception of the goodness of the world (i.e. altered beliefs and cognitions) and spiritual or existential crisis (Farnsworth, 2019; Frankfurt & Frazier, 2016; Jinkerson, 2016; Nazarov et al., 2015). Recent evidence further suggests that perpetration-based MI events (transgression made by oneself) are more closely associated with guilt/shame, whereas betrayal-based MI events (witnessing or learning about transgressions made by another) are more closely associated with

anger (Jordan et al., 2017). Thus, MI itself is not conceptualized as psychopathology (Currier et al., 2017; Gray, Nash, & Litz, 2017), although maladaptive coping behaviours to severe moral pain can result in psychopathology (discussed next).

1.5 Negative Health Outcomes of Moral Injury

Exposure to PMIEs has been associated with the development of PTSD (Barnes et al., 2019; Farnsworth et al., 2019; Nazarov et al., 2018) and it's comorbid conditions including depression (Frankfurt et al., 2018), substance use (Jinkerson, 2016), and suicidality (Bryan et al., 2018).

PTSD is characterized by intrusive symptoms, avoidance, physiological hyperarousal and negative alterations in cognition and mood (APA, 2013). Approximately 30% of individuals develop a dissociative subtype of PTSD characterized instead by symptoms of hypoarousal and associated feelings of unreality (derealisation) and/or detachment from bodily experiences (depersonalization; Lanius et al., 2012; Spiegel et al., 2013). Importantly, when PTSD is comorbid with MI, the likelihood of suicidal thoughts and behaviours has been shown to increase dramatically (Bryan et al., 2018)

The emotional symptoms of MI appear to play a pivotal role in the development of psychopathology. For example, previous research has shown persistent guilt, shame and anger consistently predict the development, severity and maintenance of PTSD (Cunningham et al., 2017; Holmes et al., 2005; Oktedalen et al., 2015) as well as prevent treatment success from traditional

cognitive-behavioural therapies (CBTs; Grunert et al., 2007; Nishith et al., 2005; Pitman et al., 1991; Resick & Schnicke, 1992; Resick et al., 2002). Furthermore, guilt and shame have been closely associated with the development of depression (Jinkerson, 2016; Kim et al., 2011; Marx et al., 2010) and suicidality (Bryan et al., 2014; McLean et al., 2017).

MI has been shown to severely impact social desire and function, often limiting social support, which both buffers against the development of PTSD and promotes recovery (e.g., Dai et al., 2016; Stevens & Jovanovic, 2019). Relatedly, MI is associated with anhedonia, or reduced positive emotion, which helps to promote social connection by enabling emotional bonding and attachment (e.g. Fisher et al., 2013).

Interestingly, data from a nationally-representative mental health survey of active Canadian military personnel indicated that exposure to PMIEs is actually associated with *increased* help-seeking behaviour, particularly from para-professionals and natural supports (Nazarov et al., 2020). This finding opposes long-standing assumptions that the guilt and shame associated with MI would motivate treatment avoidance. This counterintuitive finding may also speak to the severity of moral pain associated with MI, which may outweigh the discomfort of seeking help. It is presently unknown whether, or how, these MI-related social alterations extend to the therapeutic alliance.

Finally, MI has shown strong positive correlations with negative alterations in cognition and mood, a diagnostic criteria for PTSD, according to the

Diagnostic and Statistical Manual, Fifth Edition (DSM-5) (APA, 2013). These symptoms are measured by the following seven items in the Clinician Administered PTSD Scale for DSM 5 (CAPS-5; Weathers et al., 2013) in fulfillment of PTSD Criterion D (APA, 2013): the inability to recall important aspect of event (D1); exaggerated negative beliefs or expectations (D2); distorted cognitions leading to blame (D3); persistent negative emotional state (D4); diminished interest or participation in activities (D5); detachment or estrangement from others (D6); persistent inability to experience positive emotions (D7). Some authors have argued that this symptom category should be used to differentiate between PTSD and MI (Farnsworth, 2019), whereas others argue that this distinction is premature (Frankfurt & Coady, 2019), particularly since MI symptom expression is still being defined.

In summary, the core symptoms of MI (e.g., self- and other-condemning cognitions and emotions, social disconnection, etc.) appear to serve as a risk factor for the development of psychopathology, which together, have the capacity to severely disturb an individual's sense of self, resulting in disrepair.

1.6 Predictors of Moral Injury

Under morally conflicting conditions, a person may act as an agent of perpetration or witness trusted others committing such atrocities (Drescher et al., 2011; Jinkerson, 2016; Litz et al., 2009). Older age and lower use of emotional distancing strategies have both been shown to predict long-term negative stress reactions following moral challenges such as re-experiencing the moral conflict,

sleep disruption, heightened self-blame, and negative alterations in mood and cognition (Larsson et al., 2018). Further, research has shown that individuals' beliefs about personal responsibility during morally ambiguous situations can contribute towards significant psychological distress including guilt, shame, and rage (Campbell et al., 2016; Fourie, 2015). This is especially relevant for those who work within a professional culture of honour, loyalty, selflessness and personal responsibility (e.g., military members, public safety personnel and other first responders; Gray et al., 2012; Yeterian et al., 2017). Finally, exposure to early maltreatment and neglect could influence risk of sustaining MI later in life, although this remains to be tested directly. For example, early adverse experience has been associated with guilt and shame proneness (Szentágotai-Tətar et al., 2015), and the development of altered blame-related cognitive schemas (Estévez et al., 2017).

1.7 The Assessment of Moral Injury

MI is a multi-faceted construct that spans multiple domains making it difficult to assess (Griffin et al., 2019). Significant limitations have been noted in reference to existing measures of MI, resulting, in part, from conflation of two distinct concepts: MI event exposure and MI symptom expression (Litz & Kerig, 2019). Of the MI scales available, most focus heavily on quantifying exposure to PMIEs rather than quantifying associated symptom distress. For example, the 6-item Moral Injury Events Scale (MIES; Nash, et al., 2013) quantifies exposure and symptom distress related to perpetration-based transgressions; however, it does not measure distress

related to betrayal-based events. Here, the presence of affective betrayal is assessed (*I feel betrayed*…), rather than betrayal-based event exposure or symptom distress (i.e. *I witnessed or learned about a betrayal* / *I am bothered by a betrayal*). Similarly, only four items on the 20-item Moral Injury Questionnaire – Military version (MIQ-M; Currier et al., 2015) measure MI symptom expression while the remaining items measure exposure to PMIEs. The Expressions of Moral Injury Scale – Military version (EMIS-M; Currier et al., 2017) focuses solely on symptom severity, offering separate scores for symptoms relating to self- and other-directed MI events. If perpetration- and betrayal-based MI indeed represent unique pathways to distress (Jordan et al., 2017) then identifying these subtypes during assessment can help to critically inform clinical care. However, this scale is only applicable to military populations limiting its use with other groups. The Moral Injury Symptom Scale – Military version (MISS-M; Koenig et al., 2018) also focuses on MI symptom expression. Items on the MISS-M also appear to capture posttraumatic cognitive appraisals as well as religious beliefs. While the MISS-M represents the most comprehensive assessment of MI developed to date, it too is specific to military populations and contains a number of religious items, thereby limiting generalizability. Thus, a well-validated measure of MI yielding separate scores for event exposure and symptom distress, is needed for both military and civilian respondents.

Importantly, symptom distress related to MI is likely to be captured while assessing for other posttraumatic symptomatology (e.g., CAPS-5). Although MI is

reported, it is not included in the event exposure outlined by CAPS-5 Criterion A (reflective of diagnostic Criteria A for PTSD in the DSM-5), which requires *exposure to actual or threatened death, serious injury, or sexual violence*. Aside from limitations within Criterion A, it is noteworthy that only one CAPS-5 item assesses moral emotions like guilt and shame, and does so alongside fear-based emotions, which are proposed to be mechanistically distinct (Barnes et al., 2019). Considering the critical role of guilt and shame in MI symptom expression, as well as its role in predicting the development of and recovery from PTSD (Bub & Lommen, 2017; Oktedalen et al., 2015), the CAPS-5 appears to be weak in assessing the relative contributions of fear versus negative moral emotions to PTSD symptomatology.

1.8 The Treatment of Moral Injury

Existing psychological interventions are proposed to treat guilt, shame, and other aspects of MI, at least to some extent. These include traditional CBTs, specifically prolonged exposure therapy (PE) and cognitive processing therapy (CPT; Held et al., 2018), as well as third wave CBTs including acceptance and commitment therapy (ACT; Farnsworth et al., 2019; Nieuwsma et al., 2015; Ojserkis et al., 2014), dialectical behaviour therapy (DBT; Görg et al., 2017; Harned et al., 2012), imagery rescripting and reprocessing therapy (IRRT; Alliger-Horn et al., 2016; Grunert et al., 2007), mindfulness-based therapies (Boyd et al., 2018), compassion-based therapies (Au et al., 2017), and spiritually-enhanced treatments (Breuninger et al., 2017; Koenig et al., 2017). Specialized interventions for treating military-

related MI have also been introduced, suggesting military-related groups may have unique treatment needs. These include Adaptive Disclosure (Gray et al., 2017; 2012; Litz et al., 2015), Trauma Informed Guilt Reduction Therapy (TrIGR; Norman et al., 2014), and Project Trauma Support (Dentry et al., 2017).

The appropriateness of traditional front-line treatments for PTSD including PE (Foa & Kozak, 1986; Foa, et al., 1991) and CPT (Resick & Schnicke, 1992) have been questioned in the treatment of MI. These interventions target symptoms of fear, avoidance, ineffective processing of trauma memories, and negative appraisals of traumatic events (Cahill et al., 2012); however, it is well-recognized that the primary response to trauma extends beyond fear-related conditioning and often involves feelings of guilt, shame, anger, disgust, and grief (Beck et al., 2011; Feiring & Taska, 2005; Nazarov et al., 2015; Wilson et al., 2006). While both PE and CPT have been associated with large effect sizes and clinically significant symptom reductions (Cahill et al., 2012; Langkaas et al., 2017; Steenkamp et al., 2015), up to half of patients retain a diagnosis of PTSD following treatment (Langkaas et al., 2017; Resick et al., 2002; Schnurr et al., 2007; Steenkamp et al., 2015) or continue to experience significant symptoms unaddressed by intervention protocols (e.g., Bradley et al., 2005; Owens, Chard, & Cox, 2008). Drop out rates as high as 51% have also been reported for these front-line PTSD treatments (Arntz et al., 2007; Imel et al., 2013). Interestingly, Canadian Armed Forces (CAF) members exposed to PMIEs are *more likely* to seek help than members not exposed to PMIEs (Nazarov et al., 2020), speaking to

the distressing nature of MI. Assuming that at least some of the participants included in the abovementioned PTSD treatment studies were MI-exposed, these high attrition rates also beg the question: why do so many eager treatment-seekers drop out from treatment? Further, due to the nature of MI, issues surrounding privacy and confidentiality may influence the type of treatment provider sought. For example, MI-exposed deployed CAF members are more likely to seek help from their general practitioner, a para-professional, or a non-professional (e.g., friends, family) than a specialized mental health care professional (e.g. psychologist). Additionally, within this group, MI-exposure was associated with a two-fold increase in help-seeking from a civilian practitioner, suggesting some avoidance of the military health care system, perhaps due to broken trust or professional ramifications associated with disclosure (Nazarov et al., 2020). Here, broken trust may also prevent full disclosure to certain providers, particularly when a dual role exists (e.g., clinicians within the military), further challenging the assessment and treatment of morally injured patients. Collectively, these and other findings have raised concern around the efficacy of traditional front-line PTSD treatments for morally injured patients, particularly with respect to whether negative moral emotions are effectively targeted and resolved (e.g., Drescher et al., 2011; Gray et al., 2017, 2012; Litz et al., 2009; Stapleton et al., 2006).

Currently, the biological underpinnings of MI are not well understood, and this additionally limits therapeutic research and outcome (Griffin et al., 2019). Neuroimaging studies have played a critical role in describing PTSD and its

dissociative subtype (Akiki et al., 2018; Harricharan et al., 2016; Lanius et al., 2012; Lanius et al., 2006; Nicholson et al., 2018). This thesis therefore aims to contribute the first neuroimaging data to examine neural activation patterns associated with MI event recall, a common component of traditional and third-wave psychotherapies. We also investigate neural activation patterns associated with two hallmark MI symptoms: MI related shame and negative alterations in cognition and mood (as measured by CAPS-5 Criterion D score). These results are expected to inform appropriate assessment and treatment of MI moving forward.

1.9 Morality in the Brain

Neuroimaging studies of human morality have reported widespread neural activation, reflective of moral cognition, behaviour, and emotion (e.g., Greene, 2001; Moll et al., 2003, 2002; Pascual et al., 2013). Prefrontal areas are commonly implicated in moral processing, including the ventromedial prefrontal (vmPFC) and dorsolateral prefrontal cortices (dlPFC; see Marazziti et al., 2013 for review). Importantly, these cortical structures function as part of large-scale intrinsic connectivity networks (ICNs), which play a fundamental role in organizing the human experience of morality (Laird et al., 2011; Menon, 2011; Seeley et al., 2007). These functionally connected, neural networks include the default mode network (DMN), the salience network (SN), and the central executive network (CEN). These networks co-operate to support higher order social function (Seeley et al., 2007) and by extension, moral cognition. Together, the DMN, CEN and SN help to enable a prosocial, o*ther-oriented* focus along

with feelings of selflessness and compassion (Morishima et al., 2012; Takagishi et al., 2010). For example, the vmPFC, a main hub within the DMN, facilitates personal identification with moral situations, participating in theory of mind (ToM), attribution of intention, empathy, and decision-making (Young & Saxe, 2009). Further, SN regions (e.g., anterior insula, anterior cingulate cortex, amygdale, thalamus, ventral striatum) are thought to critically enable positive moral emotion, and represent areas of the brain that are highly involved in emotional bonding and attachment (Fisher et al., 2013; Insel & Fernald, 2004; Keverne & Curley, 2004; Marazziti et al., 2013).

Norm violation, which is inherent to any MI, has been associated with neural activity in the right insula, dorsolateral prefrontal, and dorsal cingulate cortices – regions responsible also for error monitoring, affective processing and regulation (Heilbronner et al., 2016). When social norms are violated (e.g., fairness, do no harm), negative moral emotion is elicited. Self-blaming emotions, like shame, have 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 (i.e. anterior insula; Bastin et al., 2016; Gilead et al., 2016; Zhu et al., 2019). Here, the dlPFC / CEN serve as a cognitive-affective control, helping to regulate negative self-conscious emotions elicited in response to perceived social norm violations (Zinchenko & Arsalidou,

2018). In summary, ICN activity and connectivity mediate automatic moral and prosocial responses, enabling meaningful interpersonal connection.

1.9.1. Altered ICNs in PTSD

A growing body of evidence has described PTSD-related alterations in ICN activity and connectivity (Akiki et al., 2018; Liu et al., 2017; Nicholson et al., 2017), and these alterations may also contribute towards altered moral processing. Here, we review the PTSD-related alterations that may be relevant to MI event recall.

Prominent alterations in DMN function (e.g., mPFC hypoactivation) have been linked to PTSD-related impairments in self-referential and social cognitive processes, manifesting as difficulties with self-evaluation, self-judgement, autobiographical memory, and sense of self (Frewen et al., 2020; Koch et al., 2016). Indeed, these processes are also critical for processing and integrating morally injurious events and for regulating moral emotion. Further, the DMN enables the "social self", including self-awareness and self-other comparison (Kjaer et al., 2002; Lou et al., 2017), as well as other core processes involved in moral reasoning. Of particular importance, the DMN mediates the ability to perspective-take and determine whether someone has positive or negative intentions (Kliemann et al., 2008; Takahashi et al., 2008), a process likely associated with negative alterations in cognition and mood (i.e. blame-based attributions and beliefs).

Notably, individuals with PTSD have shown increased connectivity between nodes involved in the DMN and SN, possibly reflecting heightened threat-detection (presumably, including moral threat) as well as compromised self-referential processing (Koch et al., 2016). The SN primarily functions to detect, filter, and integrate salient stimuli (Dosenbach et al., 2007; Lovero et al., 2009; Seeley et al., 2007). Here, increased salience information processing inhibits or *interferes* with cortical activity, resulting in *undermodulation* of limbic activity by prefrontal controls (Lanius et al., 2010) – a pattern also correlated with chronic *flight and fight* states (Koch et al., 2016). Conversely, the dlPFC in concert with the larger CEN, is thought to serve as a cognitive control or override, helping to modulate (dampen) SN hyperactivity. Behaviourally, the dlPFC/CEN is activated by critical thinking, working memory, and reasoned analysis or logic to moral situations and challenges (see Mendez, 2009; Zinchenko & Arsalidou, 2018). However, activity within these network regions is compromised in individuals with PTSD, including those with emotional trauma and presumably, some with comorbid MI (Lanius et al., 2015). These alterations can instead result in *overmodulation* of limbic activity by hyperactivity in prefrontal regions (e.g., mPFC, dlPFC) resulting in emotional detachment among other hypoarousal symptoms (Nicholson et al., 2017). This under- / overmodulation pattern has been associated with insula hyper and hypoactivity, respectively. Here, subcortical (limbic) activity is thought to originate from the a small region in the brainstem, known as the periaqueductal grey (Harricharan et al., 2016) and work in concert

with the larger innate alarm system to facilitate quick defensive action (Lanius et al., 2016). We hypothesize that this pattern will extend to MI-exposed individuals when PTSD is comorbid, also influencing symptom expression and challenging memory integration.

1.10 Moral Emotion in the Brain and Body

Emotions are experienced in the body (Damasio & Carvalho, 2013; Nummenmaa et al., 2018; Ogden et al., 2006). According to the *Somatic Marker Hypothesis*, subjective emotions are triggered by the perception of emotion-related bodily states that reflect skeletomuscular, neuroendocrine, and autonomic nervous system changes (Damasio & Carvalho, 2013). Here, physiological changes are perceived as emotion-related bodily sensations, which serve as a basis for subjective, conscious emotion. Emotion, in turn, helps us to better respond to and learn from the environment. Existing evidence suggests that the bodily changes associated with specific emotions are distinct and culturally-universal (Nummenmaa et al., 2014; 2018). These emotion-related somatosensory patterns develop in childhood, becoming discrete over time (Hietanen et al., 2016). Remarkably, emotion-related somatosensory patterns have been scarcely studied in clinically relevant groups likely to experience emotion differently, with the exception of schizophrenia wherein a diffuse pattern of sensation emerged (Torregrossa et al., 2019). Of interest, alexithymia is a subclinical condition characterized by low emotional awareness, and an altered emotional expression and experience, including emotional numbing.

Alexithymia symptoms appear transdiagnostically across psychiatric disorders, including conditions commonly comorbid with MI such as PTSD (Taylor & Bagby, 2004; Treasure & Schmidt, 2013), depression (Frewen et al., 2008; Li et al., 2015), self-harm (Norman & Borrill, 2015), and suicidality (Davey et al., 2018). Moreover, alexithymia symptoms have shown treatment resistance with traditional approaches (Luminet et al., 2018). Alexithymia has been consistently associated with exposure to adverse and traumatic experiences (Frewen et al., 2008), ostensibly extending to PMIEs. Moreover, alexithymia is increasingly associated with intimate partner violence, particularly among trauma-exposed military samples, making it both a consequence and vehicle of trauma (Berke et al., 2017). It is presently unknown how individuals with alexithymia represent emotions in their bodies, and whether altered somatosensory processing may contribute towards the condition. Considering its relevance to MI, we explore further the emotion-specific somatosensory maps in a group with and without alexithymia in Chapter 4.

1.11. The Human Defense Cascade & Tonic immobility

The human defense cascade can drastically alter emotional experience, when innate, survival-based responses are activated. Active (*fight or flight*) and passive (*immobility*) defenses are elicited instinctively, and dynamically, according to conscious and subconscious subjective threat appraisal (Kozlowska et al., 2015). Behavioural responses are mediated by the innate alarm system, a fast, subcortical

neural network that coordinates and facilitates defensive behaviour (Lanius et al., 2016).

Tonic immobility (TI) is a specialized late-stage defense response, elicited by perceived physical or psychological restraint (i.e., perceived inescapability or a sense that personal resources are overwhelmed/overpowered), and/or extreme fear or panic (e.g., Gallup, 1974; Kozlowska et al., 2015; Volchan et al., 2011). TI is characterized by altered sensorimotor and perceptual processes including physical immobility (motionlessness), tense body posture (increased muscle tone), loss of agency, analgesia, depersonalization, and derealisation (Abrams et al., 2009). Critically, TI serves to disconnect an individual from the environment as well as their body, to enable survival (see Chapter 3 for further literature review). Although TI has long been associated with dissociative symptoms in both community and psychiatric samples, here, we suggest it *is* a dissociative response by function. To do so, we conceptualize TI within the defense cascade model of dissociation (Kozlowska et al., 2015; Schauer & Elbert, 2010). Within this model, dissociation increases along a continuum as late-stage defenses are elicited. Thus, TI is driven by parasympathetic dominance and associated opioid-mediated dissociation. TI may be an adaptive strategy under imminent threat (e.g., reduced pain as mediated by the opioid system); however, this state disturbs engagement with the environment, reduces sensory integration (mediated by thalamic relay sites), and may lead to impaired memory encoding and integration (Kozlowska et al., 2015; Lanius et al., 2018; Schauer & Elbert, 2010).

Arguably, persistent and severe blame-related emotion generated by MI may serve as an inescapable and overwhelming internal threat. In such cases, MI could conceivably trigger a TI response post-MI or post-trauma. Specifically, blame-related emotions including guilt, shame, and anger have been associated with reductions in respiratory sinus arrhythmia (RSA; Bleil et al., 2008; Freed & D'Andrea, 2015). Higher shame, for example, has been associated with lower RSA at baseline, as well as during trauma reminders and recovery (Freed & D'Andrea, 2015). Lower RSA is consistent with evidence of sympathetic shut down, characterized by autonomic hypoarousal and dissociative symptomology (Schalinski et al., 2015). Authors have suggested that this physiological response is experienced subjectively as dissonance, or dissociation, between the ideal and 'real' sense of self (Kruger, 2017; Mills, 2005). Taken together, these studies provide evidence that blame-related emotions, particularly when chronically activated, can result in sympathetic shut-down, possibly reflective of posttraumatic TI.

Existing measures of TI, however, are limited by an exclusive focus on peri-traumatic responses (Abrams et al., 2009; Fusé et al., 2007) despite an emerging literature base describing posttraumatic expressions of immobility (e.g., de Kleine et al., 2018). Therefore, in Chapter 3, we evaluate the first comprehensive, self-report measure of posttraumatic TI.

1.12 Dissertation Objectives

In summary, MI is an important predictor of psychopathology including the development of PTSD, depression, and suicidality. Shame is thought to play a critical role in the experience and symptom expression of MI. Despite its documented negative health outcomes, little is known about the biological correlates of MI. From a neurobiological perspective, our understanding and treatment of MI among individuals with PTSD has been limited. To date, no research has reported on neural correlates of active MI event recall – a common component of front-line psychological treatments. Therefore, the primary aim of this thesis, was to expand our understanding of how morally injurious events are recalled and processed in the brain, particularly when PTSD is comorbid. Further, we aim to evaluate a novel measure of post traumatic TI and investigate aberrant patterns of emotion-specific bodily sensations in alexithymia, whereby both TI and alexithymia are hypothesized to be related to MI symptom expression. Finally, in Chapter 5, we present a neuroscientifically-informed framework of embodied MI event processing when PTSD is comorbid.

Chapter 2: Shame on the brain: Neural correlates of MI event recall

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General Purpose

MI has been associated with reduced social function, persistent blame-related emotion, and negative health outcomes including the development of PTSD and suicidality. In this study, we examine the neural activation patterns of MI event recall in military-related PTSD, relative to civilian MI-exposed controls. We also explore the neural correlates of two core MI symptoms: state shame (induced by event recall) and alterations in cognition and mood (measured by CAPS-5 Criterion D Severity Score). Here, we do not attempt to disaggregate between types of MI, or to control for specific blame related emotions (e.g., guilt, shame, anger), but instead, aim to broadly describe the neural correlates associated with the recalling and processing of a complex MI event (Farnsworth et al., 2017).

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.

1.0 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 MI (Litz et al., 2009; Thompson, 2015). MI (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 MI (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 behaviour 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 (C. J. 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; P. 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., 2018b; 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 (Cavanna & Trimble 2006; Bluhm et

al. 2009; Daniels et al. 2010; Van der Kolk, 2014; Tursich et al. 2015; Akiki et al. 2017; Fenster et al. 2018; Frewen et al. 2020). 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 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 towards 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 towards either under- or over-modulation of limbic activity (Lanius et al., 2010; Lanius et al., 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 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 (dlPFC), alongside increased activation within areas associated with salience and viscerosensory 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.

2.0 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 centres. Study procedures were approved by the Health Sciences Research Ethics Board of Western 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 prior to study involvement. For control participants, lifetime psychiatric illness or psychotropic medication served as an additional exclusion criterion. The MI Events Scale (MIES; Nashet 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 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 & fMRI Protocol

Laying supine on the MR scanner bed, participants were exposed first to their neutral memory script, and following a two-minute break to their MI memory script. Scripts were presented sentence by sentence. Sentence 1 was visually displayed for 5s 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 25s. 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. Stimulus presentation was controlled by a PC running E-Prime 3.0 software (Psychology Software Tools, Pittsburgh, PA). 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, Erlangen, Germany) 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=256x256 mm, flip angle=90°, in-plane resolution=2x2 mm). 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 (Welcome Department of Cognitive Neurology, London, UK) within MATLAB R2019. Our standard preprocessing routine included discarding 4 initial volumes, re-orientation 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, Cambridge, MA), 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 modelled 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 ANOVA in order 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 *p*-FDR < .05 k =10, observed at the cluster-corrected level in order to control for multiple comparisons (see Eklund et al., 2016) with the initial uncorrected cluster-forming threshold in SPM set at $p < .001$, k=20.

3.0 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, closer examination of the influence of age on the BOLD signal was found to reveal non-significant 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.

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

Asterisks indicate significantly higher clinical symptom values in the trauma-exposed control group. Abbreviations: PTSD= Posttraumatic Stress Disorder, CAPS = Clinician Administered PTSD Scale for DSM-5, CTQ = Childhood Trauma Questionnaire, MDI = Multiscale Dissociation Inventory, MDD = Major Depressive Disorder.

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=0.055, k= 71) and the left dlPFC [MNI= -32 46 8, F=21.38, Z=4.21, pFDR cluster corrected=0.055, 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 dlPFC, 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, 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).

Table 2. Independent Samples t-Tests

Condition	**Comparison**	**Brain Region**	**H**	**Cluster Size**	**BA**	**MNI Coordinate**			**t Stat.**	**Z Score**	***p-FDR cluster level***
						x	**y**	**z**			
MI	PTSD > Control	Dorsal Anterior Cingulate Cortex		175	32	-2	32	14	5.56	5.13	.001
		Dorsolateral PFC	L	133	8,9, 10	-38	46	10	4.46	4.23	.005
		Posterior Insula	R	106		38	-14	-6	4.37	4.15	.011
		Postcentral Gyrus	L	69	40	-64	-22	18	4.02	3.84	.049
	Control > PTSD	*Ns*									
Neutral	PTSD > Control	*Ns*									
	Control > PTSD	*Ns*									

Direct group comparisons for the MI 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: PFC = prefrontal cortex, H = hemisphere, BA = Brodmann area, FDR = false discovery rate correction, PTSD = posttraumatic stress disorder.

Table 3. Clinical Correlation Analysis

Measure	**Group**	**Condition**	**Correlation**	**Brain Region**	**H**	**Cluster Size**	**BA**	**MNI Coordinate**			**t Stat.**	**Z Score**	***p-FDR cluster level***
								x	**y**	**z**			
Shame	PTSD	MI Recall	Negative	Superior Frontal Gyrus	R	102	8	26	12	48	5.66	4.50	.021
Alterations in Cognitions and Mood	PTSD	MI Recall	Negative	Posterior Insula	L	92		-40	-18	12	6.45	4.94	.033

Correlations between neural activation during MI 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).

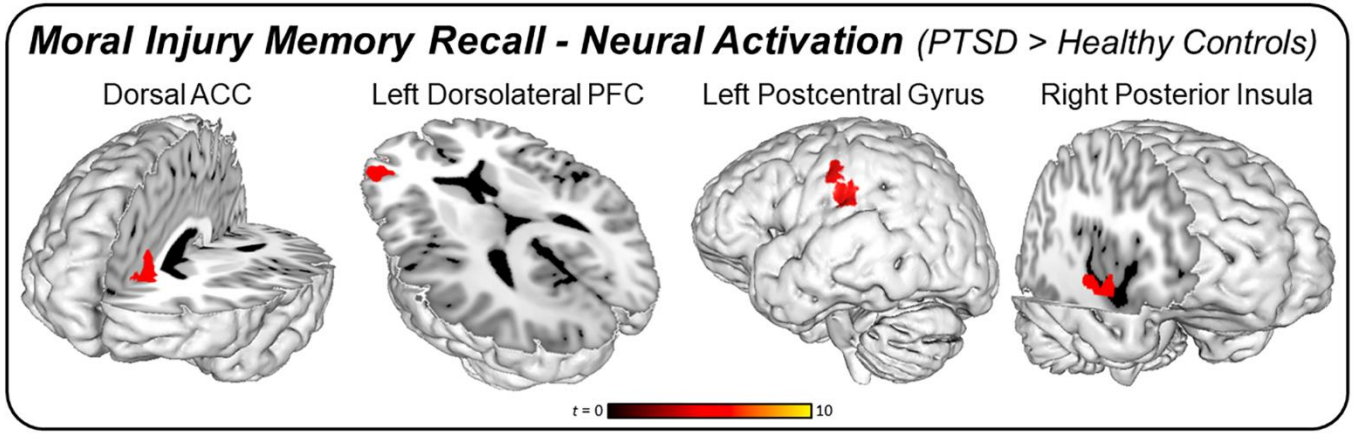

Figure 1. Brain areas showing greater activation in the PTSD group as compared to MI-exposed controls during MI memory recall. Neural activation results are reported at the FDR-cluster corrected threshold ($p<.05$, $k=10$). Abbreviations: PFC = prefrontal cortex, PTSD = posttraumatic stress disorder, ACC = Anterior Cingulate Cortex.

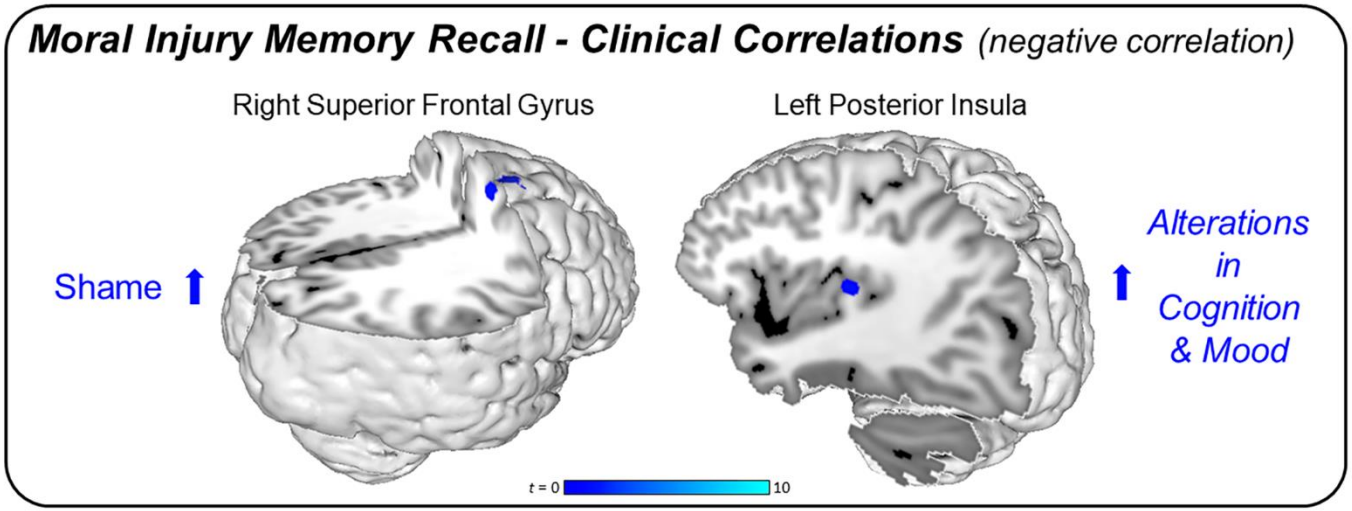

Figure 2. Brain areas showing significant correlations to clinical PTSD symptoms in the PTSD group during MI memory recall. Trait measures of PTSD clinical symptoms: symptoms of altered cognitions and mood (CAPS cluster D), were negatively correlated to the left posterior insula during MI recall. Paradigm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

4.0 Discussion

Our findings point towards 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 salience network nodes (right posterior insula, dACC), regions involved in defensive responding (left postcentral gyrus), as well as hyperactivity in brain areas responsible for top-down cognitive control of emotions (left dlPFC). 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 induction of basic negative emotions such as anger (Gilead 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 dlPFC/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 processes including the processing of negative self-conscious emotions, self-evaluation, self-judgement, 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-occuring with decreased SFG acitvation 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 hypoactivity in the posterior insula is associated with altered socio-emotional processing (Akiki et al., 2017; Koch et al., 2016).

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 towards 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.

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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Chapter 3: Assessing post-traumatic tonic immobility responses: The Scale for Tonic immobility Occurring Post-trauma (STOP)

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General Purpose

The previous chapter provided preliminary evidence showing that the presence of PTSD significantly alters MI event recall, relevant to MI-exposed controls. Aberrant somatosensory (viscerosensory) processing appears to underpin MI and associated persistent blame-related emotion. Constant high-burden emotions, like shame, could conceivably trigger post-traumatic tonic immobility (TI), a late-stage defense response prompted by inescapable or otherwise overwhelming stress. Thus, the following study was performed to evaluate the STOP - a novel self-report measure of post-traumatic TI characterized by sensorimotor and perceptual alterations, somatic detachment, memory difficulties, residual symptoms, and negative self-blaming affect including guilt and shame.

Abstract

Background: Peri-traumatic tonic immobility (TI) has been associated with the development and course of posttraumatic stress disorder (PTSD). Despite serving as an adaptive late-stage defense response, TI that continues in response to post-traumatic reminders may lead to reduced functioning and a diminished sense of well-being. At present, no validated self-report measures assess post-traumatic TI responses specifically.

Methods: The primary objective of the present study was to evaluate the Scale for Tonic immobility Occurring Post-trauma (STOP), the first self-report measure developed to assess for the presence and severity of TI responses that persist following trauma exposure as part of post-traumatic symptomatology. Trauma-exposed clinical and non-clinical participants (N = 462) with a history of TI completed a demographic questionnaire, the STOP, and measures of post-traumatic symptoms, dissociation, anxiety and depression.

Results: STOP assessed four latent constructs, which were interpreted following the human defense cascade model. Together, these factors capture the sensorimotor and perceptual alterations, and dissociative experiences, associated with post-traumatic TI as a trauma-related altered state. Residual symptoms and the experience of negative affect following this response (including guilt and shame) are also represented. STOP scores demonstrated excellent reliability, as well as good construct and convergent validity, with other measures of

dissociation and PTSD. Results from the present study suggest TI is most consistent with other dissociative post-traumatic symptomatology.

Conclusions: STOP demonstrates excellent preliminary psychometric properties and may be useful for researchers and clinicians wishing to assess chronic forms of TI across trauma-exposed, clinical and community samples.

Introduction

Humans, like other animals, exhibit a series of active and passive defense responses, which are prompted when exposed to threat (Blanchard & Blanchard, 1989; Fanselow, 1994; Kozlowska et al., 2015; Vila et al., 2007). The defense cascade model (Schauer & Elbert, 2010) provides a key evolutionary framework for understanding how an individual defends against varying levels of attack. Defense responses are dynamic, and elicited according to subjective appraisal of threat in relation to the level of personal power required to overcome threat (Schauer & Elbert, 2010). Passive defense responses are thought to serve as a last line of protection when threat is greatest and escape least possible (Kozlowska et al., 2015). Tonic immobility (TI) is an important passive defense response characterized by alterations in experience including physical immobility (motionlessness), tense body posture (increased muscle tone), loss of agency, analgesia, depersonalization, and derealisation (Abrams et al., 2009). TI, sometimes referred to as freezing, differs from the momentary orienting response thought to precede defensive responding (Kozlowska et al., 2015). In sharp contrast with active avoidance behaviour, immobility responses are understood as a set of evolutionarily-derived, defensive reflexes activated in response to overwhelming, unavoidable, threat.

Despite serving as an adaptive acute defense response, peri-traumatic TI is related to increased psychological impairment over the long-term. It has been associated with the development and severity of posttraumatic stress disorder

(PTSD) in survivors of physical (Maia et al., 2015) as well as of sexual and non-sexual psychological trauma (Möller et al., 2017; Mooren & van Minnen, 2014; Rocha-Rego et al., 2009). In a sample of men and women with PTSD, for example, peri-traumatic TI was a better predictor of PTSD development and prognosis than peri-traumatic panic (Lima et al., 2010). Moreover, peri-traumatic TI responses have been associated with poor treatment response to front-line pharmacological interventions for PTSD (Fiszman et al., 2008; Lima et al., 2010).

Despite knowledge of the impact peri-traumatic TI may have on the development and course of PTSD, little is known about post-traumatic TI. Recent work suggests that post-traumatic symptomatology is influenced by peri-traumatic responses (Aho et al., 2017; Gandubert et al., 2016; Sugar & Ford, 2012). Evidence suggests a substantial proportion of individuals who immobilize peri-traumatically experience TI in response to post-traumatic reminders (de Kleine et al., 2018). Several authors suggest that exposure to traumatic reminders may re-instantiate the original cascade of defensive stages, resulting in post-traumatic symptomology (de Kleine et al., 2018; Keane et al., Caddell, 1985; Schauer & Elbert, 2010). Consistent with this notion, prior TI experiences have been recently linked to increased TI proneness in both healthy controls and individuals with PTSD during stressful stimuli presentation (Fragkaki et al., 2016). TI proneness has also been related to prior adverse experience and insecure childhood attachment in samples of healthy female and adolescent participants (Hagenaars et

al., 2012; Niermann et al., 2015). Here, TI may serve as a defensive response that persists following initial (or repeated) trauma exposure.

TI responses that continue to be expressed over the long-term may prove detrimental to one's health and social functioning. This physiologically taxing event serves vitally to disconnect an individual from normal waking experience, imposing a potent form of interpersonal avoidance. Over the long-term, this non-responsive state may undermine the healing potential of interpersonal connection (Bryant, 2016). Accordingly, authors have called for research investigating the relation between chronic TI responses and psychopathology (Fragkaki et al., 2016). Unfortunately, to date, no self-report measures assess specifically for post-traumatic TI, hindering research on this topic. In the present study, we evaluate the factor structure and psychometric properties of the first self-report Scale for Tonic immobility Occurring Post-trauma (STOP).

Tonic Immobility

Biological indicators consistent with TI states in mammals have now been reported in humans, and are proposed to be homologous (Möller et al., 2017). For example, following exposure to an inescapable biological stressor (20-second inhalation of 20% CO2/balance O2), participant-reported feelings of immobility more than doubled (Schmidt et al., 2008). Moreover, whereas trauma-exposed community members demonstrated heart rate deceleration and decreased body sway when shown threat-based pictures depicting no means of escape, amplitude of heart rate and sway increased when a clear escape route was depicted (Bastos et

al., 2016). In addition to significant reductions in body sway (Azevedo et al., 2005; Bastos et al., 2016; Fragkaki et al., 2017; Gladwin et al., 2016; Hagenaars et al., 2012; Roelofs et al., 2010; Stins & Beek, 2007; E. Volchan et al., 2017), TI responses have been associated with increased muscle stiffness (Azevedo et al., 2005). Changes in heart rate during TI responses have also been reported, although results remain equivocal. Specifically, whereas one body of evidence suggests TI is characterized by tachycardia or heart rate acceleration (Volchan et al., 2017), another suggests it is associated with bradycardia or heart rate deceleration (Hagenaars et al., 2014). It is possible these conflicting data reflect the simultaneous activation of two opposing parts of the autonomic nervous system, sympathetic and parasympathetic, as observed in animal models of TI (Hagenaars et al., 2014; Roelofs, 2016; Vila et al., 2007).

Dissociation is defined as a disturbance in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behaviour (APA, 2013). Critically, dissociation is thought to increase alongside parasympathetic activity as an organism progresses through the cascade, from active to passive responses (Fenster et al., 2018; Schalinski et al., 2015; Schauer & Elbert, 2010). Here, dissociation serves a highly adaptive purpose in TI: an individual may endure unremitting threat, and injury, by becoming unresponsive to the environment (Kozlowska et al., 2015; Ogden et al., 2006).

Measures of Post-traumatic Tonic immobility

Current measures assessing peri-traumatic TI (Abrams et al., 2009; Fusé et al., 2007), do not assess responses that continue to occur post-traumatically outside the context of an acute traumatic incident. A few measures assay aspects of post-traumatic TI; however, a comprehensive self-report measure is presently lacking. Whereas the Somatoform Dissociation Questionnaire (SDQ-20) evaluates the severity of somatoform manifestations of structural dissociation (Nijenhuis et al., 1996), the STOP focuses on the post-traumatic TI response itself, as an expression of the human defense cascade. Although the SDQ-20 assesses some symptoms broadly consistent with TI (e.g., item 19: I am paralysed for a while; item 20: I grow stiff for a while), other items pertain to symptoms that extend beyond what is expected to occur during post-traumatic TI (e.g., item 2: I dislike tastes that I usually like; item 10: I feel pain in my genitals; item 17: I cannot sleep for nights on end, but remain very active during daytime). Furthermore, the SDQ-20 fails to adequately assess relevant aspects of depersonalization thought to occur during TI responses (i.e., out of body experience).

The Shutdown Dissociation Scale (Shut-D) (Schalinski et al., 2015) represents the first theoretically derived measure to focus on post-traumatic "shut down" symptomatology within a dissociative framework. The Shut-D assesses dissociative symptoms thought to occur alongside parasympathetic dominance ("shut down") during late-stage defense responding (Schalinski et al., 2015). Unfortunately, the Shut-D is administered as a structured clinical interview,

limiting its feasibility across settings. Conversely, the STOP represents the first self-report measure to capture TI specifically as part of this parasympathetic cascade.

Critically, neither the SDQ-20 nor the Shut-D considers the potential residual symptoms or affect associated with post-traumatic TI. Given patient report and emergent literature, it is essential that tools assessing post-traumatic TI include items that measure non-fear primary responses to trauma, including guilt and shame (Badour et al., 2015).

The Present Study

In the present study, we evaluate the factor structure and psychometric properties of STOP, designed as the first self-report measure of post-traumatic TI. STOP assesses for the presence and severity of altered sensorimotor and perceptual experiences associated with TI occurring more than one month after acute trauma in response to traumatic reminders. Unlike the SDQ-20 and Shut-D, STOP items additionally assess the physical and emotional consequences of chronic TI, further facilitating clinical recognition and future research. Specifically, STOP was developed to inform treatment planning, guide the development of targeted interventions, and result in a better understanding of this altered state.

We predicted that STOP scores would be strongly and positively associated with other measures of post-traumatic symptom severity and dissociation. As persistent TI responses have been linked with state (Niermann et

al., 2015) and trait (Schmidt et al., 2008) anxiety as well as severe depression (Möller et al., 2017), we also expected STOP scores to be positively associated with measures of anxiety and depression.

Methods

Participants

North American participants (N pooled = 713) were recruited using two validated web-based, crowd-sourcing platforms (CrowdFlower; Amazon's Mechanical Turk) across two independent rounds of data collection ($N_{Crowdflower}$= 340; N_{MTurk}= 373) (Chandler & Shapiro, 2016; Frewen et al., 2015). Participants voluntarily responded to an online advertisement, self-identifying based on the following eligibility criteria: i) no history of head injury (no loss of consciousness due to physical trauma, lack of oxygen, or electric shock), ii) at least one episode of TI in response to a past traumatic event occurring more than one month prior, and iii) at least one episode of TI in the past month, in response to reminder(s) related to the original traumatic event (i.e., post-traumatic TI).

Participants who completed less than one third of the survey battery were excluded from analyses. Data were manually inspected and a total of 251 participants were excluded based on one or more of the following reasons: incomplete data (n = 170), incoherent response (n = 72), suspected random responding (e.g., entered 1's for all responses even when not applicable; n = 4), duplicate entry (n = 3), or self-reported head injury (n = 2). Excluded participants did not differ significantly from those retained on any demographic measure.

Remaining participants (N pooled = 462) provided demographic information (Table 1). Participants (54.8% female) were generally of middle age (M = 34.33, SD = 10.29), ranging from 18-65 years old. Ninety participants reported a peri-traumatic TI response which occurred less than one month prior to participation, and were therefore excluded from these analyses pertaining to post-traumatic TI responses. Thus, 372 participant responses were retained for subsequent analyses ($N_{Crowdflower}$ = 181; N_{MTurk} = 191).

Table 1. Demographic and Psychological Characteristics

Characteristic	%	(*N* = 462)
Gender		
Female	54.8%	(253)
Male	44.8%	(207)
Choose not to say	0.4%	(2)
Ethnicity		
Caucasian	63.9%	(295)
African American / Canadian	8.4%	(39)
Latin American / Canadian	7.1%	(33)
Asian American / Canadian	7.1%	(33)
Native American / Canadian	3.9%	(18)
Mixed Race	3.9%	(18)
Other	4.1%	(19)
Choose not to say	1.5%	(7)
Marital Status		
Single	43.9%	(203)
Common-Law or Married	48.0%	(222)
Divorced	6.5%	(30)
Other	1.1%	(5)
Choose not to say	0.4%	(2)
Education		
< High School	0.9%	(4)
High School	12.8%	(59)
Some Post-Secondary	25.8%	(119)
University Degree	31.2%	(144)
College Diploma	16.2%	(75)
Graduate or Professional School	12.6%	(58)
Other	0.6%	(2)
Employment		
Part-time or Full-time	69.7%	(322)
Self-employed	12.6%	(58)
Unemployed	8.2%	(38)
Not able to work	1.9%	(9)
Student	5.8%	(27)
Other	1.7%	(8)
Psychiatric Diagnosis		
Yes, currently	16.2%	(75)
Yes, in the past but not currently	10.6%	(49)
Never	69.7%	(322)
Choose not to say	3.5%	(16)

Measures

Scale for Tonic immobility Occurring Post-trauma (STOP). STOP represents a novel, self-report measure informed by first-person accounts regarding the phenomenology of TI. STOP items were developed based on themes identified across first-person reports (N = 6), descriptions in the literature, and clinical expertise. STOP includes constructs described in peri-traumatic TI scales, such as fear, dissociation, and physical immobility (Abrams et al., 2009; Fusé et al., 2007). Additionally, this measure was designed to capture the complex sensory-perceptual, cognitive, behavioural, and affective experiences preceding, during, and following a post-traumatic TI response. Initially, 41 items were categorized across 15 domains: Fear/Panic, Analgesia, Alterations in Breathing, Alterations in Voice, Alterations in Vision, Alterations in Hearing, Physical Immobility, Loss of Agency, Emotional Detachment, Feelings of Safety, Altered Perception of Time, Altered Sense of Self, Altered Cognition, Sleep, and Collapsed Immobility. Respondents were asked to rate their past-month TI experiences on a 5-point Likert-type scale (0=Never; 4=Extremely). STOP items refer to "freezing" rather than "tonic immobility" to increase accessibility to the intended lay audience (see Appendix A for scale).

Other Self-Report Measures

PTSD Checklist for DSM-5 (PCL-5). The 20-item PCL-5 (Blevins et al., 2015) was administered to all participants to measure past-month PTSD symptoms. Scores range from 0-80, with higher scores indicating greater PTSD

severity (probable PTSD ≥ 38). The PCL-5 has demonstrated strong psychometric properties including high internal consistency (α > .91), and good convergent and discriminant validity (Blevins et al., 2015). Ten additional items were appended (Frewen et al., 2015) to assess for dissociative experiences related to trauma-related altered states of consciousness (TRASC) (Frewen & Lanius, 2014).

Patient Health Questionnaire (PHQ-4). The 4-item PHQ-4 (Kroenke et al., 2009). was administered to all participants to measure symptoms of anxiety and depression. Scores on this abbreviated inventory range from 4-16. The PHQ-4 has demonstrated good construct validity and reliability (α =.85) (Kroenke et al., 2009).

Peri-traumatic Dissociative Experiences Questionnaire (PDEQ). A 7-item abbreviated version of the PDEQ (Engelhard et al., 2003; Marmar et al., 1997) was administered in the first round of data collection only ($N_{Crowdflower}$ = 181). The PDEQ measures dissociation at the time of a traumatic event and has well-established psychometric properties (Birmes et al., 2005). Scores range from 7-35, with higher scores indicating greater peri-traumatic dissociation.

Dissociative Symptoms Scale (DSS). The 20-item DSS (Carlson et al., 2016) was also administered in the first round of data collection only (NCrowdflower = 181). The DSS measures moderately severe levels of depersonalization, derealization, gaps in awareness or memory, and dissociative

re-experiencing in clinical and non-clinical populations ($\alpha > .87$) (Carlson et al., 2016).

Procedure

This study was approved by the Western University Ethics Board for Health Sciences Research (#108288). To preserve participant anonymity and confidentiality, data collection occurred on a secure, encrypted website (Qualtics) independent of crowd-sourcing access sites.

Participants who self-identified as eligible for the study were presented with a letter of information and provided written consent prior to completing survey material. Consenting participants completed demographic information, followed by the STOP and additional measures of PTSD, dissociation, anxiety and depression. The location of items and response scales were varied to ensure participants were attending to item content and response quality. Participants included in the final sample responded to validity items accurately (e.g., "What year is it?"). Participants were provided with a code to tender $1 compensation comparative to rates offered by similar studies.

Statistical Analysis

We used a stepwise approach to determine the ideal item-count and factor structure of the 41-item STOP. First, exploratory factor analysis (EFA) was conducted with a principal axis extraction and Promax (oblique) rotation. Second, exploratory structural equation models (ESEM) were conducted with a maximum likelihood estimator and a target rotation with cross-loadings specified to

approximately zero. Using these two methods, items with strong cross-loadings (i.e. > .40) were removed. Finally, we tested the suitability of a simple structure to STOP with confirmatory factor analysis (CFA) with maximum likelihood estimation and robust standard errors. EFA procedures were conducted using SPSS software, and ESEM and CFA procedures were performed in MPlus version 7.4 (Muthén & Muthén, 2015).

Factor structure was explored initially using EFA and ESCM in Sample 2 (n MTurk = 191) and testing the resultant model for fit (CFA) in Sample 1 (Crowdflower = 181). The opposite procedure was also conducted (i.e., EFA on Sample 1, CFA Sample 2). Results of these cross-validation analyses yielded consistent factor structures in Samples 1 and 2. Since a near-identical factor structure emerged when considering the total (pooled) sample, the results reported here will focus on the total sample.

Results

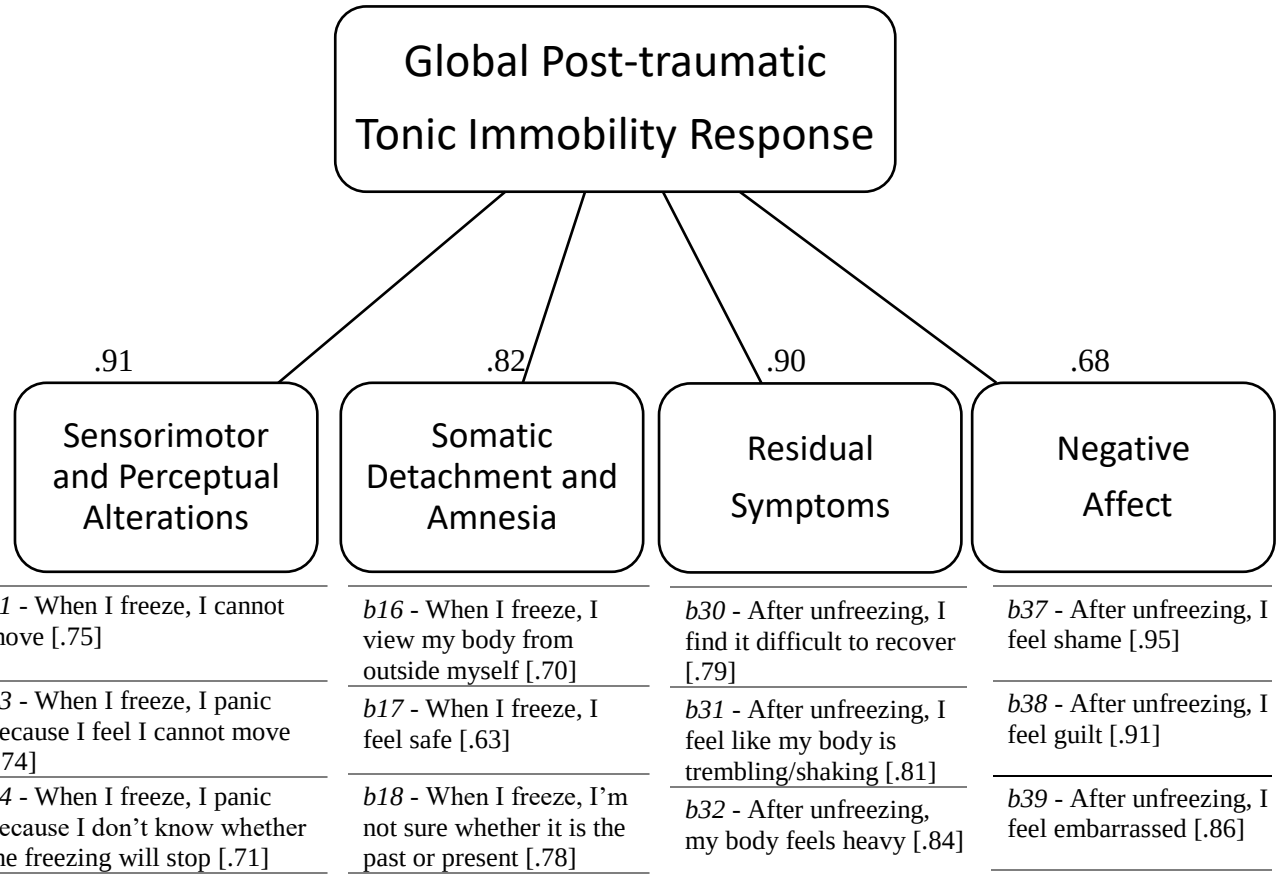
Exploratory Analysis

Factor loadings and intercorrelations of EFA conducted on the 41-item STOP were examined. Six latent variables emerged with an eigenvalue greater than one, rendering a solution accounting for 65.3% of the variance. Each eigenvalue was compared to the 95th percentile of a sampling distribution of 100 randomly generated eigenvalue correlation matrices. Four factors emerged before the randomly generated eigenvalues exceeded those from our data, accounting for 59.7% of the variance. Pattern and structural matrices were used to interpret the

factor structure, with items supporting the following factor labels (% variance explained): 1. Sensorimotor and Perceptual Alterations (45.5%); 2. Somatic Detachment and Amnesia (6.2%); 3. Residual Symptoms (4.8%); and 4. Negative Affect (3.1%; Figure 1).

Due to inconsistent factor loadings, three items were removed from the final scale versions (b2, b22, b40) and excluded from CFA analyses. Factors were strongly and positively correlated in the factor correlation matrix (r's between .4 and .7). Therefore, in CFA analyses, a hierarchical structure was tested.

Figure 1. Hierarchical factor structure and standardized factor loadings of the four-factor CFA model 2.



Confirmatory Factor Analysis

Using a hierarchical 4-factor structure, CFA metrics suggested an acceptable model fit. Model fit was assessed by evaluating the global fit indices, including the Root Mean Square Error of Approximation (RMSEA), Standardized Root Mean Residual (SRMR), Tucker-Lewis Index (TLI) and Comparative Fit Index (CFI) (Hu & Bentler, 1999; Marsh et al., 2004). Minimal modifications were made to improve overall fit by allowing one pair of items to correlate residuals (Table 2). Each of the four factors loaded strongly on its higher-order parent factor, supporting the rationale for a global score calculation, in addition to subscale scores (see SI for scoring procedure).

Table 2. Tested Models and Associated Fit Indices of the STOP with CFA (n = 372)

Model	$\chi^2_{(df)}$	BIC	RMSEA [CI]	CFI	TLI	SRMR
STOP						
1. Base 4-Factor	1299.55 (430), $p < .0001$	36075.85	.074 [.069-.078]	.89	.88	.06
2. Base 4-Factor – allow Correlated Residual Items: *b3* with *b4*; Eliminate item: *b40*	1101.99 (400), $p < .0001$	34788.56	.069 [.064-.074]	.91	.90	.06

Notes. BIC = Bayesian Information Criterion; RMSEA = Root Mean Square Error of Approximation; CI = Confidence Interval 95%; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; SRMR = Standard Root Mean Square Residual. The traditional "gold standard" RMSEA threshold of 0.05 or less (Hu & Bentler, 1999) has been identified as often too stringent, affecting the generalizability of the model (Marsh et al., 2004). Therefore, acceptable model fit was evaluated by adopting the following standards: RMSEA < .08, SRMR < .08, TLI > .85 and CFI > .85.

Reliability and Validity

Internal Consistency of STOP Subscales

STOP subscales demonstrated excellent internal consistency (Table 3); Chronbach's alpha coefficients could not be improved through item deletion.

Construct & Convergent Validity

Controlling for age and gender, STOP demonstrated good construct validity, and good convergent validity with other measures of traumatic stress and dissociative symptomatology (Table 3).

Discriminant Validity

Subscale and global STOP scores demonstrated strong positive associations with other measures of dissociation and PTSD symptomatology (Table 3). In descending order, the strongest association was found between global STOP scores and TRASC scores. The association was slightly reduced when current PTSD symptoms were also considered (PCL + TRASC items), and reduced further when considering PTSD symptoms alone (PCL-20 items only). A positive correlation between global STOP scores and current dissociative symptoms (DSS) was observed, in addition to a weaker but positive association with peri-traumatic dissociative experiences (PDEQ). Finally, a weak positive correlation was found between global STOP scores, anxiety and depression (PHQ-4).

Table 3. Descriptive Statistics for STOP subscales and correlations with symptom measures

	M	**SD**	**α**	***r*** **PCL**	***r*** **TRASC**	***r*** **PCL+**	***r*** **DSS**	***r*** **PDEQ**	***r*** **PHQ-4**
Global Post-traumatic Tonic Immobility Response	52.74	28.30	.96	.50**	.56**	.53**	.42**	.39**	.14*
Sensorimotor and Perceptual Alterations	2.11	1.01	.92	.38**	.43**	.41**	.32**	.36**	.10
Somatic Detachment and Amnesia	1.29	1.09	.93	.47**	.60**	.53**	.46**	.32**	.04
Residual Symptoms	2.08	1.22	.90	.44**	.42**	.45**	.36**	.44**	.27**
Negative Affect	1.68	1.43	.93	.44**	.45**	.45**	.31**	.32**	.23**
Cronbach's α	-	-	-	.97	.97	.98	.97	.85	.90

Notes. r_{PCL} = PTSD Checklist - 20 item version, r_{TRASC} = Ten items designed to measure trauma-related altered states of consciousness (TRASC), r_{PCL}+ = PTSD Checklist - 20 item version + 10 TRASC items. * $p < .01$. ** $p < .0001$.

Of note, 51.7% (n = 166) of respondents completing the PCL-5 (n = 321) met criteria for probable PTSD (PCL-5 ≥ 38). Of the respondents meeting probable PTSD criteria, 63.8% (n = 106) indicated additional symptoms consistent with the PTSD dissociative subtype. The latter was quantified by endorsement of at least moderate distress (≥ 3) over the past week on one or more of the following items: (i) derealisation, ii) out-of-body experience, or iii) disturbed body ownership. Given these findings, we suggest that TI may be a dissociative phenomenon relevant to community and psychiatric samples.

Discussion

Peri-traumatic TI has long-term implications for functioning and well-being. Accordingly, it is critical that TI responses expressed as part of post-traumatic sequelae be identified to facilitate treatment. In contrast to the assessment literature examining peri-traumatic TI, little formal research has been conducted to inform the assessment of post-traumatic TI. We tested the first self-report measure of post-traumatic TI in two trauma-exposed samples. Overall, STOP demonstrated excellent psychometric properties, providing an important step towards validation. Internal consistency was shown to be excellent for all subscales. Good construct and convergent validity was demonstrated with other measures of post-traumatic stress and dissociation. Interestingly, global STOP scores were most strongly associated with other trauma-related dissociative symptoms. Less significant associations were found with non-dissociative PTSD symptom severity and peri-traumatic dissociative symptoms. This pattern of

results is appropriate given that we would expect post-traumatic TI to be more closely associated with present-day dissociative symptomatology than past peri-traumatic dissociative responses.

Notably, subscale items clustered together to describe the phenomenological experience and aftermath of post-traumatic TI responses. Items belonging to Sensorimotor and Perceptual Alterations and Somatic Detachment and Amnesia describe alterations in consciousness consistent with the transition to, and experience of, post-traumatic TI. STOP improves upon existing measures by also quantifying the consequential effects (Residual Symptoms) and emotional impact (Negative Affect) relevant to TI responses.

The Onset and Maintenance of Tonic immobility

The Sensorimotor and Perceptual Alterations and the Somatic Detachment and Amnesia subscales contain items that describe alterations in experience that accompany the onset and maintenance of TI. This transition may include disturbances in somatic experience (e.g., b1: 'I cannot move'); emotional experience (e.g., b12: 'I feel numb'); and perception of time (e.g., b20: 'it feels like the experience lasts for a long time'; Frewen & Lanius, 2014). Items describe loss of speech as this function is secondary to respiration (i.e., b5: 'I take short quick breaths'; b8: 'I cannot speak even if I wanted to'), along with seemingly adaptive alterations in vision (i.e., b10: 'my vision becomes narrowed'; b9: 'I am still able to move my eyes'; Lojowska et al., 2015). The final stages in the defense cascade, beyond TI, are thought to result in eventual loss of consciousness

(Schalinski et al., 2015; Schauer & Elbert, 2010). Two items pertaining to a flag and faint response (b26: 'my muscles go limp', b27: 'I collapse to the floor') were retained as part of the Somatic Detachment and Amnesia subscale, providing an indicator of response severity.

STOP items reflect an updated conceptualization of the human TI response as an evolutionarily adaptive defense response. TI and dissociation are conceptualized traditionally as separate constructs that co-occur frequently in response to acute stress (Abrams et al., 2009). Some authors caution against a unified conceptualization of these constructs (i.e. attributing dissociative symptoms to the cognitive experience of TI) (Lanius et al., 2014). Schauer & Elbert (2010) hypothesize that sympathetic and parasympathetic nervous system activity underpin the hyperarousal and "shut-down" symptomatology observed, respectively, in PTSD and its dissociative subtype. In our own work, we posit that TI is characterized by parasympathetic dominance and by associated dissociative symptoms. As a late-stage defensive response, these dissociative symptoms function to disengage and protect the individual from injury (Dalenberg et al., 2012; Schauer & Elbert, 2010). Accordingly, a number of items within this subscale represent depersonalization symptoms (e.g., b16: 'I view my body from outside myself'). Dissociating the body from conscious awareness is inherent to TI, and can function as an adaptive strategy under imminent threat (e.g., reduced pain). This state, however, also serves to disturb integration of the traumatic experience with one's sense of self (Spiegel et al., 2013). For example, TI was

endorsed as evoking feelings of safety (b17: 'I feel safe') and amnesia (b34: 'I have difficulty remembering the experience'), possibly reflecting the protective function of TI.

Residual Symptoms of Tonic immobility

The Residual Symptoms subscale refers largely to the aftermath of TI (e.g., b33: 'After unfreezing my muscles hurt'). Here, items reflect the heightened impact post-traumatic TI has on the brain and body, following transitions between TI responses and normal-waking states of consciousness (e.g., b30: 'After unfreezing, I find it difficult to recover').

The aftermath of post-traumatic TI was further captured on the Negative Affect subscale, which indexes social-moral feelings of guilt, shame, and/or embarrassment that may emerge following an episode (e.g., b37: After unfreezing, I feel shame). TI involves a loss of agency and a failure to act, both of which may represent socially violating behaviour capable of eliciting negative social-moral emotion (Tangney et al., 2007). Guilt is elicited by negative evaluation of specific action(s), motivating approach behaviour intended to repair the wrongdoing; shame serves to condemn the self thereby motivating self-evaluation and withdrawal (Tangney et al., 1996). Both shame and dissociation have been identified as forms of interpersonal avoidance (Dorahy, 2010), serving ultimately to increase social distance (e.g., alienation). We propose that shame, like TI, serves as a functional and protective response to threat. Any social avoidance that may result is better viewed as a secondary effect of a response

intended to prevent further social harm, ridicule, and/or condemnation. Moreover, embarrassment is related directly to the response of an actual or perceived audience who witnesses the humiliating behaviour (Bastin et al., 2016). An individual who immobilizes in front of others, for example, may worry about their social image thereby motivating efforts to preserve reputation, rather than the reparative and avoidant efforts seen in guilt and shame, respectively (Tangney et al., 1996).

Limitations

Although provocative, the present study has a number of limitations including a reliance on retrospective, self-report data. As participants were invited to self-select based on at least one previous TI experience, selection bias may contribute to the present findings, limiting generalizability to other samples. Further limiting these findings, qualitative information about the measure was not collected in this sample. Crowd-sourcing platforms are increasingly used in studies of PTSD, however, it is not well understood how data quality compares to in-vivo methods and results should be interpreted with caution. Although the STOP is designed to measure post-traumatic TI responses, we are unable to definitively link this experience to trauma without future examination. In the present study, the STOP was validated in two independent, trauma-exposed (post-trauma) samples. Research utilizing multi-method and multi-informant approaches is required to determine convergent and discriminant validity, and to strengthen evidence for the structural model developed in this study.

Future Directions

The present findings suggests a need to consider TI items in measures of post-traumatic symptomatology and dissociation used in clinical and research settings. It will be important to understand the role of shame and guilt in relation to TI moving forward. Relations between sex, gender and TI, in addition to clinical and cultural differences in the prevalence and expression of TI responses, should be examined (Sallin et al., 2016). Evidence suggests that aversive stimuli can produce TI rather than active defensive action, as the former is more adaptive than the latter (Azevedo et al., 2005). Future research should therefore examine the relation between intrusive symptoms and post-traumatic TI responses in individuals with and without a prior history of peri-traumatic TI. Future work is needed to explore the intersection between TI and the dissociative subtype of PTSD, and other dissociative phenomena. Finally, dissociative symptoms consistent with post-traumatic TI responses (e.g., depersonalization, analgesia, amnesia) present transdiagnostically across psychiatric disorders including personality disorders, schizophrenia, eating and mood disorders (Lyssenko et al., 2018; Şar, 2014; Sar et al., 2017). Symptoms relevant to TI may present clinically as conversion disorder (Del Río-Casanova et al., 2018; Sallin et al., 2016; Sar et al., 2009) or somatoform disorder (Kienle et al., 2017; Sar, 2011). Future research is needed to investigate the intersection between post-traumatic TI and these disorders.

Conclusion

STOP represents the first self-report measure of post-traumatic TI to demonstrate high-quality psychometric properties and utility in both clinical and community samples. Existing measures of TI reflect models that include fear, physical immobility, and dissociation. In addition to capturing these models, STOP is the first measure capable of capturing residual effects associated with post-traumatic TI. We expect that clinicians and researchers will find this preliminary scale useful for detecting and evaluating post-traumatic TI in trauma-exposed groups.

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Appendix A. Supplemental Information

STOP

Scale for Tonic immobility Occurring Post-trauma

SECTION A

Instructions:

People respond to very stressful or traumatic events in many different ways. During a very stressful or traumatic event, some individuals may feel “frozen” in their body (i.e. immobilized or unable to move). During this freezing response, individuals may experience changes in bodily sensations, thoughts, and emotions.

Sometimes, people continue to experience this freezing response after the traumatic event is over (for example, when reminded of the original traumatic event).

We invite you to complete this questions below regarding your own experiences with freezing. Take your time and try to be as honest as possible. There are no right or wrong answers and we appreciate your thoughtful effort.

1. Briefly describe the traumatic event(s) during which <u>you first felt frozen:</u>

Date of event(s):

_____

Briefly describe what happened and how you responded:

_____

_____

_____

_____

OR

☐ **I have never felt frozen in response to a traumatic event**
(please stop here and discontinue the questionnaire)

2. During the past month, have you felt frozen when reminded of past traumatic event(s)?

(please circle):

YES NO

IF YES, please answer questions 3-5 below.

IF NO, please stop here and discontinue the questionnaire.

3. What types of traumatic reminders have caused you to feel frozen over the past month (i.e. triggers)?

_____

_____

4. How many times did you feel frozen in response to traumatic reminders over the past month?

_____

_____

5. How long does it take for you to feel better (recover) after a post-traumatic freezing response?

_____

_____

SECTION B

STOP

Instructions: Only complete this section if it has been at least one month since the traumatic event(s) during which you first felt frozen. Carefully read each statement and consider whether you have experienced what is being described during the past month. For each statement, select the number that best describes the intensity of your experience during the past month.

Please use the following scale:

0 = NEVER 1 = A LITTLE 2 = MODERATELY 3 = QUITE A BIT 4 = EXTREMELY

1. When I freeze, I cannot move	0	1	2	3	4
2. When I freeze, I panic because I feel I cannot move	0	1	2	3	4
3. When I freeze, I panic because I don't know whether the freezing will stop	0	1	2	3	4
4. When I freeze, I take short quick breaths	0	1	2	3	4
5. When I freeze, I cannot speak even if I wanted to	0	1	2	3	4
6. When I freeze, I am still able to move my eyes	0	1	2	3	4
7. When I freeze, my vision becomes narrowed	0	1	2	3	4
8. When I freeze, I feel numb	0	1	2	3	4
9. When I freeze, my hearing becomes better than usual	0	1	2	3	4
10. When I freeze, I feel disconnected from parts of my body	0	1	2	3	4
11. When I freeze, I view my body from outside myself	0	1	2	3	4
12. When I freeze, I feel safe	0	1	2	3	4
13. When I freeze, I'm not sure whether it is the past or present	0	1	2	3	4
14. When I freeze, it feels like the experience lasts for a long time	0	1	2	3	4
15. When I freeze, I feel like I may disappear	0	1	2	3	4
16. When I freeze, I feel like I have no boundary around my body	0	1	2	3	4
17. When I freeze, my muscles go limp	0	1	2	3	4
18. When I freeze, I collapse to the floor	0	1	2	3	4
19. My body unfreezes piece by piece	0	1	2	3	4
20. After unfreezing, I feel a great sense of relief	0	1	2	3	4
21. After unfreezing, I find it difficult to recover	0	1	2	3	4
22. After unfreezing, I feel like my body is trembling/shaking	0	1	2	3	4

23. After unfreezing, my body feels heavy	0	1	2	3	4
24. After unfreezing, my muscles hurt	0	1	2	3	4
25. After unfreezing, I have difficulty remembering the experience	0	1	2	3	4
26. After unfreezing, I feel exhausted	0	1	2	3	4
27. After unfreezing, I feel shame	0	1	2	3	4
28. After unfreezing, I feel guilt	0	1	2	3	4
29. After unfreezing, I feel embarrassed	0	1	2	3	4
30. I wake up from sleep to find my body is 'frozen' even though I am mentally awake	0	1	2	3	4

Scoring the STOP:

The final STOP contains 30 items, each contributing to one of four subscale scores as well as a global post-traumatic tonic immobility response score. Mean subscale scores range from 0-4 and can be calculated by summing responses and dividing by the number of subscale items. By subscale, higher scores indicate a greater experience of fear as well as a greater number of tonic immobility symptoms (*Sensorimotor and Perceptual Alterations*); greater detachment from one's physical experience (*Somatic Detachment and Amnesia*); stronger after-effects following the episode (*Residual Symptoms);* and greater negative affect specific to guilt, shame and embarrassment (*Negative Affect*). A global *post-traumatic tonic immobility response* score ranging from 0-120 is obtained by summing all items, with higher global scores indicating greater severity of tonic immobility responses over the past one-month period.

Chapter 4: Mapping alexithymia: Level of emotional awareness differentiates emotion-specific somatosensory maps

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General Purpose

In this study, we explore emotion-specific bodily sensation patterns, or bodily sensation maps, in a group of trauma-exposed respondents with and without probable alexithymia. Alexithymia is a subclinical condition that occurs across clinical disorders including PTSD. Here, participants endorsing alexithymia reported a muted and undifferentiated pattern of emotion-related bodily sensation, as compared to non-alexithymia controls. Given the findings presented in Chapter 1, we posit whether such altered somatosensory patterns may underpin the cognitive-emotional difficulties observed in this alexithymia, and whether these findings may extend to other conditions involving altered emotional awareness and somatosensory processing such as MI.

Abstract

Background: Emotions have been associated with culturally universal and distinct bodily sensation "maps". Despite this knowledge, to date few studies have explored emotion-specific topography along clinically relevant dimensions, such as alexithymia.

Objective: We aimed to investigate emotion-specific topographies among individuals exposed to childhood maltreatment or neglect with absent (n = 51) or with probable (n = 46) alexithymia in adulthood, as defined by scores on the Toronto Alexithymia Scale (TAS-20).

Participants and Setting: Ninety-eight adult participants with exposure to childhood maltreatment or neglect were recruited to complete an online survey.

Methods: Using the well-validated emBODY tool (Nummenmaa et al., 2014), participants reported on their somatic experience of 17 emotions.

Results: Random effects analyses revealed topographically distinct bodily sensation t-maps that differentiated participants who endorsed probable alexithymia from those who did not (p-FDR < .05). Consistent with our a priori hypothesis, the probable alexithymia group reported a muted, diffuse and undifferentiated pattern of emotion-specific bodily sensation, whereas the non-alexithymia group reported a more distinct and localized pattern.

Conclusions: These results suggest that difficulty identifying and labeling emotions, as observed in alexithymia, may arise, in part, from an altered perception of somatic activation. It is well-established that childhood

maltreatment predict the development of alexithymia symptoms. The preliminary findings presented here expand our working understanding of the physical markers of childhood trauma, and may be used in practice to aid detection and monitor treatment outcome.

Introduction

Prevalent emotion-processing theories, including the somatic marker hypothesis (Damasio & Carvalho, 2013), suggest subjective feelings are triggered by the perception of emotion-related bodily states, reflecting changes in skeletomuscular, neuroendocrine, and autonomic nervous system activity (e.g., Barrett et al, 2007; Damasio & Carvalho, 2013). Adjustments in sympathetic and parasympathetic activity, for example, occur in response to both conscious and subconscious stimuli – an effect that becomes particularly salient within the context of threat processing (Ruth A Lanius et al., 2017). Following threat detection, a series of physiological defense responses help to prepare the body and mind for survival and motivate subsequent action (i.e., flight, fight, immobilize, and beyond; Bastos et al., 2016). Whether threatening or pleasurable, emotions are experienced in the body, facilitating our ability to identify, respond to, and interact with, our internal and external environment (Damasio & Carvalho, 2013).

Different emotions have been associated with discernible patterns of bodily sensations, suggesting some universality as to how emotions are organized and represented in the body. The emBODY tool is a well-validated topographical self-report method used to generate the bodily sensation "maps" associated with different emotions (Nummenmaa et al., 2014). Earlier work has reported distinct patterns of physical sensations for six basic (e.g., anger) and seven complex (e.g., contempt) emotions, along with a neutral feeling state. Critically, similar bodily sensation maps have been found across West European and East Asian samples,

suggesting the cross-cultural preservation of this topography (Nummenmaa et al., 2014). Indeed, these somatic representations emerge in early childhood (Bucci, 1997) and develop into discrete patterns over time (Hietanen et al., 2016), shaping, in part, the way children perceive, label, and interpret their emotions (Hietanen et al., 2016). Taken together, this work has identified a preliminary set of universal somatosensory maps that can be used for comparison to other groups, including clinical samples.

Despite evidence for universal somatosensory maps, bodily sensation patterns have yet to be investigated in populations likely to experience alterations in emotional experience, with the exception of one notable study exploring somatosensory maps in schizophrenia (Torregrossa et al., 2019). In this study, patients reported less discrete and less clear bodily sensations across emotions, consistent with illness-related deficits and alterations in social cognition and function (Billeke & Aboitiz, 2013).

Alterations in emotional experience are particularly relevant to alexithymia, a subclinical condition characterized by difficulty in identifying, interpreting, and describing emotions (Luminet et al., 2018; Taylor et al., 1997). In alexithymia, internal experience is believed to be minimized and attention focused externally, an effect that some authors have attributed to altered interoception (Breweret al. 2016; Murphy et al., 2018). Here, interoception refers to the somatosensory ability that allows one to feel what is occurring inside the

body and ultimately discriminate between different physiological and emotion-related bodily states (Khalsa et al., 2018).

Alexithymia affects approximately ten percent of the general population; it is somewhat more common among men and positively correlated with age (e.g., Franz et al., 2008; Mattila et al., 2007). The condition is linked consistently with exposure to adverse and traumatic experiences, with meta-analytic work confirming that alexithymia symptoms exist commonly among individuals with posttraumatic stress disorder (PTSD; Frewen et al., 2008). Alexithymia is further associated with the development and maintenance of psychopathology, including anxiety, depression, eating disorders, and PTSD (Taylor & Bagby, 2004; Treasure & Schmidt, 2013). Finally, individual studies point toward the transdiagnostic representation of alexithymia symptoms across a wide range of disorders including depression (Li et al., 2015), fibromyalgia (Calsius et al., 2015), somatoform and conversion disorders (Del Río-Casanova et al., 2018), substance use disorders (Hamidi et al., 2010), eating disorders (Westwood et al., 2017), autism spectrum disorders (Bird & Cook, 2013), and psychotic disorders (O'Driscoll et al., 2014). Interestingly, the development of alexithymia is associated closely with exposure to adverse experiences in childhood (Samur et al., 2013), a developmental period during which emotional processes, including early somatosensory maps, are thought to take shape (Hietanen et al., 2016). Accordingly, it appears probable that exposure to adverse childhood experiences might alter the formation of this common set of somatosensory maps over the

course of development, with alterations in this mapping observable into adulthood.

In addition to disturbances in the experience and expression of emotion, alexithymia is associated with cognitive difficulties in recognizing, describing, and distinguishing feelings from bodily sensations and with affective difficulties in emotion regulation (see Goerlich, 2018 for review). Together, these disturbances in cognitive and in emotional processing have been associated with a high burden of illness, including delayed treatment seeking, increased severity of illness, and increased health care utilization (Lumley & Norman, 1996). Moreover, alexithymia is increasingly associated with intimate partner violence, particularly among trauma-exposed military samples (Berke et al., 2017). Here, alterations in the cognitive and affective processing of incoming information (e.g., sensory) may interfere significantly with the ability to perceive, interpret, and appropriately respond to social stimuli and relational stressors (Taft et al., 2011). Given that alexithymia is further associated with suicidality (Davey et al., 2018), self-harm (H. Norman & Borrill, 2015), and reduced treatment response (Luminet et al., 2018), this subclinical condition represents a critical subject of inquiry.

In the current study, we sought to explore the topographical distribution of emotion-related bodily sensations in adult respondents exposed to early maltreatment or neglect with and without probable alexithymia (as captured by the well-validated Toronto Alexithymia Scale; Bagby et al., 1994). Our a priori

hypotheses were two-fold: i) groups with and without probable alexithymia would endorse significant differences in emotion-specific bodily sensation; and ii) these maps would reveal critical information about how these two groups differentially organize their emotional states? Whereas we expected the non-alexithymia group would report more distinct sensation patterns, we expected the probable alexithymia group to report less distinct or undifferentiated sensation patterns, consistent with established research.

Method

Participants

Participants were English-speaking North Americans who voluntarily responded to an online survey. Of the 565 participants who consented and initiated the survey, 153 participants required at least 45 minutes to complete the survey – this cut-off was pre-determined by the research team as the absolute minimum amount of time required to respond to the entire survey battery. Participants taking less than 45 minutes were therefore excluded from the sample ($n_{excluded} = 412$). Participants who did not complete any items on either of the two primary measures, the Toronto Alexithymia Scale (TAS-20) or the emBODY tool, were also excluded from analyses ($n_{excluded} = 18$). The emBODY tool did not display properly on certain mobile devices (i.e., smartphones and tablets), invalidating the bodily sensation maps for an additional 35 participants. Such cases were clearly identifiable by a distinct pattern of dots located sparsely across each body (these subjects could not drag the cursor thereby restricting the

colouring function). EmBODY output was visually inspected independently by two members of the research team, yielding identical decisions to exclude these 35 maps. Finally, two cases were excluded due to inaccessible (corrupted) data files. The final sample (N = 98) was calculated on the basis of the primary hypothesis and was deemed acceptable given a 95% CI and 10% Margin of Error.

Measures

EmBODY tool (electronic version). EmBODY is a unique computer-based, topographical self-report method used to generate emotion-specific bodily sensation maps (Nummenmaa et al., 2014). Previous work using the emBODY tool has indicated that emotion words, guided emotion imagery, emotional movies, and facial expressions are all equally effective in inducing the target emotion (Nummenmaa et al., 2014). Thus, in the current study, we elected to use only emotion words to evoke task ratings. EmBODY has been validated in both Finnish- and Swedish-speaking samples, with replicated bodily sensation maps emerging for all basic emotions (mean *r*s = 0.75 for between-samples matched emotions such as happiness; mean *r*s = 0.36 for between-samples mismatched emotions such as happiness vs anger). This tool has been further validated with East Asian (Taiwanese) participants, yielding similar results (mean *r*s = 0.70). Following methodology described in Nummenmaa et al. (2014), participants were asked to describe their somatic experience of 17 emotion words, including six basic emotions (fear, anger, disgust, sadness, happiness, surprise), ten complex emotions (anxiety, love, depression, contempt, pride, shame, jealousy, numb,

frozen, empty), and one neutral condition. The original 14 emotion words presented by Nummenmaa et al., (2014) were employed, substituting jealousy for envy, and adding three new emotion conditions: numb, frozen, and empty. These additional emotion words were explored in response to the extensive literature describing emotional states associated with trauma exposure (for review see Fonzo, 2018). For each emotion word (e.g., fear, anxiety, love), two blank body silhouettes were presented and the participant was asked to color in bodily regions wherein bodily sensation was felt to increase or "activate" in relation to the word on the first body, and areas felt to decrease or "deactivate" in relation to the word on the second body.

Toronto Alexithymia Scale (TAS-20). The Toronto Alexithymia Scale (TAS-20) is a 20-item scale used commonly to measure alexithymia (Bagby et al., 1994), a construct characterized by the inability to identify and describe one's emotional state. Items are rated using a 5-point Likert-type scale (1 = *Strongly Disagree* to 5 = S*trongly Agree*), with five items reverse scored (items 4, 5, 10, 18 and 19). The total TAS-20 score was the focus of our analysis and was yielded by summing items. The TAS-20 has demonstrated adequate internal consistency (α = .81) as well as test-retest reliability (.77, $p < .01$; Bagby et al., 1994). Research using the TAS-20 commonly employs the following cut-off scores: total scores $\leq$ 51 indicate non-alexithymia, scores falling between 52 to 60 indicate possible alexithymia, and scores $\geq$ 61 indicate the presence of alexithymia. TAS item #19 was found to be improperly displayed in the survey battery, leading to a high

nonresponse (5.2%) or neutral response rate for this item (i.e. 16.3% rated as "neither agree nor disagree"). Thus, item #19 was eliminated, and the range and cut-offs were adjusted to reflect this modification (adj. cut-offs: total score ≤ 46 indicate the absence of alexithymia, scores between 47-55 indicate possible alexithymia, and scores ≥ 56 indicate the presence of alexithymia). In light of the adjustment made to participant scores, we elected to use the TAS-20 total score to differentiate between two groups: a *non-alexithymia* group (TAS-20 total score ≤ 46) and a *probable alexithymia* group that represented participant scores consistent with possible to present alexithymia (TAS-20 total score > 46). Similar practices have been adopted by other users of this scale (e.g., Starita & di Pellegrino, 2018), and remain consistent with criticisms that the original cut-off for alexithymia (TAS ≥ 61) is overly conservative (Franz et al., 2008).

The Childhood Trauma Questionnaire (CTQ-28). The CTQ-28, developed by Bernstein et al. (1994), is a 28-item self-report measure that evaluates the frequency, severity, and duration of a wide range of interpersonal traumatic events experienced in childhood (up to 18 years). The CTQ-28 measures five domains of childhood maltreatment and neglect (emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect). Items are rated using a 5-point Likert-type scale (1 = *Never True* to 5 = *Very Often True*). The sum of the five items for each subscale ranges from 5 to 25, and a total summed score ranges from 25-125. The CTQ-28 has demonstrated strong psychometric properties, including high internal consistency (α = 0.79 to 0.94;

Bernstein et al., 1994), and is an internationally established tool for the retrospective assessment of child maltreatment and neglect. In this study, the CTQ-28 was used to confirm exposure to childhood maltreatment or neglect, collectively referred to here as *adverse childhood experience*.

Procedure

This study was approved by the New School IRB. North American participants (as per IP address), aged 18-58, who were fluent and literate in English were eligible for recruitment. Potential participants viewed the following online advertisement for the study: *"Psychology study seeks paid volunteers aged 18-58. In order to participate, you must be fluent in English and comfortable with reading words on a screen. The study involves answering questions about yourself regarding past experiences, psychological well-being, and mood. The study also involves two short tasks, which involve looking at pictures or reading words and coloring. The study will take approximately 45 minutes to an hour to participate, and pays $1."* The advertisement and survey battery was administered using Amazon's Mechanical Turk (MTurk), a web-based platform recognized as a valid recruitment strategy for mental health research (Chandler & Shapiro, 2016). Data collection occurred on a secure, encrypted website (Qualtrics) independent of crowd-sourcing access sites to preserve participant anonymity and confidentiality. Participants who self-identified as eligible for the study were subsequently presented with a letter of information and consented to participation by checking a box prior to completing survey material. Consenting participants completed a

demographic survey, followed by the electronic emBODY tool and a series of clinical self-report measures. The location of items and response scales were varied to ensure participants were attending to item content and to confirm response quality. Compensation rates were comparable to similar studies at the time of administration. Supporting data and materials are available upon request from the corresponding author.

Statistical Analysis

Demographics and psychological measures. Groups were compared across demographic and clinical characteristics. A between-group independent samples *t*-test was conducted to assess differences in TAS-20 total score. A significant non-normality effect was detected by the Shapiro-Wilks test for age and CTQ scores; therefore, nonparametric Mann-Whitney U tests were performed to account for non-normality, and in some cases, unequal variances. Chi-square tests were conducted to assess between-group differences in gender, ethnicity, and education.

EmBODY tool. Individual data files representing raw intensity value by voxel were preprocessed according to the procedure described in Nummenmaa et al. (2014). Total body space is represented by 50,364 data points (pixels). The painting tool itself has a diameter of 12 pixels and paint intensity ranged from (no paint) 0 to 100 (fully saturated), depending on click duration.

Subject-wise activation and deactivation data were stored as integers, with finished images stored in matrices. Mass univariate t-tests were used to compare

pixel wise activations and deactivations, of the bodily sensation maps for each emotion against zero. False Discovery Rate (FDR) correction with an alpha level of 0.05 was applied to *t*-maps to control for false positives due to multiple comparisons. Activation and deactivation maps were first explored separately, and subsequently combined into a single body space to explore subject-wise unique sensation maps. The latter *combined* sensation maps were generated by subtracting absolute deactivation values from activation values before running t-tests.

Finally, between-group comparisons were explored. To determine the extent to which sensations covered the total body space, activation, as well as deactivation pixels, were counted and normalized by the total body space. A group mean was then calculated for each voxel in the body. Activation and deactivation values were separately considered in reference to total body space, yielding a ratio score (%) representing how much of the total body space was colored in response to that emotion. Group medians were calculated and compared using nonparametric tests (Kruskal-Wallis H). Statistical procedures were conducted using SPSS software version 24 and MATLAB version 2017b.

Results

Demographics and psychological measures

The proportions of participants based on sex, education, and ethnicity did not differ significantly between groups. Mean age differed significantly between groups ($U = 918.50$, $p = .046$), with slightly older participants in the non-

alexithymia group. CTQ scores did not differ significantly between groups, with the exception of scores on the sexual abuse subscale (U = 888.50, p = .011). Participants in the probable alexithymia group reported higher rates of childhood sexual abuse than those in the non-alexithymia group (Table 1).

Missing values were minimal, representing less than 5% of TAS-20 and less than 3% of CTQ-28 responses (Enders, 2001). A Missing Values Analysis indicated that Little's (1988) test of Missing Completely at Random (MCAR) was not significant for TAS-20 (χ^2 (208) = 233.65, p = .107) nor CTQ-28 (χ^2 (127) = 151.47, p = .068). Missing values were therefore imputed using an Expectation Maximization (EM) method for both measures (Enders, 2011). Items were imputed by subscale to account for the increased correlations observed between items belonging to the same scale.

Table 1. Demographic and clinical characteristics

Characteristic	Total Sample (N = 98) *M* ± SD	Non-alexithymia (*n* = 51) *M* ± SD	Probable Alexithymia (*n* = 47) *M* ± SD	Test Statistic
TAS-20 Total Score	46.63 ± 12.81	35.98 ± 6.14	58.19 ± 6.55	t (96) = -17.33, p = .000*
Age	38.38 ± 10.28	40.45 ± 11.26	36.13 ± 8.67	U = 918.50, p = .046*
Sex (% F)	60.2	56.9	63.8	X^2 (2, N = 98) = 1.3, p = .528
CTQ EA	11.86 ± 5.96	11.29 ± 6.27	12.48 ± 5.60	U = 985.50, p = .128
CTQ PA	8.55 ± 4.37	8.43 ± 4.94	8.69 ± 3.70	U = 1029.50, p = .219
CTQ SA	8.32 ± 5.58	6.90 ± 4.26	9.85 ± 6.43	U = 888.50, p = .011*
CTQ EN	12.66 ± 5.22	12.51 ± 5.83	12.83 ± 4.51	U = 1126.50, p = .608
CTQ PN	8.45 ± 3.86	8.09 ± 3.83	8.83 ± 3.90	U = 1041.50, p = .255
CTQ Total	49.84 ± 20.05	47.23 ± 20.98	52.68 ± 18.80	U = 923.00, p = .050*
Education (%)				X^2 (6, N = 96) = 6.4, p = .380
High School	10.2	7.8	12.8	
Post-Secondary	69.4	74.5	63.8	
Graduate School	18.4	13.7	23.4	
Prefer not to say	2.0	4.0	-	
Ethnicity (%)				X^2 (7, N = 98) = 4.8, p = .685
Caucasian	72.4	74.5	70.2	
Asian	8.2	7.8	8.5	
Hispanic	5.1	5.9	4.3	
African American	10.2	7.8	12.8	
Black non-American	1.0	2.0	-	
Indigenous	2.0	2.0	2.1	
Mixed	1.0	-	2.1	

*p < 0.05. Where a significant Shapiro-Wilks test was detected (*), nonparametric Mann-Whitney U tests were conducted. Independent Samples t-tests and Chi Square tests were conducted where appropriate. TAS-20: Toronto Alexithymia Scale, CTQ: Childhood Trauma Questionnaire (EA = Emotional Abuse, PA = Physical Abuse, SA = Sexual Abuse, EN = Emotional Neglect, PN = Physical Neglect).

Within-Group Analysis

Activation bodily sensation maps. Within the non-alexithymia group (TAS-20 total score ≤ 46), bodily sensation maps revealed a distinct pattern across emotions (Fig. 1 - Upper). Here, activating sensations are shown in red and yellow, with yellow regions representing the strongest sensations (as shown by the colour bar). Within the non-alexithymia group, *anger*, *happiness*, *love*, *pride* showed stronger patterns of activation, as indicated by a greater range of positive t-values (red-yellow). A number of activation maps emerged comparatively blank, suggesting relatively low experienced activation for the *neutral, sadness, depression*, *numb*, *frozen*, and *empty* conditions. Other emotions, including *surprise*, *jealousy*, *fear*, *anxiety*, *disgust*, *contempt,* and *shame,* were reflected by localized patterns of moderate activation centred primarily in the head, chest, and abdomen regions.

Within the probable alexithymia group (TAS-20 total score > 46), bodily intensity maps revealed less variation in intensity of activation sensations across emotions (Fig. 1 - Lower); these maps also appeared less distinct in activation patterns across emotions. For example, similar bodily sensation maps emerged for *fear*, *anger*, *happiness*, *surprise*, *anxiety*, *pride,* and *love*. Another similar pattern emerged for *disgust, sadness, depression, contempt, shame, jealousy,* and *frozen* Activation maps for the *neutral*, *numb* and *empty* conditions emerged relatively blank. Taken together, individuals in the probable alexithymia group displayed a muted and less distinct pattern of perceived activation across emotions.

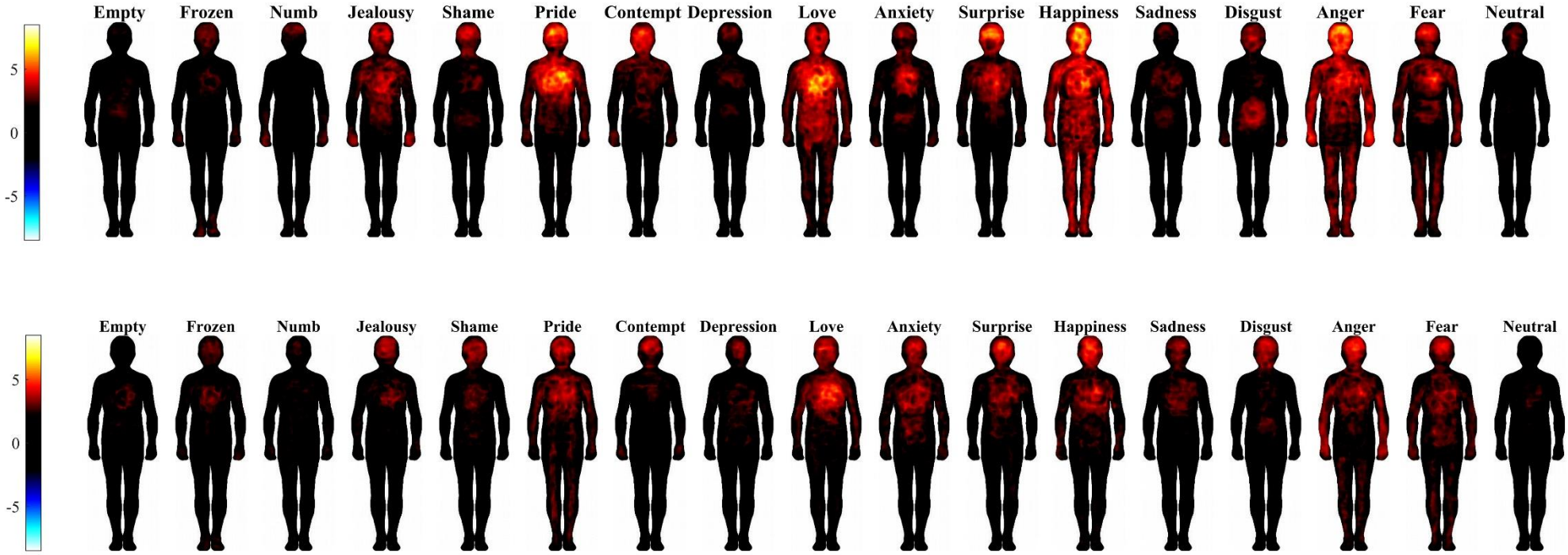


Figure 1. Activation sensation bodily topography reported by non-alexithymia (Upper; $p < 0.05$ FDR corrected; $t > 2.1$) and probable alexithymia (Lower*;* $p < 0.05$ FDR corrected; $t > 2.3$) groups. The color bar indicates the *t*-statistic range.

Deactivation bodily sensation maps. Within the non-alexithymia group, negative t-values were observed in approximately half of the emotions, suggesting some emotions involve deactivation sensations and some do not (Fig. 2 - Upper). Emotions characterized by greater deactivation included *fear*, *sadness*, *anxiety*, *depression*, *shame*, *numb*, *frozen*, and *empty*. The most intense deactivating sensations were represented in *sadness*, *depression*, *numb*, *frozen*, and *empty*. Emotions described as having strong activation patterns (i.e., *anger*, *happiness*, *surprise*, *love*, *contempt*, and *pride*) showed almost no FDR-corrected deactivation sensations. By contrast, deactivation sensation maps within the probable alexithymia group displayed less intensity and distinction across emotions (Fig. 2 - Lower). In both groups, *numb*, *frozen*, and *empty* were associated with sensation decrease rather than increase representing a draining away or flattening of affect.

Consistent with the study hypotheses, within the non-alexithymia group, activation and deactivation maps displayed greater variation in pattern and intensity across each of the 17 emotion conditions. Within the probable alexithymia group, however, activation and deactivation maps revealed a muted pattern of intensity across the 17 emotions, often showing one or two different patterns of sensation, making emotions less distinguishable from one another.

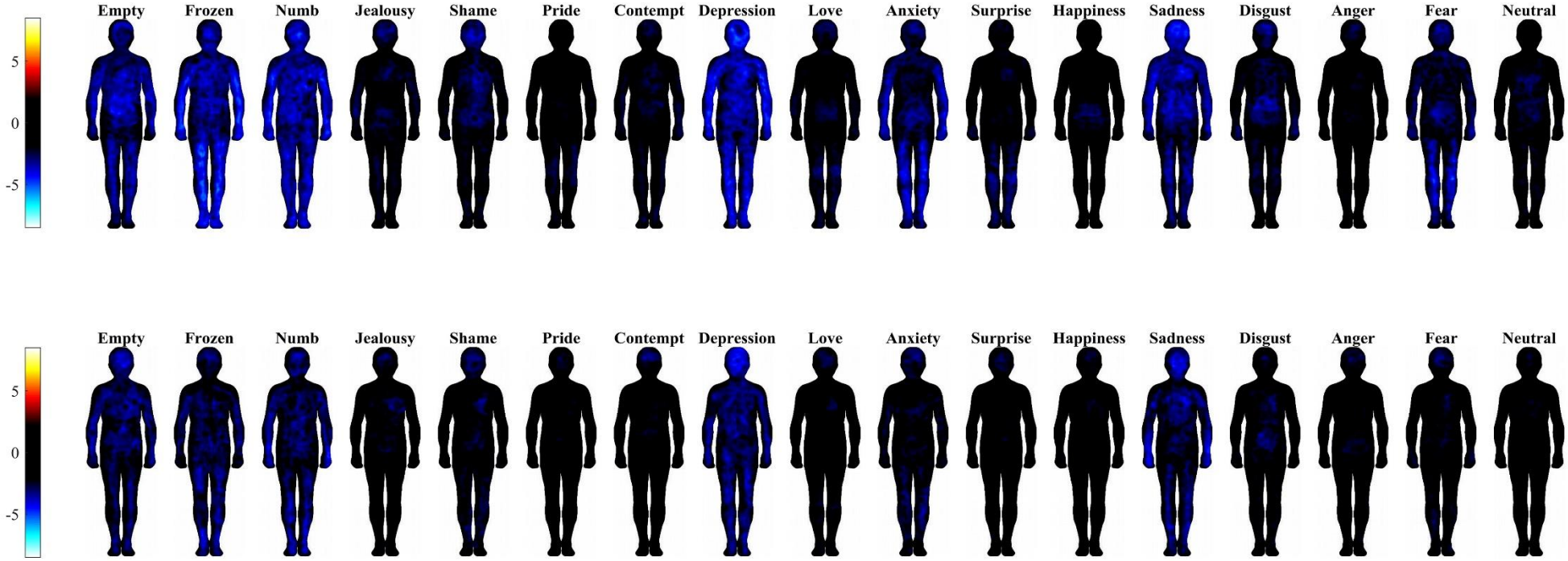


Figure 2. Deactivation bodily sensation topographyreported by non-alexithymia (Upper; $p < 0.05$ FDR corrected; $t > 2.0$) and probable alexithymia (Lower; $p < 0.05$ FDR corrected; $t > 2.2$) groups. The color bar indicates the *t*-statistic range.

Combined bodily sensation maps. Combined bodily sensation maps (i.e., activation maps minus deactivation maps) were explored. Within the non-alexithymia group, a localized and subtle pattern of sensation emerged for several emotions (Fig. 3 - Upper). For example, whereas *depression* was associated with greater deactivation in the arms and legs, *anger* was associated with greater activation in the head, hands, and, to a lesser extent, chest and arms. L*ove* was associated with greater activation in the chest and pelvis, *pride* was associated with greater activation in the chest and head, and *happiness* was associated with whole body activation. The combined map for probable alexithymia did not survive correction, indicating that participants reported widespread body coverage for both activation and deactivation sensations across emotions (Fig. 3 - Lower). The uncorrected map itself reveals this diffuse and generalized pattern of somatic activation and deactivation across all 17 emotions, highlighting the comparative lack of selective protuberance among emotion-specific bodily sensations for those in the probable alexithymia group.

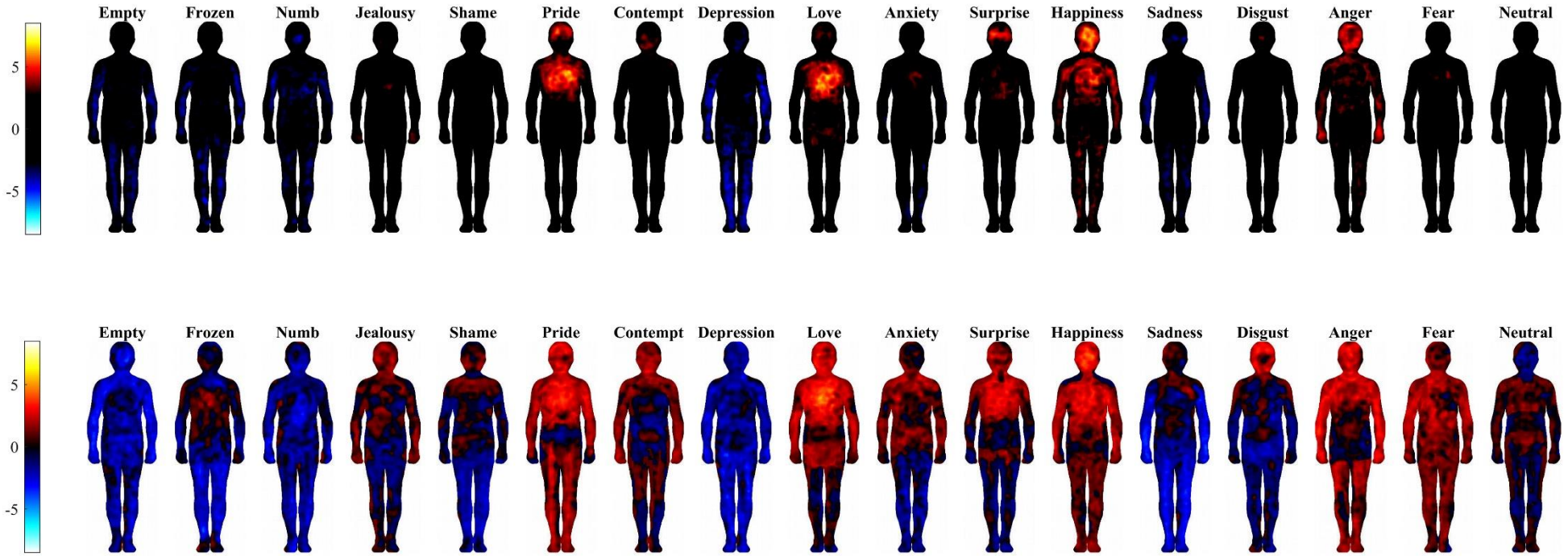


Figure 3. Combined bodily sensation topography reported by non-alexithymia (Upper; $p < 0.05$ FDR corrected; $t > 2.7$) and probable alexithymia (Lower; $p = 0.003$ uncorrected) groups. The color bar indicates the *t*-statistic range.

Between-Group Analysis

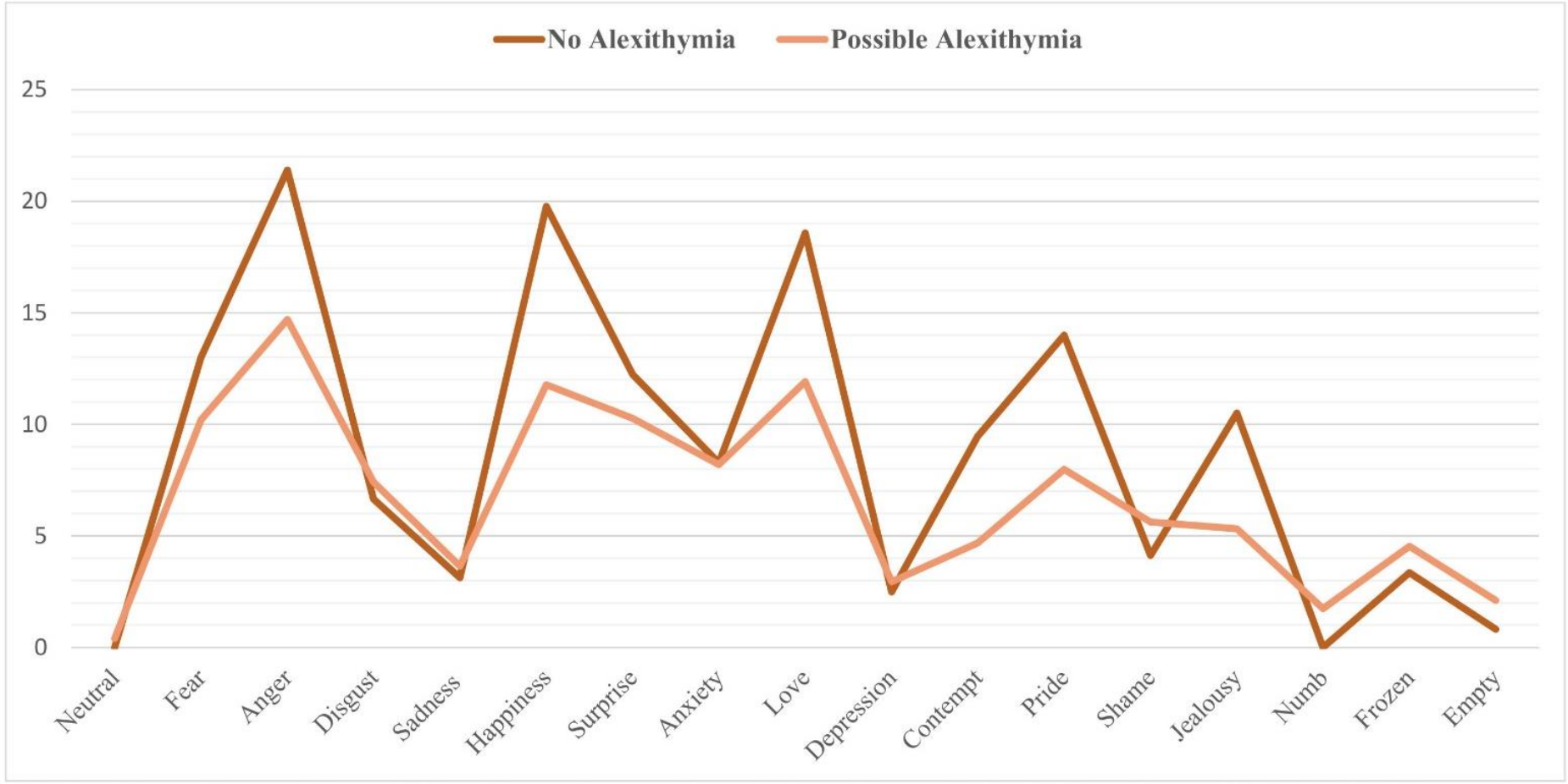
Group median scores were calculated for whole-body activation and deactivation ratings and presented as an estimation of that emotion distributions' central tendency (Fig. 4 & 5). A Kruskal-Wallis H test showed that there was a statistically significant difference in the amount of body space coloured in reference to activation sensations between groups for *happiness*, $\chi^2(1) = 7.8$, $p = 0.005$ and *jealousy*, $\chi^2(1) = 4.4$, $p = 0.04$, suggesting these group medians were unequal. In reference to deactivation sensations, a statistically significant difference emerged in the amount of body space coloured for *fear*, $\chi^2(1) = 10.1$, $p = 0.002$. Mean rank scores are displayed in Table 2. For all three emotions, the probable alexithymia group coloured less of the total available body space than the non-alexithymia group.

Table 2. Mean rank total body coverage scores

	Mean Rank	
Activation Sensations	Non-alexithymia	Probable alexithymia
Happiness	57.2	41.2
Jealousy	55.3	43.2
Deactivation Sensations		
Fear	58.2	40.0

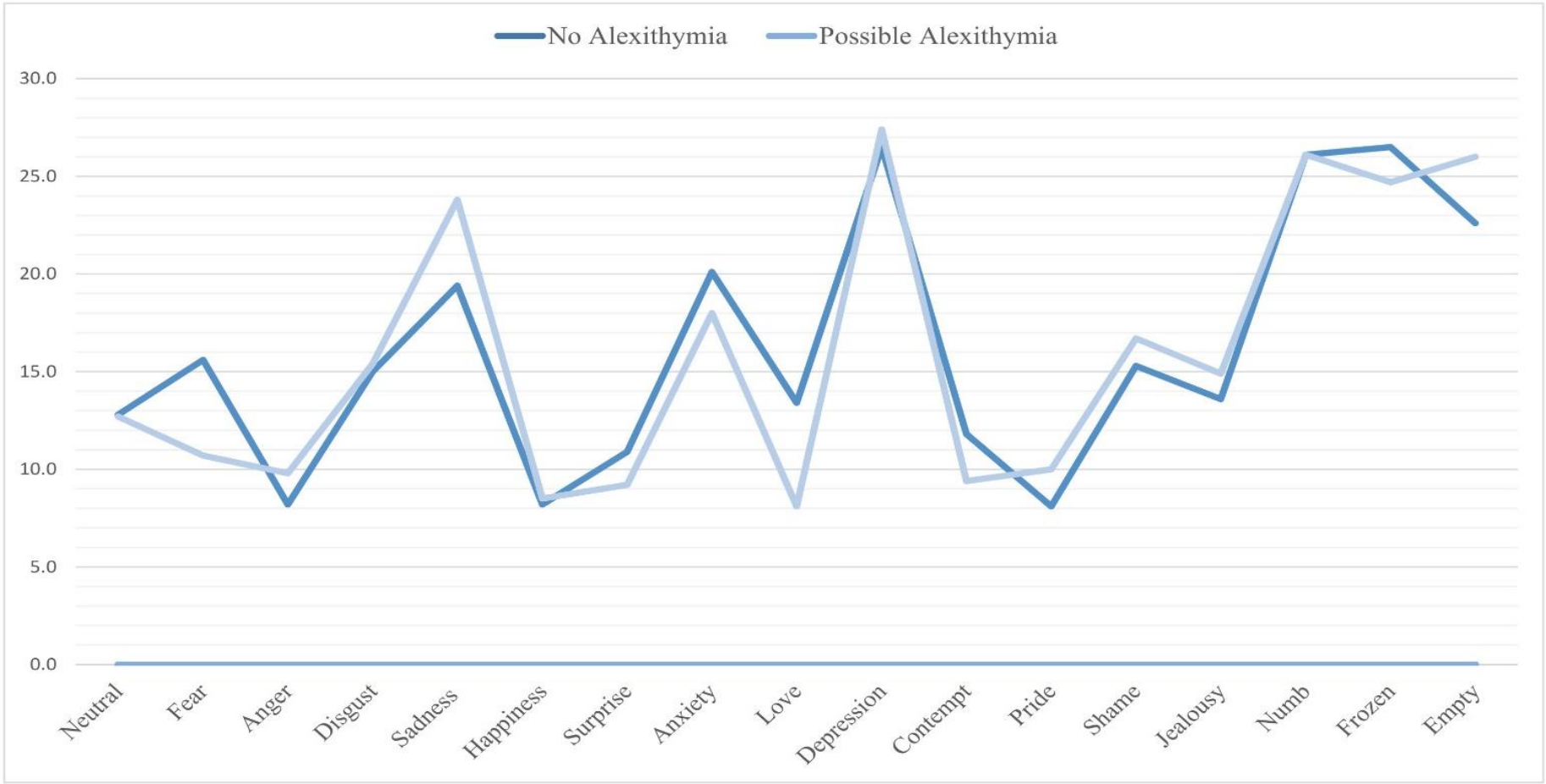
*$p < 0.05$

Overall, the probable alexithymia group reported a comparatively blunted degree of sensation; however, statistically significant median differences were noted for only 3 of 17 emotions tested. Group differences were not found in the majority of conditions, suggesting it is not the amount of sensation, but rather its location and intensity that may differ between groups.



M (SD)	Neutr	Fear	Anger	Disg	Sad	Happ	Surpr	Anx	Love	Dep	Cont	Pride	Sham	Jeal	Numb	Froze	Empt
Non-Alex	0 (14.2)	13.0 (18.2)	21.4 (21.6)	6.6 (11.3)	3.1 (8.2)	19.8 (26.0)	12.2 (12.8)	8.3 (11.4)	18.6 (22.7)	2.5 (7.9)	9.5 (11.3)	14 (18.9)	4.1 (13.1)	10.5 (15.5)	0 (12.9)	3.3 (11.0)	0.8 (5.6)
Prob Alex	0.4 (9.3)	10.2 (21.5)	14.7 (21.0)	7.4 (8.5)	3.6 (13.2)	11.8 (18.8)	10.3 (15.4)	8.2 (19.5)	11.9 (21.2)	2.9 (15.3)	4.7 (11.7)	8.0 (22.9)	5.6 (8.7)	5.3 (13.5)	1.7 (18.4)	4.5 (12.0)	2.1 (10.3)

Figure 4. Medians and standard deviations for percentage of body space coloured in activation sensations (%) by group



M (SD)	Neutr	Fear	Anger	Disg	Sad	Happ	Surpr	Anx	Love	Dep	Cont	Pride	Sham	Jeal	Numb	Froze	Empt
Non-Alex	0 (12.8)	14.6 (15.6)	2.6 (8.2)	6.8 (15)	20.3 (19.4)	0.7 (8.2)	6.1 (10.9)	17.7 (20.1)	4.2 (13.4)	28.2 (26.5)	6.1 (11.8)	0.9 (8.1)	9.4 (15.3)	4.7 (13.6)	24.4 (26.1)	21.1 (26.5)	15.3 (22.6)
Prob Alex	0.4 (12.7)	4.2 (10.7)	4.4 (9.8)	7.3 15.4)	13.1 (23.8)	1.7 (8.5)	2.9 (9.2)	8.2 (18)	3.9 (8.1)	17.3 (27.4)	3.3 (9.4)	1.9 (10.0)	8 (16.7)	4.2 (14.9)	9.2 (26.1)	11.8 (24.7)	11.8 (26)

Figure 5. Medians and standard deviations for percentage of body space coloured in deactivation sensations (%) by group

Discussion

Existing evidence suggests that the bodily changes associated with specific emotions are distinct and culturally universal (Nummenmaa et al., 2014; Nummenmaa et al., 2018). To our knowledge, this is the first study to reveal that alexithymia may alter the expected topography of these somatic maps. Specifically, respondents without alexithymia reported bodily sensation maps that were largely consistent with, or trending toward, topographies presented in earlier work (Fig. 1-3 - Upper; Nummenmaa et al., 2014, 2018). Conversely, respondents endorsing probable alexithymia reported wide-spread activation and deactivation sensations throughout the body that were lacking in intensity (Fig. 1 & 2 - Lower) and in specificity (Fig. 3 - Lower). The latter pattern was consistent with the undifferentiated bodily maps of individuals with schizophrenia (Torregrossa et al., 2019), and may point towards the underlying role of alexithymia in the bodily representation of emotion in this population.

A few significant differences also emerged between groups with respect to how several emotions were represented in the body (Table 2). As compared to the non-alexithymia group, the probable alexithymia group reported significantly less activating sensations for *happiness* and *jealousy* and significantly less deactivating sensations in reference to *fear*. Taken together, these findings point towards a potentially muted or blunted pattern of sensation experience among the probable alexithymia group, a pattern that may associate significantly with alexithymia.

Experiencing a localized rather than a diffused pattern of bodily activation and deactivation may facilitate the ability to discriminate between emotions more readily, as would be expected in the non-alexithymia group. Interestingly, the diffused distribution of sensations that emerged in the probable alexithymia group is in keeping with the well-documented challenges with localizing, identifying, and expressing feelings in the body typically observed in alexithymia (Luminet et al., 2018). Bodily sensations were not localized, suggesting an indistinct, and possibly confusing, pattern of sensory experience (Fig. 3 - Lower) that contrasted sharply with the emotion-specific sensations described by those without alexithymia (Fig. 3 - Upper). It follows that for individuals with difficulty distinguishing emotion-specific sensations (e.g., due to lack of specificity), difficulties in identifying, interpreting and expressing emotion appropriately may emerge, as observed in alexithymia.

The lack of somatosensory specificity observed here is consistent with the notion of altered interoception in alexithymia. Children and adults who struggle with interoceptive awareness are likely to have difficulty identifying when they feel hungry or thirsty, or, hot or cold. Consequently, interoceptive impairment has the potential to severely alter emotional experience, expression, and regulation (Khalsa et al., 2018). When interoception is affected, the subjective experience of emotions, and the sensations proposed to underpin them, may also be altered significantly.

Our findings are consistent with research associating alexithymia with reduced sensory sensitivity (Serafini et al., 2016) and are in line with the results of fMRI connectivity studies in PTSD, a condition often involving alexithymia. For example, as compared with controls, individuals with PTSD (at rest) show widespread activation among brain regions responsible for salience processing (e.g., hyperactivity in the ventral anterior cingulate cortex and the parahippocampus/amygdala) and threat detection (locus coeruleus, superior colliculus, amygdala, and prefrontal cortex), that is accompanied by a weak pattern of connectivity with regions responsible for emotion regulation, interoception and self-referential processing (i.e. hypoactivity in the posterior insula, cerebellar pyramis and middle frontal gyrus; Koch et al., 2016; Lanius et al., 2016). This pattern is thought to reflect increased threat detection and hypervigilance, at the cost of internal awareness, emotion regulation and self-referential processing (Koch et al., 2016)

Altered interoception, however, is not the only relevant factor in understanding the findings presented here. The patterns observed in sensation maps could also reflect alterations in multiple processes, including: i) alterations in physiological circuitry; ii) alterations interpreting universal sensations; and/or iii) alterations in the communication of emotion, including the inability to describe and/or translate bodily sensations in the emBODY task itself.

An interesting association exists between alexithymia symptoms and dissociative responses. A moderate and positive correlation has been shown, for

example, between scores on the TAS–20 and scores on the Dissociative Experiences Scale in a PTSD (r =.4, p < .001; Frewen et al., 2008). Given that childhood trauma and maltreatment has been related to dissociative responses (e.g., Lanius et al., 2011), and given the established link between early trauma and alexithymia symptoms, we speculate that dissociation may unite these findings under a common framework. Traditionally, emotions have been viewed as motivating adaptive behaviour; however, emotions become futile in chronically traumatizing situations, like repeated childhood abuse, wherein is not possible or adaptive to translate the emotion into an active defense response (i.e. flight or fight). Here, the victim must instead disconnect to survive (Lanius et al., 2011; Frewen et al., 2008), suggesting that alexithymia symptoms may represent or involve underlying dissociative processes (i.e. the opioid-mediated stress response; Lanius et al., 2018).

Although preliminary, these results have important clinical implications. Deficits in emotion-related bodily sensations are likely to contribute to poor social functioning and represent an important target for psychosocial interventions. The assessment of alexithymia, as well as of emotional awareness more generally, may also be enhanced through administration of the emBODY task. The present findings confirm that this task allows for pictorial expression of emotion-specific sensation, extending commonly used self-report measures like the TAS-20. Similarly, this task may also be used to capture autobiographical memories of body sensations experienced during traumatic and non-traumatic events that are

not easily expressed verbally. In addition, these data contribute to the validation of the TAS-20 as a measure of alexithymia, by corroborating high and low scores on the TAS-20 with corresponding physiological markers (i.e. patterns of bodily sensations that appear to be consistent with the presence and absence of alexithymia). Finally, the current findings suggest mindfulness-based interventions may be experienced differently, depending on an individual's level of emotional awareness. For example, building mindful, conscious awareness of somatosensory patterning in those with probable alexithymia may be uncomfortable, specifically, if increasing awareness of widespread body activation or deactivation like numbness (Frewen et al., 2020).

Limitations

This study utilized data from an online convenience sample, introducing a possible volunteer bias that may have impacted results. These data are part of a larger cross-sectional self-report survey study with a retrospective design, which is limited by predisposition to recall and response bias. Limitations to the TAS-20 have also been noted and may have influenced the results presented here (e.g., TAS-20 has been criticized for measuring general negative affect rather than alexithymia). Further, as we did not control for other conditions associated with alexithymia symptoms in this study, these results should be interpreted with caution. Well-controlled research is needed to determine the degree to which the patterns observed here are specific and contributable to alexithymia symptoms.

Future Directions

While the TAS-20 cut-off employed here is consistent with an interpretation of "possible" to "present" alexithymia, future work is needed to confirm these findings in a larger sample diagnosed specifically with alexithymia. Replication is also needed for these results to be considered reliable and generalizable, a pattern that would further confirm that the bodily sensation maps presented here are attributable to alexithymia. This work may include examination of state and trait levels of emotional awareness, as well as the capture of present-moment bodily states induced through provocation methods. Given the present findings, future research might also explore the mediating role of interoception in relation to emotional awareness and somatosensory topography. . Longitudinal research is required to illuminate how early childhood maltreatment and neglect impact the development of bodily sensation maps. Future research should investigate these patterns in children with and without a history of maltreatment and neglect. Given the significant difference that emerged in our sample between the non-alexithymia and alexithymia groups for childhood sexual abuse, future research should explore the unique contributions of particular forms of trauma on alexithymia symptoms. Finally, investigation into other relevant clinical populations is warranted. Trauma-related dissociation, for example, should be explored, as dissociation relies on detachment from bodily sensations. Future research should investigate the intersection between alexithymia and dissociative symptom sets, for example, exploring dissociation as a potential mediator between childhood trauma exposure and the onset of alexithymia symptoms.

Similarly, future research is need to investigate the bodily sensation maps associated with various dimensions of dissociation (e.g., depersonalization, derealisation, somatic dissociation, etc.).

Conclusion

Our findings suggest that alexithymia symptoms are associated with altered sensory-emotional representations in the body and, provided this pattern is stable and replicable, these data may be used to inform policies that guide the assessment and treatment of complex posttraumatic stress disorder (PTSD) in both child and adult survivors of abuse. Alexithymia has long been associated with poor treatment response and these symptoms are not routinely assessed or targeted in existing PTSD treatments. Embody represents a non-verbal emotional expression tool suitable for use with children as well as those who struggle with verbally expressing certain somatic processes and experiences (i.e. a current limitation of talk therapies).

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Chapter 5: Conclusions

MI is an important predictor of psychopathology including PTSD, depression, and suicide (Griffin et al., 2019). PTSD is the main psychiatric consequence resulting from exposure to traumatic events, highlighting the potentially injurious nature of both fear-based and morality-based trauma.

MI and associated symptoms (e.g., shame, social alienation and disconnection resulting from ruptured bonds, broken trust, loss of meaning, existential crisis) appear to represent aspects of PTSD that are not entirely addressed by current first-line treatment modalities (Bradley et al., 2005; Owens et al., 2008), and further, may show resistance to such treatments (Steenkamp et al., 2015). Moreover, some authors have argued that PE and CPT are not indicated for certain MI-exposed patient groups, for example, when distorted cognitions are absent and descriptions about the MI event are factually accurate (Farnsworth, 2019). Here, alternative interventions, including third wave CBTs, may be better suited, although they do not consider altered somatosensory processes described in this work.

The biological basis of MI remains relatively unexplored, limiting clinical conceptualization and treatment. Thus, the aim of this thesis was to expand upon this knowledge area. First, we investigated neural correlates of MI event recall in military members and PSP with PTSD, relative to civilian MI-exposed controls, and examined the neural activation patterns associated with hallmark MI symptoms in PTSD, namely, shame and alterations to cognition and emotion. We

further explored two MI-relevant phenomena on the biological level: posttraumatic TI and emotion-specific sensory patterning in the body.

The central hypothesis that we tested was that MI event recall would differ significantly in MI-exposed individuals with PTSD, as compared to MI-exposed controls. Consistent with PTSD-related alterations, we expected to see a pattern of heightened sensory processing and altered prefrontal activity in regions (dm|PFC, dlPFC) that also critically contribute towards moral cognition, moral emotion, and autobiographical memory processing. Moreover, we examined the bodily sensation patterns, or "maps", associated with different emotions (denoting how emotions are experienced in the body) in trauma-exposed participants with and without symptoms of alexithymia. Finally, we tested the factor structure of a novel self-report measure of posttraumatic TI, and conceptually explored posttraumatic TI within a dissociative framework. Here, both alexithymia and posttraumatic TI are hypothesized to be potential symptomatic consequences of MI event exposure.

5.1 Summary of Findings

MI event recall is a common component of front-line therapeutic interventions. In the first study, described in Chapter 2 (Lloyd et al., unpublished), we investigated neural activation patterns associated with MI event recall in MI-exposed military members and PSP with PTSD, relative to civilian MI-exposed controls. Consistent with predictions, we found that individuals with PTSD showed significantly heightened neural activity in regions responsible for salience

processing (dACC), particularly with respect to viscerosensory information (right posterior insula), as well as areas associated with defensive responding (left postcentral gyrus), and top-down emotion regulation (left dlPFC). Further, MI-related state shame showed an inverse relation with neural activity in the right SFG (within the dmPFC), a brain region largely responsible for processing and regulating negative self-blaming emotion and processing autobiographical memory. This finding is consistent with previous work showing heavy sensory processing inhibits self-related processing (via mPFC hypoactivity; Frewen et al., 2020; Goldberg et al., 2006) and associating this pattern with chronic defensive states (mediated by the left postcentral gyrus in concert with the larger innate alarm system; Lanius et al., 2016; Steuwe et al., 2015). Additionally, more severe negative alterations in cognition and mood were instead associated with left posterior insula hypoactivity, reflective of a pattern of viscerosensory dampening. We interpret this hyper- and hypoactivity in the posterior insula to reflect, respectively, heightened and dampened experience of unpleasant bodily sensations related to blame-based emotion.

Taken together, these neuroimaging findings are broadly in line with previous studies showing a biphasic pattern of emotional under- and over-modulation in PTSD, as mediated by insula over- and underactivity, respectively (Frewen & Lanius, 2006; Hopper et al., 2007; Lanius et al., 2010), where emotional undermodulation is associated with symptoms of hyperarousal, and emotional overmodulation is associated with emotional detachment and

symptoms of hypoarousal. In study one, MI event recall and acute shame responses were both associated with neural patterns suggestive of viscerosensory undermodulation (e.g., posterior insula hyperactivity and SFG hypoactivity, respectively). Conversely, more severe negative alterations in cognition and mood were associated with a pattern of overmodulated viscerosensory activity (e.g., posterior insula hypoactivity). We hypothesize that this heightened viscerosensory information, in turn, prompts recruitment of regulatory cognitive controls (e.g., dlPFC) to dampen and diminish distressing bodily sensations (Zinchenko & Arsalidou, 2018).

Interestingly, more severe alterations were associated with posterior insula hypoactivity, suggesting diminished viscerosensory experience. Alterations in cognition and mood (i.e. difficulties with traumatic memory recall, social disconnection, dampened positive emotion, as well as descriptive and prescriptive cognitions that reflect potential changes in identity and perceptions of the goodness of the world) reflect more persistent negative beliefs and functional changes that can be provoked by MI event exposure. This finding is consistent with previous research that links emotional overmodulation (insula hypoactivity) with profound detachment from emotional states, as well as a lack of interoceptive awareness indicated by dissociative symptoms (Lanius et al., 2010), emotional numbing (Krystal & Krystal, 1988; Van der Kolk & McFarlane, 1996), and alexithymia (Badura, 2003; Paul A. Frewen et al., 2008). This dysregulated visceral activity - whether intensified or numbed - provides little space for

positive moral emotion. As SN regions (e.g., anterior insula, anterior cingulate cortex, amygdale, thalamus, ventral striatum) are thought to critically enable positive moral emotion, emotional bonding and attachment (Fisher et al., 2013; Insel & Fernald, 2004; Keverne & Curley, 2004; Marazziti et al., 2013), we suggest that dysregulation in this area likely underpins, at least in part, symptoms of anhedonia, apathy, and social disconnection.

Importantly, each of these cortical regions (dlPFC, SFG, dACC, postcentral gyrus, posterior insula) is critically involved in moral processing, the experience and regulation of self-conscious emotion, and sensory integration and memory processing. Thus, the altered PTSD somatosensory processing exhibited in the PTSD group during MI event recall may affect several other processes needed for MI memory processing. Given the associations between MI event recall and altered activity among regions contributing towards somatosensory and emotional processing presented in study one, studies 3 and 4 investigated trauma-related symptoms characterized by altered somatosensory and emotional experience to gain further insight into the experience of MI.

In study two (Chapter 3; Lloyd et al., 2019), we investigated a new measure of posttraumatic TI, titled the Scal*e for Tonic immobility Occurring Post-trauma (STOP),* where TI is conceptualized as a last resort defense response within the defense cascade model of dissociation (Kozlowska et al., 2015; Schauer & Elbert, 2010). Within this model, dissociative responses are associated with reduced sensory integration (mediated by thalamic relay sites), possibly

leading to impaired memory encoding and integration. Consistent with this view, our findings suggest that TI results in an altered perceptual, sensorimotor and emotional experience including somatic detachment and dissociation – in response to overwhelmed personal resources. Although the direct relation between MI event exposure and TI remains unexplored, our results suggest this may be a particularly deleterious combination. Research has consistently shown that MI event exposure can lead to PTSD symptoms, and we presume this to include symptoms of TI. Further, our findings link negative alterations in cognition and mood with dampening of sympathetically driven viscerosensory information (mediated by posterior insula hypoactivity), a state that appears to be consistent with the physiological correlates of TI (i.e. sympathetic blunting and parasympathetic dominance). Importantly, the dissociative subtype of PTSD has been linked to a longer course of illness, higher treatment resistance and poorer functional outcomes (Armour et al., 2014; Bennett et al., 2015; Spiegel et al., 2013). Additionally, TI relies on social disengagement, potentially contributing towards, or compounding, the social impairment observed in MI. Moreover, our findings suggest that negative self-blaming emotions (e.g., guilt, shame, and embarrassment) can result from posttraumatic TI responses, perhaps intensifying existing guilt and shame. Unfortunately, as shown in Chapter 2, individuals with PTSD are likely to have difficulties regulating such negative self-blaming emotions due to hypoactivity within the SFG/mPFC. It is conceivable that posttraumatic TI responses may occur in response to persistent overwhelming

somatosensory information during MI recall in PTSD. As the assessment of TI states within posttraumatic symptomatology was effectively absent due to a dearth of appropriate self-report measures, we developed the STOP to support research and clinical recognition of posttraumatic TI states moving forward.

In the third study (Chapter 4, Lloyd et al., unpublished), we provide evidence that emotions are experienced in the body, and that level of emotional awareness may play a role in differentiating emotion-specific somatosensory maps. Specifically, we describe the emotion-specific somatosensory topography reported by trauma-exposed participants with and without alexithymia, a subclinical condition characterized by a lack of emotional awareness or, more specifically, difficulty in identifying and describing feelings and in distinguishing feelings from the bodily sensations of emotional arousal. Overall, the probable alexithymia group reported a comparatively blunted degree of sensation, relative to trauma-exposed controls; however, group differences were not found among the majority of emotion conditions, suggesting it is not the amount of sensation, but rather its location and intensity that differs between groups. Here, we posit that this diffuse and undifferentiated pattern of bodily sensation might contribute towards the emotional identification and interpretation difficulties observed in alexithymia. The lack of somatosensory specificity observed here is consistent with the notion of altered interoception in alexithymia, which has the potential to severely alter emotional experience, expression, and regulation (Khalsa et al., 2018). Given the altered somatosensory processing suggested in Chapter 2, we

may expect a similarly altered pattern of emotion-specific bodily sensation to be associated with the dampened viscerosensory activity observed in morally injured persons with greater negative alterations in cognition and mood. Although it remains to be tested, this brain and body pattern would also be consistent with posttraumatic TI within the defense cascade model of dissociation.

Thus, consistent with the central aim of this thesis, the abovementioned findings have critically expanded our understanding of how MI events are recalled and processed in the brain in MI-exposed military members and PSP with PTSD, relative to civilian MI-exposed controls. Next, we present a new embodied model of MI in PTSD, informed by the neuroscientific findings described in the foregoing discussion, to help guide psychotherapeutic interventions moving forward.

5.2 Towards Embodied Moral Injury Event Processing

Given the altered somatosensory processes associated with MI event recall and primary MI symptoms when PTSD is present, we propose a new framework of MI when PTSD is comorbid (Figure 1). Although MI event processing concerns higher-order cortical function (i.e. moral cognition), we attempt to expand upon current models to reflect a more comprehensive view. Our model therefore considers a bottom-up perspective, wherein subcortical processes influence sensory integration and cortical function necessary to MI event processing. This framework reflects the neural activation pattern of MI event recall and symptom expression described in this work. Further, we propose that when MI and PTSD

co-occur, heightened viscerosensory information may serve as an inescapable threat that has the potential to functionally elicit defensive responding, including late-stage posttraumatic TI responses (mediated by sympathetic shut down) and associated viscerosensory dampening and dissociative symptomatology. Normally, viscerosensory information (mediated by the posterior insula) gives rise to raw, emotion-specific bodily sensations that serve to motivate behaviour. Here, the posterior insula receives input from subcortical structures, whereas the anterior portion works in concert to facilitate conscious interoception and perception of higher-order emotion. Hyperactivity in the posterior insula is proposed to reflect heightened unpleasant visceral sensations related to blame-based emotions (e.g., *a pit in my stomach, internal gnawing*), which in turn, appears to alter activity within cortical structures that enable emotional regulation and memory integration (e.g., SFG/dmPFC, dlPFC). We further speculate that repeated recruitment of prefrontal controls (aimed at dampening blame-based emotion, e.g., dlPFC) results in emotional numbing (i.e. overmodulation of viscerosensory information). Theoretically, altered integration of MI sensory experiences and memory may also lead to impaired encoding of and responding to present-day internal and external stimuli, which may, in turn, contribute towards reduced social functioning.

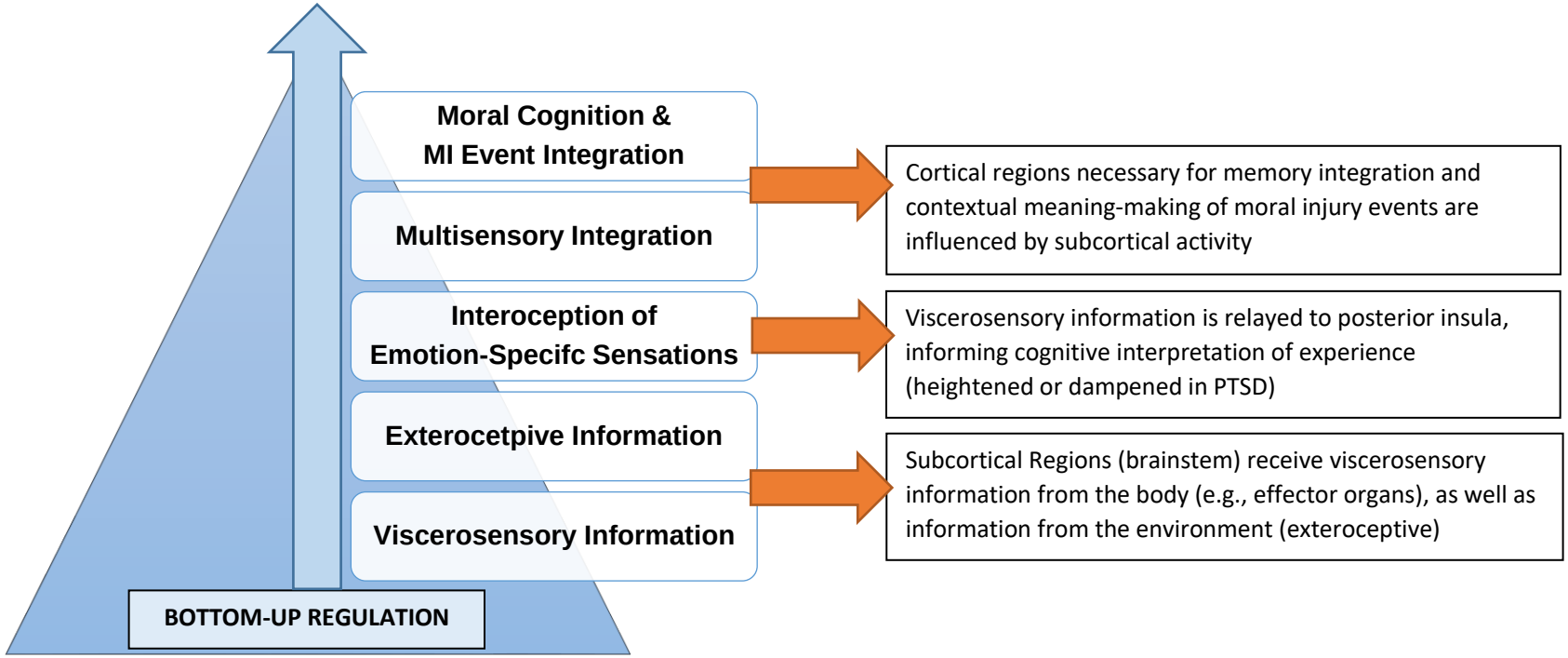

Figure 1. Towards Embodied Moral Injury Event Processing.Increased cortical activity among key SN nodes (dACC, posterior insula) reflects increased viscerosensory information processing when recalling a morally violating event, likely driven by activation within the innate alarm system, a subcortical defensive network that mediates survival-based responses like *flight*, *flight*, and *immobilization*. Cortical regions (dlPFC/CEN) attempt to regulate these strong, unpleasant blame-related bodily sensations (mediated by posterior insula hyperactivity). However, prefrontal hypoactivity (SFG) appears to impede normal emotion regulation when shame is present, altering integration of morally injurious events. Central to the DMN, the SFG facilitates a broad range of self-referential processing tasks, and hypoactivity in this area may point towards compromised self-evaluation, self-judgement, and emotional regulation. More severe negative alterations in cognition and mood are associated with a pattern of emotional numbing (posterior insula hypoactivity), possibly reflecting a compensatory strategy. Emotional numbing also characterizes alexithymia and immobilization symptoms.

5.3 Limitations

The results of the studies presented in this thesis should be interpreted with several limitations in mind. Perhaps the most significant limitation of all three studies was their cross-sectional design. This study design significantly limits the extent to which we can assume causality in our findings, and future well-controlled studies are needed to confirm these relations. Further, all studies were limited by self-selection and self-report biases. In study one, we did not control for specific emotions or differentiate between perpetration- and betrayal-based MI events. Our aim was to approach MI recall holistically as it may occur in real life or clinical settings. As more recent research has suggested a link between MI event-type and specific MI symptom expression, future studies controlling for MI exposure type and specific emotional expression are warranted. Further, this work did not explicitly test the relation between MI and TI, or, explore emotion-specific bodily sensation maps specifically within MI-exposed participants. Thus, any inferences made between these concepts are done so with great caution. For a detailed list of limitations, by study, please see the respective chapter.

5.4 Clinical Implications & Future Directions

The findings presented in this thesis suggest that morally injured individuals with military-related PTSD may have unique treatment needs. Traditional exposure-based methods for PTSD may be very difficult to tolerate when MI is also present, given the heightened viscerosensory information processing associated with MI event recall and the experience of MI-related shame. This is consistent with

research showing high drop-out rates for these treatments (e.g., Imel et al., 2013). Furthermore, CPT focuses heavily on cognitive reappraisal and contextualizing traumatic events to help alleviate guilt; however, this protocol does not target persistent sensory-emotional aspects of MI when comorbid with PTSD. This is consistent with research showing high residual symptoms that remain following such interventions (Langkaas et al., 2017). Thus, for this reason, in addition to the others noted earlier, PE and CPT may not be indicated for processing certain MI events.

Our findings further support alternative or adjunctive treatments to traditional front-line interventions for PTSD (i.e. prolonged exposure, CPT) when MI is comorbid. Therapeutic modalities aimed at increasing embodiment and somatosensory integration may be suitable adjuncts or alternatives for patients with MI and PTSD, particularly when exhibiting symptoms of alexithymia and/or TI as part of their symptom profile (indicating reduced sensory and memory integration). These psychotherapeutic approaches are aimed at promoting the resumption of bottom-up self regulation and restoring body-based control over automatic processes. Future research is needed to explore the relative efficacy of such treatments for ameliorating viscerosensory difficulties among individuals with comorbid MI and PTSD. Treatments that maximize group dynamics to re-establish trust and social bonds, as well as promote corrective social feedback are attractive interventions for MI, given the social nature of this injury. However, additional consideration should be given to individuals who are unlikely to seek

or engage in a group-based therapy due to severity of illness or other relevant factors (e.g., severe developmental or attachment trauma, when disclosure is professionally harmful, etc.).

Preventatively, these findings may have implications for enhanced operational training, through the inclusion of moral dissonance drills within high arousal environments. However, it is important to recognize that despite extensive training and preparation, events that transgress deeply held personal values may invariably result in MI symptom expression. Thus, psychoeducation about strong moral responses is indicated in operational settings and future study should explore MI protective factors (e.g., maintaining strong social connection and institutional support, fostering a culture of healthy coping strategies within high-risk groups, etc.).

5.5 *General Conclusions*

This thesis critically expands our understanding of MI when PTSD is present by describing the neurobiological underpinnings of MI event recall and core symptom expression (i.e., shame and negative alterations in cognition and mood). To the best of our knowledge, study one was the first study to describe the neural correlates of active MI event recall and MI symptom expression. This work highlights the importance of assessing and treating symptoms outside of the traditional conceptualization of PTSD, which may be particularly relevant to MI including posttraumatic TI as well as aberrant patterns of emotion-specific bodily sensation. This thesis makes a significant contribution to growing efforts to

address symptoms of MI and proposes new directions for assessment and treatment among individuals with comorbid MI and PTSD.

Relative to MI-exposed controls, individuals with military-related PTSD appear to be inundated with raw viscerosensory signals associated with blame-related emotions, when they approach their MI memory, and this may necessitate emotional detachment as a compensatory strategy, particularly when negative alterations in cognition and mood are present. This neural pattern may be reflected in the body through an aberrant pattern of intensified or muted emotion-specific bodily sensations, with the latter possibly reflective of symptoms of chronic TI or alexithymia. Although top-down interventions (i.e., third-wave CBTs) may be helpful in rehabilitating and regulating cortical activity, uptake of these strategies may be difficult without bottom-up regulatory techniques aimed at ameliorating strong, or dissociated, visceral sensations associated with MI and blame-based emotion.

Taken together, these findings extend our knowledge of and ability to treat MI symptom expression in PTSD. We hope that these findings will lead to changes in assessment and treatment practices that will allow individuals with MI and PTSD to live more functional, meaningful, and joyful lives.

References for Introduction and Conclusion

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