CORRELATES OF AUTOBIOGRAPHICAL MEMORY

NEURAL CORRELATES OF AUTOBIOGRAPHICAL MEMORY IN PTSD

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Abstract

This thesis examines the neural correlates of autobiographical memory retrieval among individuals with military-related PTSD, a population with increased illness burden and decreased treatment response. Although cognitive deficits, including in autobiographical recall, have been identified in PTSD, the mechanisms underlying these deficits remains unclear. Here, a series of studies have pointed towards alterations in the neural pathways underlying autobiographical recall in PTSD. Despite these observations, it remains unclear how key clinical variables, such as dissociation and early life trauma, might impact patterns of neural response during autobiographical recall in PTSD. The original work presented in this thesis examines emotional and traumatic memory retrieval in a sample of patients with military-related PTSD, trauma-exposed participants, and healthy controls and includes analysis of the relation between patterns of neural activation during autobiographical memory retrieval and key clinical variables associated previously with disease-related alterations in cognitive functioning. Our findings of increased lingual activity point towards aberrant implicitly cued memory function in military members and veterans with PTSD during traumatic memory retrieval. In addition, activation in the frontal operculum suggests that trauma-exposed military members and veterans without PTSD showed increased engagement of emotion regulatory strategies during retrieval of negative memories. Finally, we found an association between dissociative symptoms and activation observed in frontal and posterior regions during trauma memory retrieval in trauma-exposed military members and veterans. In addition to increasing core scientific

knowledge surrounding the neural correlates of autobiographical memory recall in trauma-exposed military members with and without PTSD, our results highlight the urgent need to characterize further the role of dissociative symptoms in cognitive dysfunction among individuals with PTSD.

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List of Abbreviations

ACC	Anterior Cingulate Cortex
AM	Autobiographical Memory
ANOVA	Analysis of Variance
BAI	Beck Anxiety Inventory
BDI	Beck Depression Inventory
CAPS	Clinician Administered PTSD Scale
CSA	Childhood Sexual Abuse
CTQ	Childhood Trauma Questionnaire
DERS	Difficulties in Emotion Regulation Scale
DID	Dissociative Identity Disorder
ELS	Early Life Stress
fMRI	Functional Magnetic Resonance Imaging
FO	Frontal Operculum
GLM	General Linear Model
HC	Healthy Controls
MCC	Middle Cingulate Cortex
MDD	Major Depressive Disorder
MDI	Multiscale Dissociation Inventory
NART	National Adult Reading Test
OFC	Orbitofrontal Cortex
OGM	Overgeneral Memory
PFC	Prefrontal Cortex
PTSD	Post-traumatic Stress Disorder
PTSD+DS	Post-traumatic Stress Disorder, dissociative subtype
PTSD-DS	Post-traumatic Stress Disorder, non dissociative subtype
RBANS	Repeatable Battery for the Assessment of Neuropsychological Status
ROI	Region of Interest
SCID	Structured Clinical Interview for DSM-IV
TEMC	Trauma-exposed Military Controls

Declaration of Academic Achievement

This thesis is comprised of 3 chapters: Chapter 1 provides background information regarding cognitive dysfunction reported in PTSD with a focus on military-related PTSD and autobiographical memory function; Chapter 2 is a manuscript of original work and is about to be submitted for publication; Chapter 3 provides a discussion of the results presented in Chapter 2 and also discusses the future directions of imaging research and cognitive function in PTSD. The original work presented in this thesis was conceived and designed by Dr. Margaret McKinnon and Dr. Ruth Lanius. Participant recruitment for the study took place in London, Ontario by London Health Sciences. I completed all the data analysis for this study. I performed a literature review and wrote the first draft of the manuscript for this study. The paper will be submitted to *Neuropsychologia*.

Chapter 1

General Introduction

This thesis outlines neuropsychological research exploring the neural correlates of cognitive dysfunction in neuropsychiatric illness, focusing, in particular, on autobiographical memory (AM) in post-traumatic stress disorder (PTSD). Briefly, two differing memory systems are described in the literature: 1) nondeclarative/ procedural memory which represents unconscious memory for skills and habits and 2) declarative memory which represents conscious memory for facts and events which includes AM (Squire, Knowlton, & Musen, 1993). Autobiographical memory represents memory for facts and past personal events and contributes to self awareness and an enduring sense of self (Conway, 2003; Conway & Pleydell-Pearce, 2000; Tulving, 1987). AM is hypothesized to be comprised of two independent components: episodic and semantic memory, where episodic memory consists of information regarding past personal events specific to time and place and semantic memory consists of context-independent information including general knowledge (Conway, 2003; Conway & Pleydell-Pearce, 2000; Tulving, 1987; Tulving, 2002). The study of AM is particularly salient to PTSD as AM dysfunction is central to the disorder, where patients may experience intrusive symptoms related to memory of a traumatic event (APA, 2013).

Notably, the current literature surrounding PTSD and AM remains varied, with the majority of studies reporting behavioural alterations in AM performance among individuals with PTSD (Hopper, et al., 2007; Lanius, et al., 2001; Lanius, et al., 2004; McKinnon, et al., 2016; Moore & Zoellner, 2007; Sartory, et al., 2013; Whalley, et al., 2013). Here, a number of studies point towards alteration in the key neural regions supporting AM among individuals with PTSD; these alterations are likely to underlie, in part, the behavioural changes in AM performance observed in this disorder. Critically, few studies have explored the relation between clinical variables (e.g. trauma severity, dissociation) and observed AM dysfunction in PTSD. The primary aim of the present thesis was to examine the functional neuroanatomy of AM retrieval in PTSD specific to a military population as military-related PTSD has been associated with an increased illness burden in PTSD. We provide further evidence of altered neural activity during AM retrieval in military members and veterans with PTSD and provide findings specific to dissociative symptoms. Research regrading dissociative symptomology and AM is currently emerging in the PTSD literature, where gaining an understanding of the relation between these two factors may prove critical in the development of targeted, successful treatments. In line with this hypothesis, we present findings that suggest an association between dissociative symptoms and traumatic memory retrieval in military trauma-exposed populations.

The following overview is comprised of five main sections that examine the current literature regarding PTSD and AM in PTSD. The first section provides a description of PTSD experienced as a result of military-related trauma, emphasizing the increased illness burden and cognitive dysfunction reported in this population. The second section begins with a general discussion of cognitive deficits in PTSD then delves into a comprehensive review of AM in PTSD. The third section details current neural models of PTSD, examining specifically emotional modulation. The focus of our original research is described in the fourth section, specific to clinical variables of interest. This chapter ends with a brief synopsis of the original research presented in chapter two.

1.1 Military-related PTSD

The current thesis focuses its investigation of PTSD on a trauma-exposed military population with and without PTSD. The original research presented in this thesis is in part a response to the increased burden of illness experienced by military members and veterans with PTSD. Research has shown that veteran samples with PTSD as compared to without PTSD present with increased disability (Goldberg, et al., 2014), increased functional impairment including impaired social and occupational functioning, and a poorer quality of life seen through increased reports of subjective distress, decreased life satisfaction, and well-being (Shea, et al., 2010; Zatzick, et al., 1997). In addition, men with PTSD who identify combat-related trauma as their most traumatic life event as compared to other trauma (e.g. physical trauma) are more likely to experience unemployment, have ongoing PTSD symptoms, and meet lifetime criteria for PTSD with the exception of men who identified molestation or sexual assault as most traumatic (Prigerson, Maciejewski, & Rosenheck, 2001). Taken together, these findings point towards an increased disease acuity among individuals with military-related PTSD, pointing towards the urgent need to elucidate the underlying mechanisms that may contribute to this increased burden of illness among military members with PTSD.

Despite knowledge of increased disease severity in military populations with PTSD, a limited amount of research has sought to identify alterations in neural functioning among military members with PTSD. Extant literature describes abnormal emotional processing, where as compared to trauma-exposed veterans without PTSD and healthy controls, veterans with PTSD exhibit increased dorsal anterior cingulate cortex (ACC) activation in response to negatively-valenced emotional images (van Rooij, et al., 2015) and, compared to trauma-exposed veterans without PTSD, decreased rostral ACC function during an emotional counting Stroop task (Shin, et al., 2001). These findings implicate the ACC in aberrant emotional processing in military-related PTSD, where this region is hypothesized to be involved in the autonomic, neuroendocrine, and behavioural expression of emotions (Lanius, et al., 2003). Additionally, Simmons et al. (2013) have shown that trauma-exposed veterans exhibit increased bilateral insular activation compared to trauma-exposed veterans without PTSD during anticipation of combat-related images where the anticipatory period was of an uncertain duration. The authors suggest that neural networks underlying unpredictability are altered in veterans with PTSD resulting in chronic distress, further highlighting the insula's role in processing emotional feeling states through the examination of internal changes in the body, including their emotional implications (Craig, 2002; Harricharan, et al., in press).

Decrements in inhibitory processes and working memory have also been shown in military-related PTSD. Van Rooij and colleagues (2014) examined reactive ("outright stopping") and proactive inhibition ("anticipation of stopping based on contextual cues") through a stop signal anticipation task where veterans with PTSD exhibited decreased inhibition of the pre/postcentral gyrus during reactive inhibition compared to veterans without PTSD and non-military controls, and decreased right inferior frontal gyrus activation compared to veterans without PTSD. The authors postulate that impaired processing of contextual cues in military PTSD patients may contribute more broadly to deficient fear processing seen in PTSD (van Rooij, et al., 2014). In a related study, the same group found that this deficit in inhibition was unaffected after treatment in veterans with PTSD compared to those without PTSD, identifying a cognitive domain uniquely affected in military-related PTSD and highlighting inhibition as a key therapeutic target (van Rooij, Geuze, Kennis, Rademaker, & Vink, 2015).

Impaired working memory has also been observed in a military population where Tian et al. (2014) found a marked decreased of prefrontal activation in veterans with PTSD as compared to healthy controls during the retrieval phase of a neutral digit span task. These results corroborate other findings in the literature that link altered working memory to decreased prefrontal activation in PTSD; the authors discuss prefrontal activation as a target for treatment as normalized right dorsolateral prefrontal activation has been associated with recovery from trauma (Lyoo, et al., 2011; Tian, et al., 2014).

Finally, a study of spontaneous brain activity examined resting state neural activity in military-related PTSD. As compared to veterans without PTSD, veterans with PTSD exhibited increased spontaneous amygdala, ventral ACC, insula, and orbitofrontal cortex activation (Yan, et al., 2013). This study also reported decreased spontaneous activity in the precuneus, dorsolateral prefrontal cortex (PFC), and thalamus in veterans with PTSD as compared to those without PTSD. Interestingly, the magnitude of this decreased activation in regions associated with self-referential processing was associated with low levels of re-experiencing symptoms in the PTSD group (Yan, et al., 2013). Yan and colleagues (2013) provide evidence for aberrant spontaneous function in key regions involved in PTSD symptomology such as the amygdala and insula, illustrating a baseline of altered function in military-related PTSD. Cumulatively, the few studies discussed above point towards abnormal neural activity in military-related PTSD, pointing towards the need to further elucidate patterns of neural activity in military-related PTSD. Identification of these networks and regions is critical not only to increase our core scientific knowledge but also to identify targeted treatments for PTSD. Critically, altered neural activity during AM retrieval has also been observed in military populations and is discussed below in section 1.2.2 alongside a detailed discussion of AM in PTSD.

1.2 Alterations in cognitive functioning among individuals with PTSD

PTSD is often discussed with respect to key clinical symptoms reflective of affective dysfunction, such as alterations in arousal and reactivity. A wide body of research, however, has also emerged surrounding the cognitive functioning of individuals with PTSD, revealing significant alterations in cognitive functioning in this population. Current literature and meta-analytic work on cognitive deficits in PTSD has identified impairments in verbal memory function, reporting low to medium effects sizes (d = -0.46, -0.74; Bremner, Vermetten, Afzal, & Vythilingam, 2004; Johnsen, & Asbjørnsen, 2008; Samuelson, et al., 2006; Scott, et al., 2014). Meta analysis also reveals that decrements in verbal memory that are related to trauma type with stronger effects observed in war-related trauma (d = -0.82) compared to sexual abuse-related trauma (d = -0.54; Johnsen, & Asbjørnsen, 2008). Johnsen and Asbjørnsen (2008) note that these experimental observations of verbal memory deficits in PTSD are in keeping with clinical observations where many patients with PTSD report experiencing memory impairment. Moreover, the authors emphasize the need for targeted treatment interventions for verbal memory in PTSD as these memory disruptions may underlie additional impairments in many aspects of daily function (Johnsen, & Asbjørnsen, 2008).

Research also points toward attentional deficits in PTSD, where altered attentional processes have been linked to the maintenance of PTSD through attentional biases to threats and symptoms of hypervigilance (Litz, et al., 1996; Scott, et al., 2014). A meta-analysis conducted by Scott and colleagues (2014) identified medium effect sizes for measures of attentional/working memory deficits in PTSD (d = -0.50). In military populations, marked deficits in sustained attention but not focus or shift of attention were found in veterans with PTSD compared to veterans without PTSD and healthy controls; these deficits were independent of intellectual functioning (Vasterling, Brailey, Constans, & Sutker, 1998; Vasterling, et al., 2002). Furthermore, decrements in visual attention have been reported in veterans with PTSD as compared to those without PTSD (Uddo, Vasterling, Brailey, & Sutker, 1993). These findings are in line with other meta-analytic work that demonstrates significant cognitive deficits in veteran populations with PTSD, as compared to those with a history of trauma, including attentional dysfunction (Qureshi, et al., 2011).

Additionally, speed of information processing in PTSD is also negatively affected, with medium effect sizes reflecting information processing dysfunction identified (d = -0.59; Scott, et al., 2014). Several authors have postulated that attentional deficits in PTSD are related to slowed information processing (Buckley, Blanchard, & Neill, 2000; Scott, et al., 2014), where research has shown that decrements on a Go/No-go task measuring attention in veterans with PTSD are related to impaired central processing as compared to healthy controls (Shucard, McCabe, & Szymanski, 2008). Although these cognitive domains overlap significantly, recent research suggests that attention and information processing are independent in healthy and neuropsychiatric populations, necessitating independent investigation of either cognitive domain (Scott, et al., 2016). Here, veterans with PTSD have demonstrated slower processing speeds as compared to veterans without PTSD on a wide battery of neuropsychological tests (e.g. the Stroop task; McNally, Kapsi, Riemann, & Zeitlin, 1990; Samuelson, et al., 2006).

Finally, research has shown impairments in executive function in patients with PTSD where a recent review reported decreased performance on neuropsychological tasks assessing executive function (e.g. Wisconsin card sorting task) in patients with PTSD compared to controls (Polak, Witteveen, Reitsma, & Olff, 2012). Furthermore, Polak and colleagues (2012) note that military-related trauma is associated with greater decrements in executive functioning as compared to other sources of trauma, such as sexual abuse. Meta-analytic work reports small effect sizes of executive impairment in PTSD compared to trauma-exposed individuals without PTSD (*Hedges' g* = 0.41) and non-trauma exposed controls (*Hedges' g* = 0.46; Woon, Farrer, Braman, Mabey, & Hedges, 2017). Here, deficits of executive function may lead to significant impairments in the daily functioning of patients with PTSD as executive function supports decision making, planning, and goal directed behaviour (Snyder, Miyake, & Hankin, 2015; Woon, et al., 2017).

Although the evidence discussed thus far points toward marked cognitive impairment in PTSD, not all literature provides corroborative evidence. For example, a study of intellectual ability, learning, memory, attention, visuospatial ability, executive functioning, language, and psychomotor speed in veterans with PTSD, veterans with a prior history of PTSD currently experiencing no symptoms, veterans without PTSD, and a healthy control group failed to report any between group differences in neuropsychological performance (Crowell, Kieffer, Siders, & Vanderploeg, 2002). Additionally, Neylan et al. (2004) reported no differences between veterans with PTSD and healthy controls on measures of attention, learning, or memory. As the literature regarding neuropsychological function in PTSD remains varied, further research is required to elucidate further the cognitive sequalae of PTSD.

1.2.1 Key variables that may impact cognitive functioning among patients with PTSD

Work in our own laboratory has focused heavily on identifying the underlying neural and behavioural correlates that may account for differences in symptom expression and cognitive dysfunction among individuals with PTSD. For example, our own work and the work of others has explored the relation between dissociative symptoms and alterations in key facets of cognitive and emotional functioning, including working memory (Elzinga, et al., 2007; McKinnon, et la., 2016), attention (de Bellis, Woolley, & Hooper, 2013; McKinnon, et al., 2016), executive functioning (Deprince, Weinzierl, & Combs, 2009; McKinnon, et al., 2016), and social cognition (McKinnon, et al., 2016; Renard, Pijnenborg, & Lysaker, 2012). Described briefly, dissociation was first identified by Janet (1901) as a psychobiological response to threat (Dalenberg, et al., 2012) and refers to a disturbance of the integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behaviour (APA, 2013).

Approximately 15-30% of patients with PTSD present with core dissociative symptoms of depersonalization (feeling outside of, or as if you do not belong to your own body) and derealization (feeling as though things around you are strange and unfamiliar), leading to a diagnosis of the recently identified dissociative subtype of PTSD (PTSD+DS; Armour, Karstoft, & Richardson, 2014; Bennett, Modrowski, Kerig, & Chaplo, 2015; Hansen, Ross, & Armour, 2017; Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012; Stein et al., 2013; Wolf, Miller, et al., 2012). Compared to non-dissociative PTSD (PTSD-DS), PTSD+DS is associated with increased symtpom severity, heightened symptoms of depression and alcohol abuse, and increased psychiatric comorbidity across military and civilian samples (Bennett et al., 2015; Blevins, Weathers, & Witte, 2014; Tsai, Armour, Southwick, & Pietrzak, 2015; Waelde, Silvern, & Fairbank, 2005; Wolf, Lunney, et al., 2012; Wolf, Miller, et al., 2012).

Interestingly, dissociative symptoms have been shown to affect cognitive function in PTSD and trauma exposure. Current work describes a relation between dissociative symptoms and attentional impairments where dissociative symptoms have been shown to predict attentional deficits in children exposed to childhood sexual abuse (Kaplow, Hall, Koenen, Dodge, & Amaya-Jackson, 2008). Similary, dissociative symptoms have been shown to correlate negatively with attentional performance as measured by the Conners' Continuous Performance Test-II in youth with exposure to pediatric maltreatment with and without PTSD (de Bellis, Woolley, & Hooper, 2013). Additionally, research has shown that veterans with PTSD and a comorbid dissociative disorder demonstrate impaired performance on measures of attention (e.g. digit span distractibility test), autobiographical memory (e.g. autobiographical memory interview), and verbal memory (e.g. Hopkins verbal learning test) as compared to veterans with PTSD without a comorbid dissociative disorder (Roca, Hart, Kimbrell, & Freeman, 2006). These findings are corroborated by research on women with exposure to childhood sexual abuse, which demonstrates an association between dissocaitive symptoms and decrements in long- and short-term visual memory and executive function as compared to women without exposure to childhood sexual abuse (Rivera-Vélez, González-Viruet, Martínez-Taboas, & Pérez-Mojica, 2014). Deficits in executive function have also been shown in children exposed to familial trauma as compared to children exposed to non-familial trauma and no trauma where dissociative symptoms and trauma-exposure status accounted for a unique portion of variance in executive performance, controlling for anxiety symptoms, socio-economic status, and potential traumatic brain injury (Deprince, Weinzierl, & Combs, 2009). Additionally, research conducted by Minshew and D'Andrea (2015) on women exposed to chronic interpersonal violence reported a negative correlation between dissociation and explicit memory for general threat words. Cumulatively, the evidence discussed here indicates an association between dissociative symptoms and a variety of cognitive functions in trauma-exposure and/or PTSD, a conclusion supported by a recent review on the topic (McKinnon, et al., 2016).

Exposure to early life adversity or childhood trauma is also discussed in the literature as a key factor affecting cognitive function. For example, research involving healthy adults has shown an association between childhood emotional abuse and impaired spatial working memory as well as an association between childhood physical neglect and impaired working and pattern recognition memory (Majer, Nater, Lin, Capuron, & Reeves, 2010). Moreover, physical abuse and neglect have been associated with decrements in cognitive flexibility in healthy adolescents as measured by the Wisconsin card sorting task (Spann, et al., 2012). Majer and colleagues (2010) and Span and colleagues (2012) emphasize the importance of considering the effects of childhood trauma in adolescents and adults as the observed deficits may lead to greater cognitive dysfunction or an increased risk for the development of psychopathology. Additionally, a review on the effects of early life stress (ELS) on cognitive function reported that ELS was associated with decreased intellectual performance, language abilities, academic success, planning, and inhibitory control (Pechtel, & Pizzagalli, 2011). Similarly, deficits in executive functioning in internationally adopted children have been reported in the literature where less time spent with the birth family before placement in an institution and a low quality of social/physical care in institutions emerged as a predictor of decreased performance on measures of executive function (e.g. the dimensional change card sort scale; Hostinar, Stellern, Schaefer, Carlson, & Gunnar, 2012). Taken together, these findings suggest that early life adversity or childhood trauma can affect cognitive function later in life, where dysfunction is recorded up to adulthood.

Likewise, illness severity has been shown in the PTSD literature to relate to cognitive dysfunction. A recent review examining executive functioning in PTSD reported a strong association between symptom severity and decrements in attention regulation and response inhibition (Aupperle, Melrose, Stein, & Paulus, 2012). In a similar vein, Iacoviello et al. (2014) have demonstrated a positive association between increased attention-bias variability and PTSD symptom severity in their PTSD and trauma-exposed control group, providing further evidence of the relation between PTSD symptom severity and attentional processes. Additionally, an imaging study examining memory of faces in PTSD observed that PTSD symptom severity predicted decreased performance on a facial recognition task (Dickie, Brunet, Akerib, & Armony, 2008). Although limited, the evidence presented here points toward an association between illness severity in PTSD and cognitive dysfunction.

Emotion regulation has also been shown to impact cognitive function as cognition is hypothesized to be regulated, in part, by affect (Storbeck, & Clore, 2007). Bardeen and Orcutt (2011) determined that attentional threat bias observed in PTSD may be explained by a difficulty disengaging from threat-related stimuli rather than an increased engagement with threat-related stimuli. Here, the authors observe that those with higher levels of posttraumatic stress were not able to employ attentional control to disengage from threat-related stimulus when perceived threat was increased (Bardeen & Orcutt, 2011). This research provides an example where emotion is shown to, in part, regulate attentional processes. The neural correlates of working memory have also been shown to be impacted by affective processing where individuals with PTSD completing a task exhibit increased activation in ventral regions associated with emotional processing in response to task-irrelevant trauma-related distractors as compared to a trauma-exposed control group (Morey, et al., 2009). Here, activation in regions associated with working memory and attention were disrupted by both task-irrelevant trauma- and non trauma-related distractors (Morey, et al., 2009). Morey and colleagues (2009) were able to provide further evidence of a relation between affect and cognition through disrupted emotional processing in response to threat-related distractors. Corroborative evidence is reported in a recent review of executive function in PTSD where Aupperle and colleagues (2012) discuss a link between emotional processing and both attention and working memory processes while also describing findings that do not uphold such a conclusion. As such, research regarding affect and cognition in PTSD remains varied and requires further investigation.

1.2.2 The current thesis: AM in military members and veterans with PTSD and/or trauma exposure

Here, we explore alterations in the neural architecture of one particular domain of cognitive functioning, autobiographical memory, among military members and veterans with PTSD. A significant body of work has sought to characterize alterations in AM among individuals with PTSD, revealing, at times conflicting findings. Notably, alterations in AM performance have been recorded across neuropsychiatric conditions, including schizophrenia (McLeod, Wood, & Brewin, 2006), depression, and PTSD (Moore & Zoellner, 2007; Parlar, Densmore, Hall, Laniys, & McKinnon, 2018). Moreover, AM dysfunction is central to PTSD as noted in the general introduction where individuals may experience intrusive symptoms related to memory of a trauma (APA, 2013). Importantly, research of AM in PTSD is crucial as memory for past personal events contributes significantly to self awareness and identity where disruptions therein may be involved in the onset and maintenance of PTSD (Contractor, et al., 2018; Kleim, & Ehlers, 2008).

Studies of AM often report overgeneral memory (OGM) retrieval in PTSD and depression where OGM consists of categorical recall that refers to groups of events with common elements (e.g. when I go the bank) and/or extended recall that refers to events that last longer than a day (e.g. I spent a fall semester of university studying in Europe), overall lacking details specific to time and place (King, et al., 2010; Kuyken, Howell, & Dalgleish, 2006; McNally, Lasko, Macklin, & Pitman, 1995; Moore & Zoellner, 2007; Schönfeld, Ehlers, Böllinghaus, & Rief, 2007; Ono, Devilly, & Shum, 2016). Meta-analytic work on trauma and PTSD has described a range of small to medium effect sizes of OGM in trauma exposure without PTSD (*Hedges' g* = 0.39 - 0.63) and a strong range of effect sizes of OGM in PTSD (*Hedges' g* = 0.90 - 1.91) where the authors suggest PTSD symptomology may contribute to the increased effect size seen in the PTSD compared to trauma-exposed group (Ono, et al., 2016).

Research has demonstrated OGM in assault survivors with and without PTSD where individuals with PTSD recalled fewer and more general memories than individuals without PTSD when instructed to supress memories of assault (Schönfeld, et al., 2007). Furthermore, Schönfeld and colleagues (2007) reported that PTSD symptom severity correlated with instances of OGM retrieval. Additionally, evidence of OGM retrieval in non-Western populations has emerged where refugee and asylum-seeking populations with PTSD and depression have been shown to retrieve a lower proportion of specific memories compared to those without PTSD or depression (Graham, Herlihy, & Brewin, 2014). Interestingly, research conducted by Ogle and colleagues (2013) on adolescent and adult populations exposed to childhood sexual abuse (CSA) report some contradictory evidence. Here, decreased specificity of memory retrieval was only found in adolescents exposed to CSA compared to adolescent controls, an effect not reported in between adults with compared to without exposure to CSA (Ogle, et al., 2013). Furthermore, adults exposed to CSA exhibited increased specificity in recall of childhood events compared to adolescents exposed to CSA (Ogle, et al., 2013). These results suggest developmental differences in OGM not seen in research previously discussed above. Moreover, Ogle et al. (2013) also reported that PTSD symptomology was correlated positively with increased autobiographical memory specificity in a subset of their sample that identified a history of CSA. Their results are corroborated by other research which demonstrated that the frequency/severity of flashbacks of trauma correlated negatively with reduced AM specificity in refugees (Moradi, et al., 2008). This evidence contradicts the findings of Schönfeld and colleagues (2007) which suggest that illness severity is related to decreased overgeneral memory recall in PTSD. These contrasting reports of OGM in PTSD raise questions of the incidence of this phenomenon and its relation to illness severity.

Although current literature above lacks a consensus, OGM retrieval has been demonstrated in military-related trauma and PTSD. For example, combat Vietnam veterans with PTSD have been shown to recall fewer specific AMs compared to combat Vietnam veterans without PTSD, demonstrating a particular difficulty in retrieving specific positive AMs (McNally, Lasko, Macklin, & Pitman, 1995). Such an effect is also recorded in Iranian combat veterans where Iranian combat veterans with PTSD are shown to produce fewer specific AM details compared to those without PTSD and a healthy control group (Moradi, Abdi, Fathi-Ashtiani, Dalgleish, & Jobson, 2012). Moreover, research has shown that overgenerality extends beyond AM where veterans with PTSD demonstrate OGM for past personal events and overgeneral imagined personal futures in response to neutral cues compared to veterans without PTSD, indicating overgeneral effects in future thinking as well (Brown, et al., 2013). Despite a lack of consensus in the literature, OGM in PTSD and depression is theorized to serve as a protective strategy to prevent the retrieval of emotionally distressing material, such as trauma memories (Healy & William, 1999; Williams, et al., 2007).

Some research of AM in PTSD also works to determine effects related specifically to episodic or semantic components of AM. Examining these systems of AM can provide crucial knowledge of the type of information that may be affected in PTSD, allowing for a deeper understanding of cognitive sequalae in PTSD and enabling a focused treatment approach. Moradi et al. (2008) reported that increased specificity of episodic AM details in a sample of refugees with PTSD was related to increased frequency of trauma-related flashbacks whereas cancer survivors with posttraumatic stress provided fewer semantic AM details compared to a healthy control group. Here, differentially affected episodic and semantic details may point toward an effect of trauma type. Additionally, research on PTSD in HIV patients has shown that individuals with PTSD and HIV recall fewer episodic and semantic details compared to a healthy control group (Abdollahi, Moradi, Hasani, & Jobson, 2012). Specific to military-related trauma, research on combat veterans has reported that combat veterans with PTSD generate fewer episodic than semantic details when recalling AMs or imagining future personal events (Brown, et al., 2014).

These findings are corroborated by further research which not only describes decreased production of episodic and semantic details in combat veterans with PTSD as compared to those without, but also finds working memory to mediate the relation between PTSD diagnosis and semantic memory recall (Moradi, et al., 2012). As deficits in executive function have been observed in PTSD, research hypothesizes that a lack of executive control allows for task-irrelevant material to interrupt the retrieval response, resulting in OGM seen in PTSD and depression (Williams, et al., 2007). However, it remains unclear why working memory specifically relates to the retrieval of semantic AM details in PTSD. Although the above studies point toward differences in episodic and semantic recall in PTSD, research remains unsure of the underlying mechanisms that may contribute to these results.

Current literature of AM in PTSD also identifies dissociative symptoms to be related to altered AM function observed in the disorder. Notably, dissociative symptomology has been associated with altered AM function in both clinical and general populations. In groups with trauma exposure, PTSD, and borderline personality disorder, dissociative symptoms have been linked to overgeneral memory retrieval (Chae, Goodman, Eisen, & Qin, 2011; Jones, et al., 1999). Studies of healthy populations corroborate these findings, demonstrating decreased recall for events and trauma-related stimuli in those with induced dissociative states or increased dissociative experiences in daily life, respectively (Bergouignan, Nyberg, & Ehrsson, 2014; Olsen, & Beck, 2012).

Similar to dissociative symptoms, early life adversity/childhood trauma has also been associated with AM dysfunction. Studies have shown a link between early life adversity and overgeneral AM retrieval in depression and trauma exposure (de Decker, Herman, Raes, & Felen, 2003; Henderson, Hargreaves, Gregory, & Williams, 2002; King, et al., 2010; Meesters, Merckelbach, Muris, & Wessel, 2000). However, little work has been completed regarding the effects of childhood trauma and the neural correlates of AM retrieval in PTSD. One study examining the neural correlates of AM retrieval in depressed individuals exposed to trauma was able to report an association between self-reported childhood physical neglect and decreased dorsal ACC activity during neutral memory retrieval (Parlar, Densmore, Hall, Lanius, & McKinnon, 2018). These findings indicate a possible role of childhood trauma in AM dysfunction in depression, notably observed in a region involved in the experience of emotion. As childhood trauma has been shown to be associated with altered AM function in depression, it raises the question of its role in other disorders.

Finally, emotion regulation has also been shown to impact AM retrieval in PTSD. For example, McNally and colleagues (1995) demonstrated OGM recall for negative and positive memories in veterans with PTSD as compared to veterans without PTSD, an effect especially pronounced in positive memory retrieval. Furthermore, the same group reported similar findings where combat veterans with PTSD demonstrated reduced AM specificity when retrieving neutral, positive, and negative memories once primed with a combat-related videotape, as compared to combat veterans without PTSD (McNally, Litz, Prassas, Shin, & Weathers, 1994). Again, the observed lack of specificity was pronounced in positive memory retrieval (McNally, et al., 1994). Taken together, these findings demonstrate parallel AM and emotional dysfunction as PTSD patients exhibit decreased AM specificity and difficulty in recalling positive information. These authors suggest that patients with PTSD may have difficulty retrieving positive information regarding the self, possibly contributing to maintenance of the disease (McNally, et al., 1995; McNally, et al., 1994).

1.2.2.1 Functional neuroanatomy of AM in PTSD

Augmenting the above behavioural research, imaging findings provide further evidence of AM dysfunction in PTSD. Despite knowledge that healthy AM retrieval includes a core network of left-lateralized regions including medial and ventrolateral prefrontal, medial and lateral temporal and retrosplenial/posterior cingulate cortices, the temporoparietal junction, and the cerebellum (Svoboda, McKinnon, & Levine, 2006), current knowledge of the neural correlates of traumatic memory recall in trauma-exposed individuals with and without PTSD continues to evolve.

Present research investigating altered AM in PTSD suggests dysfunction specific to cortical midline structures (Sartory, et al., 2013). A recent metanalysis conducted by Sartory and colleagues (2013) reported that patients with PTSD exhibit increased activation in the midline retrosplenial cortex, precuneus, ACC, and left angular gyrus during symptom provocation compared to controls. These regions are hypothesized to be central to self-referential processing and AM function (Cavanna, & Trimble, 2006; Menon, 2011; Sartory, et al., 2013; Sestieri, Corbetta, Romani, & Shulman, 2011; Spreng, Mar, & Kim, 2009; Svoboda, et al., 2006), suggesting that individuals with PTSD may exhibit an increased level of re-experiencing associated with intrusive recall of traumatic memories (Sartory, et al., 2013). These meta-analytic findings are derived from studies that examine a range of trauma indices, such as sexual abuse and motor vehicle accidents. Research specific to military-related PTSD also reports similar findings. For example, Shin and colleagues (2004) found decreased medial frontal activation during traumatic compared to neutral AM recall in veterans with PTSD as compared to veterans without PTSD. Furthermore, their results showed that medial frontal activation was inversely correlated with changes in amygdala function in the PTSD group, a region heavily implicated in the limbic response, where symptom severity correlated positively with right amygdala activation during traumatic memory retrieval (Shin, et al., 2004). The authors speculate that their results provide further evidence of a reciprocal relation between medial frontal regions and the amygdala which may underlie heightened emotionality observed in PTSD (Shin, et al., 2004). However, altered amygdala activation was not reported in veterans with PTSD or trauma-exposed veterans without PTSD in a similar study that investigated response to traumatic and neutral AMs (Britton, Phan, Taylor, Fig, & Liberzon, 2005). Decreased medial prefrontal activation in veterans with PTSD as compared to those without was also observed by Bremner and colleagues (1999), alongside decreased middle temporal activation in the PTSD compared to non-PTSD group in response to trauma imagery and sounds. Bremner et al. (1999) posit that decreased medial frontal activation contributes to the altered emotional response seen in PTSD due to its hypothesized role in emotion. They also suggest that decreased medial temporal activation may relate to heightened fear responses in PTSD as the medial temporal lobe has been shown to have inhibitory connections with the amygdala (Bremner, et al., 1999). Taken together, findings regarding military-related PTSD and the neural correlates of AM primarily emphasize altered medial frontal activation in this population, proposing a marked impairment in emotional processing.

Current literature also suggests flashback retrieval may be linked to brain activity indicative of nonverbal retrieval during trauma memory recall in PTSD. Lanius and colleagues (2004) demonstrated increased activity between the right anterior cingulate gyrus and the right occipital lobe, parietal lobe, posterior cingulate gyrus, and caudate during traumatic memory recall in PTSD as compared to controls. Notably, these posterior regions are hypothesized to contribute to nonverbal episodic retrieval (Cabeza & Nyberg, 1997; Cabeza, & Nyberg, 2000) and may be implicated in AM dysfunction in PTSD. The authors suggest those in the PTSD group may have experienced trauma memory recall as imaged-based flashbacks, a conclusion supported through the evidenced nonverbal retrieval (Lanius, et al., 2004). It is important to note that decreased or failed medial prefrontal activation is often reported in PTSD literature regarding symptom provocation (Hopper, Frewen, van der Kolk, & Lanius, 2007). For example, Lanius and colleagues (2001) reported decreased medial frontal cortex activity in PTSD compared to controls during trauma memory retrieval. The authors posit that these findings may be related to high levels of arousal experienced during the recall of emotional memories (Lanius, et al., 2001). Cumulatively, the above research suggests that retrieval of traumatic events may be associated with high levels of emotional arousal and re-experiencing that may or may not be able to be verbally described, such as bodily sensation and/or visual images.

The original research presented in this thesis includes an important between-subjects comparison between trauma exposed populations with and without PTSD. Although not all PTSD literature includes a trauma-exposed control group, such a comparison can prove critical in discerning phenomenon due to trauma exposure and/or the development of PTSD. In their meta-analytic work, Sartory and colleagues (2013) reported increased activity in lateral and dorsal sensory association areas in trauma-exposed participants compared to PTSD during symptom provocation (majority of which consisted of autobiographical material). The authors suggest these findings indicate increased attention to stimulus material in the trauma-exposed group, therefore suggesting a lack of attention to stimulus material in the PTSD group (Sartory, et al., 2013). They propose the observed lack of sensory activation in PTSD patients may indicate that self-referential processing in PTSD, induced via symptom provocation, may have an inhibiting effect on the ability to process concomitant environmental stimuli (Sartory, et al., 2013). Similarly, a study conducted by Hayes and colleagues (2011) found that individuals with PTSD relied more heavily on gist-based cues than contextual details when completing a recognition task of trauma scenes they had been exposed to a week prior, resulting in increased false alarm rates compared to trauma-exposed controls. These findings provide behavioral evidence of altered attention to stimuli in patients with PTSD, specific to episodic memory. Taken together, a consistent difference between trauma-exposed individuals with and without PTSD has emerged. Our current research aims to augment the literature and investigate a comparison between these two groups specific to personal, emotional AMs and military-related trauma.

Extant literature also implicates altered thalamic, anterior cingulate, and insula function in altered AM in PTSD. For example, high levels of arousal experienced during the retrieval of traumatic and negatively valenced memories have been linked to decreased thalamic function in PTSD (Lanius, et al., 2001; Lanius, et al., 2003). Altered thalamic function may hold implications for sensory processing; these authors discuss the possibility of a disrupted relay of sensory information from the thalamus to the frontal cortex, cingulate gyrus, amygdala, and hippocampus (Lanius, et al., 2001). This disruption may also contribute to dissociation and/or flashback-like recall (Lanius, et al., 2001). Notably, diminished ACC activation during symptom provocation is commonly reported in the PTSD literature (Hopper, et al., 2007) where the region is hypothesized to be critical to the experiential aspects of emotion, including autonomic, neuroendocrine, and behavioural expression (Lanius, et al., 2003). Indeed, Lanius and colleagues (2003) posit that decreased thalamic and anterior cingulate activation during negative memory retrieval in PTSD compared to controls may contribute to emotion dysregulation observed in the disorder.

Research has also implicated the insula in aberrant AM function in PTSD, a region commonly activated alongside the limbic system (Karama, Armony, & Beauregard, 2011) and has been associated with involuntary vivid recollection. Here, the insula is hypothesized to play a significant role in processing emotional feeling states through monitoring and identification of internal changes within the body, including their emotional implications (Craig, 2002; Harricharan, et al., in press). In the PTSD literature, high levels of re-experiencing symptoms during online traumatic memory retrieval have been associated with increased right anterior insula activity (Hopper, et al., 2007). Additionally, the same study reported a negative correlation between dissociative symptoms and right insula activity, suggesting a neural substrate for emotional underengagement seen in dissociative responses (Hopper, et al., 2007). Insula activation has also been directly related to flashback recall. A study conducted by Whalley and colleagues (2013) reported right insula activity to be correlated positively with flashback as compared to episodic components of the same traumatic memory in PTSD compared to their comparator groups. These research findings together suggest that insular function may underlie involuntary flashbacks in PTSD, a role in keeping with its primary hypothesized function in regulating emotional and body state responses. However, not all research is able to provide corroborative findings. For example, Shin et al. (2004) did not report increased insula activation during trauma memory retrieval in veterans with PTSD as compared to those without.

In line with Hopper and colleagues' investigation of dissociative symptoms and their relation to AM, Lanius and colleagues (2002) conducted research on dissociative responses and their relation to symptom provocation in PTSD. Here, PTSD patients in a dissociative state exhibited increased activation of the superior and middle temporal gyri, the inferior frontal gyrus, the occipital lobe, the parietal lobe, the medial frontal gyrus, medial prefrontal cortex, and ACC as compared to those without PTSD (Lanius, et al., 2002). The authors suggest these regions underlie dissociative responses in PTSD where excessive frontal (e.g. medial frontal cortex) inhibition of limbic activation is hypothesized to lead to dissociative symptomology (Lanius, et al., 2002; Lanius, et al., 2010). As the current literature on dissociative symptoms and AM function in PTSD is limited, research presented in chapter two includes an investigation of dissociative symptoms and the neural correlates of AM function in military members/veterans with and without PTSD.

1.3 Neural Models of PTSD

In the present thesis, we focus on identifying alterations in patterns of neural activation that may underlie the poor AM performance observed among military members and veterans with and without PTSD and healthy controls. Our work here is informed heavily by prior research that has identified clear differences in patterns of neural activation between individuals with and without the dissociative symptoms of PTSD. Critically, the majority of these studies have compared patterns of neural activation between individuals with and without the dissociative subtype of PTSD. As noted, approximately 15-30% of individuals with PTSD present with dissociative symptoms of depersonalization (feeling outside of, or as if you do not belong to your own body) and derealization (feeling as though things around you are strange or unfamiliar), leading to a diagnosis of the recently identified dissociative subtype (Armour, Karstoft, & Richardson, 2014; Bennett, Modrowski, Kerig, & Chaplo, 2015; Hansen, Ross, & Armour, 2017; Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012; Stein et al., 2013; Wolf, Miller, et al., 2012). Recent work has identified two contrasting patterns of emotion modulation in PTSD, describing patterns of under and overmodulation in PTSD-DS and PTSD+DS, respectively (Lanius, et al., 2010).

Notably, individuals with PTSD+DS exhibit a top-down pattern of emotion overmodulation hypothesized to arise out of excessive corticolimbic inhibition (Lanius, et al., 2010). Here, emotion overmodulation is linked to an abnormal increase in the activation of emotion and arousal modulation areas, including the medial PFC and dorsal ACC (Lanius, et al., 2010). This pattern of hyperactivation subsequently results in decreased activation of limbic regions, most notably the amygdala (Lanius, et al., 2010). Emotion overmodulation has been strongly associated with hypoarousal and emotional detachment central to depersonalization and derealization symptoms of PTSD+DS (Flemingham, et al., 2008; Lanius, et al., 2010; McKinnon, et al., 2016; Nicholson, et al., 2015; Nicholson, et al., 2017).

The bottom-up pattern of emotion undermodulation observed in PTSD-DS is thought to arise from a failure of corticolimbic inhibition (Lanius, et al., 2010). Here, midline anterior regions involved in emotion and arousal modulation display abnormally low activation, including the ventromedial prefrontal cortex (PFC) and the rostral anterior cingulate cortex (ACC; Lanius, et al., 2010). This decrease in corticolimbic inhibition is associated with increased limbic activity, a pattern most prominent in the amygdala (Lanius, et al., 2010). Indeed, amygdala dysfunction has been shown to have a significant role in the development and maintenance of symptoms experienced in PTSD (Aghajani, et al., 2016; Birn, et al., 2014; Etkin, & Wager, 2007; Lanius, Frewen, Tursich, Jetly, & McKinnon, 2015; Mickleborough, et al., 2011; Patel, et al., 2012; Pitman, et al., 2012; Shin, & Liberzon, 2010; Stevens, et al., 2013; Weston, 2014). This bottom-up pattern of emotion undermodulation is thought to give rise to hyperarousal and re-experiencing symptoms observed most frequently in individuals with a non-dissociative presentation of PTSD (Harricharan, et al., in press; Lanius, et al., 2010; Lanius, et al., 2003; Nicholson, et al., 2017; Weston, 2014).

Patterns of emotion under and overmodulation in PTSD are relevant to the current thesis as key regions involved in altered emotion modulation in PTSD overlap with key regions identified in the functional neuroanatomy of altered AM function in PTSD, such as the ACC and medial frontal regions. As the original research presented in this thesis includes the recall of emotional AMs, regions implicated in both altered emotional and AM neural function are of interest. Additionally, as research in chapter two examines dissociative symptoms and the neural correlates of AM retrieval in PTSD, frontal regions implicated in the dissociative response described above are of interest as they may be related to AM retrieval (Lanius, et al., 2002).

1.4 Focus of the present investigation

The current thesis examines the functional neuroanatomy of AM retrieval in trauma-exposed military members and veterans with and without PTSD, and healthy controls. Additional to examining the functional neuroanatomy of AM retrieval in PTSD, the current thesis also investigated the association between clinical variables and the neural correlates of AM retrieval in PTSD. The current thesis focused on the following three clinical variables: 1) dissociative symptoms, 2) early life adversity/childhood trauma, 3) and emotion regulation. As noted throughout this chapter, evidence has accumulated throughout the literature demonstrating an association between dissociative symptomology and poorer cognitive function, including AM performance. Critically, the study of dissociative symptoms in military-related PTSD is needed as both military-related trauma exposure and dissociative symptoms have been identified to result in an increased acuity of PTSD, as discussed above. In a similar vein, early life adversity has been identified to contribute to cognitive dysfunction in PTSD. Notably, exposure to childhood trauma has been shown to occur at an increased rate in military compared to civilian populations (Affifi, et al., 2006) and has been linked to a increased perceived need for and higher use of mental healthcare (Turner, et al., 2017), reduced health-related quality of life (Katon, et al., 2015) and suicide attempts and suicide (Affifi, et al., 2006). Taken together, these findings present a need to investigate the effects of early life adversity/childhood trauma in military members with PTSD. Finally, emotion regulation in AM was considered in this thesis given the reports of altered emotion modulation in PTSD, discussed above (see the following review for further evidence of an association between emotion and cognition in PTSD: Hayes, VanElzakker, & Shin, 2012).

1.5 Overview of the presented work

The experimental research presented in chapter two reflects a functional magnetic resonance imaging study examining emotional AM retrieval in trauma-exposed military members/veterans with and without PTSD, and healthy controls. Our findings reveal altered neural activation during AM retrieval in PTSD and trauma-exposed controls. Our research also demonstrates a significant positive correlation between dissociative symptoms and activity in frontal and posterior regions during trauma memory retrieval in a trauma-exposed military population. The findings presented in chapter two substantiate the current notion of altered neural activation during AM retrieval in PTSD and highlight a need to further examine dissociative symptomology in PTSD, significantly contributing to the burgeoning field of cognitive research in PTSD. Furthermore, our original research also provides insight into military-related PTSD and the mechanisms which may result in the reported increased illness burden.

Chapter 2

Neural Correlates of Autobiographical Memory in Military Members and Veterans Recollecting Traumatic Experiences

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Abstract

Background. Extensive research suggests post-traumatic stress disorder (PTSD) is associated with alterations in autobiographical memory (AM); however, the neural correlates of abnormal AM in this disorder remain to be identified. Moreover, little work has focused on identifying differences in patterns of neural activation between trauma-exposed populations who did and who did not go on to develop PTSD following trauma exposure. Notably, PTSD is characterized by contrasting patterns of emotional under and overmodulation, as observed in PTSD and its dissociative subtype (PTSD+DS), respectively. The purpose of the present study was to examine the role of key brain regions in AM retrieval among previously unstudied trauma-exposed military members and veterans with and without PTSD, and healthy controls. ***Methods***. Participants recalled an emotionally neutral, positive, negative, and traumatic/ most stressful memory under fMRI scanning. Clinical assessments indexed key variables, including dissociation, expected to be associated with altered patterns of neural activation during AM retrieval. ***Results***. The trauma-exposed military control (TEMC) group exhibited increased left frontal operculum (FO) activation during negative memory retrieval compared to baseline function. The TEMC group also displayed bilateral FO activation during negative compared to neutral memory retrieval. The PTSD group demonstrated increased left lingual activation during trauma memory retrieval compared to baseline function. Left lingual activation was also observed in the PTSD group compared to TEMC during traumatic memory retrieval. Increased symptoms of depersonalization and derealization were associated with activation in the right lateral orbital gyrus, right supramarginal

gyrus, left superior frontal gyrus, left precentral gyrus, bilateral lingual gyrus, and right middle cingulate gyrus in a combined PTSD and TEMC group during trauma memory retrieval. ***Discussion***. The left lingual activation observed in the PTSD group during traumatic memory retrieval is in keeping with the role of this region in implicitly cued, nonverbal memory retrieval. By contrast, FO activity observed in the TEMC during negative memory points towards the use of emotional regulation strategies to gate intrusive memory recall. Our finding of an association between frontal and posterior activation and dissociative symptoms during trauma memory retrieval provides not only further evidence of frontal involvement in the dissociative response but also points to posterior involvement in dissociative responses among individuals with military-related trauma exposure.

Introduction

Autobiographical memory (AM) consists of memories for past personal events and for factual information (Tulving, 1987) that together contribute to the development and maintenance of an enduring sense of self (Conway & Pleydell-Pearce, 2000). Numerous studies point towards AM disruption in neuropsychiatric illness, including among patients with schizophrenia (McLeod, Wood, & Brewin, 2006), major depressive disorder, and post-traumatic stress disorder (PTSD; Moore & Zoellner, 2007; Parlar, Densmore, Hall, Lanius, & McKinnon, 2018) where disruptions in AM are thought to be involved in the onset and maintenance of these conditions (Contractor, et al., 2018; Kleim, & Ehlers, 2008; Sumner, Griffith, & Mineka, 2010; McLeod, et al., 2006). In addition to its core affective components, memory disruption is central to PTSD, where symptoms of PTSD include difficulty recalling aspects of traumatic experience as well as the vivid re-experiencing of traumatic memories (APA, 2013).

Broadly, memory is thought to be encoded implicitly and explicitly. Whereas implicitly cued memory retrieval is guided by unconscious processes, explicitly cued memory retrieval is guided by conscious processes (Tulving, 1987). The explicit memory system is further divided into semantic and episodic components, where semantic memory involves recall for information related to general knowledge (e.g., I first deployed to Afghanistan) and episodic memory involves recall of information related to past personal events specific to time and place (e.g., I recall my shock when a mine exploded under our vehicle; Conway, 2003; Conway & Pleydell-Pearce, 2000; Tulving, 1987; Tulving, 2002). Autobiographical memory is considered a form of explicit memory comprised of both episodic and semantic components (Rubin, 2005).

Notably, disruptions in AM performance have been observed in PTSD. These disruptions include a pattern of "overgeneral" AM recall characterized by poor episodic recall of not only the index trauma (McNally, Lasko, Macklin, & Pitman, 1995; McNally, Litz, Prassas, Shin, & Weathers, 1994; Moradi, Abdi, Fathi-Ashtiani, Dalgleish, & Jobson, 2012) but also events unrelated to the trauma (Crane, et al., 2014; Graham, Herlihy, & Brewin, 2014; Kuyken, Howell, & Dalgleish, 2006; McNally, et al., 1995; Moore & Zoellner, 2007; Robinson & Jobson, 2013; Schönfeld, Ehlers, Böllinghaus, & Rief, 2007; Ono, Devilly, & Shum, 2016; Verfaellie & Vasterling 2009). Although the majority of studies point towards this pattern of fragmented or impoverished AM in PTSD, a smaller number of studies reveal heightened AM recall among individuals with PTSD (Ashbaugh, Marinos, & Bujaki, 2018; Moradi, et al., 2008; Ogle, et al., 2008; Ogle, et al., 2013), particularly among individuals who have suffered single-blow as opposed to repeated developmental trauma, with some studies unable to report a relation between PTSD and OGM (Kleim, Griffith, Gäbler, Schützwohl, & Maercker, 2013; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001). Despite this lack of consensus in the literature surrounding AM performance in PTSD, recent meta-analytic work has identified a strong effect size for AM dysfunction in PTSD (*Hedges' g* = 0.90-1.91; Ono, et al., 2016).

Related work has sought to identify two contrasting patterns of emotion modulation in PTSD and its dissociative subtype (PTSD+DS) where the ability to gate emotional responses and to regulate autobiographical memory retrieval may rely, in part, on emotional regulation strategies (Lanius, et al., 2010; Lanius, et al., 2002; Sartory, et al., 2013). Here, approximately 15-30% of individuals with PTSD report experiencing dissociative symptoms of depersonalization (feeling outside of, or as if you do not belong to your own body) and derealization (feeling as though things around you are strange or unfamiliar), thus presenting with the recently identified dissociative subtype of PTSD (Armour, Karstoft, & Richardson, 2014; Bennett, Modrowski, Kerig, & Chaplo, 2015; Hansen, Ross, & Armour, 2017; Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012; Stein et al., 2013; Wolf, Miller, et al., 2012). In contrast to PTSD patients without this dissociative subtype, patients with PTSD+DS present with increased symptom severity, heightened symptoms of depression and alcohol abuse, and increased psychiatric comorbidity across both military and civilian samples (Bennett et al., 2015; Blevins, Weathers, & Witte, 2014; Tsai, Armour, Southwick, & Pietrzak, 2015; Waelde, Silvern, & Fairbank, 2005; Wolf, Lunney, et al., 2012; Wolf, Miller, et al., 2012). Indeed, dissociative symptoms have emerged as a key predictor of impaired cognitive functioning in patients with PTSD (Boyd, et al., 2018; Fani, et al., 2018; McKinnon et al., 2016) and with depression (Parlar, Frewen, Oremus, Lanius, & McKinnon, 2016), and contribute significantly to impaired functioning (e.g., work, family, social) among individuals with PTSD (Boyd, et al., 2018; McKinnon, et al., 2016). These findings are in keeping with previous work showing an association between with decrements in working memory, attention, verbal memory, and executive functioning in PTSD (Kaplow, Hall, Koenen, Dodge, & Amaya-Jackson, 2008; Minshew & D’Andrea, 2015; Morgan, Doran, Steffian, Hazlett, & Southwick, 2006; Rivera-Vélez, González-Viruet, Martínez-Taboas, & Pérez-Mojica, 2014; Roca, Hart, Kimbrell, & Freeman, 2006; Twamley et al., 2009).

Interestingly, dissociative symptoms have also been linked to aberrant AM function in both clinical and general populations. For example, dissociative symptomology in groups with trauma exposure, PTSD, and borderline personality disorder have been linked to altered AM function, including overgeneral recall (Jones, et al., 1999) altered prefrontal and limbic activation (Lanius, et al., 2002), and inaccurate event recall (Chae, Goodman, Eisen, & Qin, 2011). Studies of healthy populations support these findings where individuals with increased dissociative experiences in daily life or induced dissociative states demonstrate decreased recall for events and trauma-related stimuli, respectively (Bergouignan, Nyberg, & Ehrsson, 2014; Olsen, & Beck, 2012). Despite knowledge of the relation between impairments in cognitive functioning, AM, and the presence of dissociative symptoms in individuals with PTSD, little work has sought to explore the neural correlates of AM in patients with dissociative symptoms.

Critically, converse patterns of emotional modulation have been described in individuals with and without the dissociative subtype of PTSD. These patterns of emotional over and undermodulation are thought to arise from distinct neural systems. Specifically, individuals with PTSD+DS display a pattern of top-down emotional overmodulation, associated with excessive corticolimbic inhibition (Lanius, et al., 2010). Here, emotional overmodulation is associated with abnormally high levels of activation in neural regions associated with the modulation of emotion and arousal, including the medial prefrontal cortex (PFC) and dorsal anterior cingulate cortex (ACC). This pattern of hyperactivation appears to result in decreased activation of limbic regions, most notably the amygdala (Lanius, et al., 2010). Indeed, this distinctive pattern of neural activity has been associated strongly with the hypoarousal and emotional detachment associated with depersonalization and derealization symptoms observed in PTSD+DS (Flemingham, et al., 2008; McKinnon, et al., 2016; Nicholson, et al., 2015; Nicholson, et al., 2017).

By contrast, the approximately 70% of individuals with a non-dissociative presentation of PTSD exhibit an altered pattern of bottom-up emotional modulation characterized by emotional undermodulation (Lanius, et al., 2010). This pattern of undermodulation is thought to result from a contrasting failure of corticolimbic inhibition. Here, medial anterior regions involved in emotion and arousal modulation display abnormally low activation, including prominently the ventromedial PFC and the rostral ACC (Lanius, et al., 2010). This decrease in corticolimbic regulation is associated with increased limbic activity, a pattern most prominent in the amygdala (Lanius, et al., 2010). Notably, this pattern of activation is thought to give rise to the hyperarousal and re-experiencing symptoms observed commonly in individuals with a non-dissociative presentation of PTSD (Harricharan, et al., in press; Lanius, et al., 2010; Lanius, et al., 2003; Nicholson, et al., 2017; Weston, 2014).

In the present study, we sought to examine the functional neuroanatomy of AM retrieval in trauma-exposed military members with and without PTSD, and healthy controls. Notably, veterans with PTSD exhibit increased disability (Goldberg, et al., 2014) and functional impairment, and a poorer quality of life (Shea, et al., 2010; Zatzick, et al., 1997), compared to veterans without PTSD. Moreover, a study involving trauma-exposed men revealed that those who identified combat as compared to other traumas (e.g. natural disaster) as their worst life event were more likely to have ongoing PTSD symptoms, meet lifetime criteria for PTSD, and experience unemployment, with the exception of those men who identified sexual assault or molestation as their index trauma (Prigerson, Maciejewski, & Rosenheck, 2001). Taken together, these findings highlight the increased illness burden observed in military members and veterans as compared to civilians. Accordingly, the present study sought to identify not only the neural correlates of AM retrieval among individuals with dissociative symptoms of PTSD, but also to address the increased acuity of PTSD in military populations and further discern the effects of military trauma on AM retrieval.

An additional aim of the present study was to conduct a preliminary analysis of differences in patterns of neural activation between trauma-exposed military members and veterans with and without PTSD, and healthy controls. Although meta-analytic work suggests that AM retrieval in healthy individuals includes a core network of left-lateralized regions including medial and ventrolateral prefrontal, medial and lateral temporal and retrosplenial/posterior cingulate cortices, the temporoparietal junction, and the cerebellum (Svoboda, McKinnon, & Levine, 2006), less is known about the neural corelates of traumatic memory recall among trauma-exposed individuals with and without PTSD. A recent meta-analysis conducted by Sartory and colleagues (2013) identified cortical midline structures as central to altered traumatic AM function in PTSD. Specifically, these authors reported that compared to controls, patients with PTSD exhibit increased activation in the midline retrosplenial cortex, precuneus, ACC, and left angular gyrus following symptom provocation aimed at eliciting autobiographical recall (Sartory, et al., 2013). Critically, these same neural regions are thought central to self-referential processing and AM function (Cavanna, & Trimble, 2006; Menon, 2011; Sartory, et al., 2013; Sestieri, Corbetta, Romani, & Shulman, 2011; Spreng, Mar, & Kim, 2009; Svoboda, et al., 2006); this pattern of activation is thus consistent with the elevated level of re-experiencing associated with intrusive recall of traumatic memories observed in some patients with PTSD (Sartory, et al., 2013).

Additional studies provide further insight into the mechanisms underlying AM recall in patients with PTSD. For example, Lanius and colleagues (2004) reported increased activity between the right anterior cingulate gyrus and the right occipital lobe, parietal lobe, posterior cingulate gyrus, and caudate during traumatic memory recall in PTSD as compared to controls. Critically, these posterior cingulate, occipital, and right parietal regions are thought to underlie, in part, nonverbal episodic retrieval (Cabeza & Nyberg, 1997; Cabeza, & Nyberg, 2000) which may be central to AM in PTSD (e.g. upon a traumatic reminder, I re-experienced the same painful sensation in my body as I experienced during the assault); these authors suggest that individuals in the PTSD group may have recalled their traumatic memories as flashbacks that were primarily image-based (Lanius, et al., 2004). Whalley et al. (2013) provide corroborative evidence for this hypothesis, demonstrating a link between occipital function and flashback recall in PTSD compared to controls, further implicating posterior regions in aberrant AM function in PTSD. Notably, patterns of decreased or absent activation of medial prefrontal areas are commonly reported in PTSD (Hopper, Frewen, van der Kolk, & Lanius, 2007). For example, Lanius and colleagues (2001) reported decreased medial frontal cortex activation in PTSD compared to controls during traumatic memory retrieval, a finding that may relate to high levels of arousal experienced in the recall of emotional material. Taken together, these findings suggest that recall of traumatic AM in PTSD is associated with not only high levels of emotional arousal but also the re-experiencing of bodily sensations and visual images that may or may not be able to be verbally described.

Notably, their meta-analytic work, Sartory and colleagues (2013) also investigated between-subject comparisons of trauma-exposed individuals with and without PTSD. As compared to patients with PTSD, trauma-exposed controls without PTSD exhibited increased activation of lateral and dorsal sensory association areas, perhaps indicating increased attention to stimulus material in the trauma-exposed control group (Sartory, et al., 2013). Sartory and colleagues (2013) hypothesize further that self-referential processing induced through symptom provocation paradigms may uniquely inhibit the ability of trauma-exposed individuals with PTSD to process concomitant stimuli; this hypothesis remains to be verified. In a similar vein, Hayes and colleagues (2011) found that patients with PTSD relied more heavily on gist-based cues than on contextual details to identify trauma scenes they had previously been exposed to, resulting in increased false alarm rates compared to trauma-exposed controls.

In addition to regions reported in this meta-analytic review, additional research has identified further that the thalamus, anterior cingulate, and insula may play a key role in AM in PTSD. For example, decreased thalamic activation in traumatized subjects with compared to without PTSD has been observed in studies examining traumatic and negatively valenced memory retrieval, a pattern thought to result from high levels of arousal during recall of emotional material (Lanius, et al., 2001; Lanius, et al., 2003). These patterns of abnormal thalamic activity may indicate further a disruption in the relay of sensory information to the frontal cortex, cingulate gyrus, amygdala, and hippocampus (Lanius, et al., 2001), a pattern that may contribute to dissociative and/or flashback-like recall (Lanius, et al., 2001). Notably, a decrease or failure in ACC activation is common in PTSD neuroimaging literature which uses script-driven imagery to provoke symptoms (Hopper, et al., 2007), where this region is critical to experiential aspects of emotion, including its autonomic, neuroendocrine, and behavioural expression (Lanius, et al., 2003). Accordingly, Lanius and colleagues (2003) suggested that decreased activation of the thalamus and ACC during the recall of negatively valenced emotional memories in patients with PTSD as compared to controls contributes significantly to the emotional dysregulation observed in the disorder.

Interestingly, involuntary vivid recollection (i.e., flashbacks) in PTSD has been associated further with alterations in insular function, a region commonly activated in close synchrony with the limbic system (Karama, Armony, & Beauregard, 2011). Here, the insula is involved heavily in processing emotional feeling states through monitoring and identification of internal changes within the body, including their emotional implications (Craig, 2002; Harricharan, et al., in press). In keeping with this hypothesis, one study examining online traumatic memory recall in PTSD found that high levels of re-experiencing symptoms correlated positively with right anterior insular activity

(Hopper, et al., 2007). Furthermore, the same study demonstrated that state dissociation during traumatic memory recall correlated negatively with right anterior insula activity, suggesting a neural substrate for the emotional underengagement observed during dissociative responses (Hopper, et al., 2007). Finally, right insular activity was correlated positively with flashback as compared to episodic components of the same traumatic memory in patients with PTSD as compared to their comparator groups (Whalley, et al., 2013). Taken together, these findings suggest that insular activation may underlie, in part, involuntary flashback in PTSD, a role consistent with its primary function in regulating emotional and body state responses (Whalley, et al., 2013).

The current study sought to examine the functional neuroanatomy of AM retrieval in neutral, positive, negative, and traumatic/most stressful memories in a military group with PTSD, a trauma-exposed military control group (TEMC), and a non-military healthy control group (HC). As discussed above, AM dysfunction has been observed during recollection of traumatic and negatively valenced AMs. Although recollection of positive AMs is not commonly addressed in the PTSD literature, some authors suggest that the salience of trauma memories and related negative affect/cognitions may reciprocally inhibit positive memory recall and related positive affect/cognitions (Brewin, Dalgleish, & Joseph, 1996; Brewin, Gregory, Lipton, & Burgess, 2010; Brewin & Holms, 2003). Indeed, an imaging study examining positive AM retrieval in PTSD revealed greater recruitment of the amygdala/hippocampus during construction of negative as compared to positive AM retrieval and greater recruitment of the ventral medial prefrontal cortex during negative compared to positive AM retrieval as compared to controls (St. Jacques, Botzung, Miles, & Rubin, 2011). These authors suggest that symptoms of hypervigilance may result in increased reactivity during initial recruitment of the memory network specific to memories of negative valence (St. Jacques et. al., 2011). In keeping with AM findings in PTSD discussed thus far, we hypothesized overall that recall of emotional events (traumatic, negative, and possibly positive) would result in a reciprocal pattern of increased brain activity in regions associated with emotional processing and decreased brain activity in regions associated with cognitive processing in PTSD compared to both control groups.

In addition, the current study sought to determine whether key clinical variables were associated with patterns of neural activation underlying AM retrieval in these groups. Specifically, we included measures of dissociation, childhood trauma, and emotion regulation to investigate their relations with AM retrieval. As noted above, dissociative symptoms show a clear association with poor cognitive function, including alterations in AM performance. In keeping with this evidence, and established patterns of corticolimbic overregulation in the dissociative subtype of PTSD, we predicted that dissociative symptoms would be correlated positively with heightened activity in frontal regions (e.g. medial PFC, dorsal ACC) associated with emotional overmodulation and correlated negatively with limbic activity in regions (e.g. insula, amygdala) associated with undermodulation across memory types.

An examination of childhood trauma in a military population is especially pertinent as exposure to childhood trauma is more likely to occur in military compared to civilian population (Affifi, et al., 2006) and is associated with an increased perceived need for and a higher use of mental healthcare (Turner, et al., 2017), reduced health-related quality of life (Katon, et al., 2015), and suicide and suicide attempts (Affifi, et al., 2006). Critically, exposure to early life adversity and childhood trauma has been shown to alter both the neural and behavioural correlates of AM retrieval. Here, exposure to childhood trauma has been linked repeatedly to overgeneral AM retrieval (de Decker, Hermans, Raes, & Eelen, 2003; Henderson, Hargreaves, Gregory, & Williams, 2002; Meesters, Merckelbach, Muris, & Wessel, 2000). Little work, however, has sought to determine whether childhood trauma affects the neural correlates of AM function in PTSD. In one study, exposure to childhood physical neglect was negatively correlated with dorsal ACC activation during negative memory retrieval in patients with depression and a history of trauma exposure, a region involved in emotion regulation (Parlar, et al., 2018). In line with these findings, we predicted that childhood trauma in PTSD would be correlated negatively with activation in regions associated with emotion regulation (e.g. dorsal ACC, medial PFC) across memory types.

Finally, our research examines the relation between emotion regulation and the neural correlates of AM function in PTSD. Interestingly, emotion regulation in military populations appears to be uniquely dysfunctional. For example, one study found that military members with PTSD have been shown to exhibit decreased dorsolateral PFC recruitment compared to trauma-exposed military controls during cognitive appraisal, suggesting altered emotional regulation of negative affective states (Rabinak, et al., 2014). Weiss et. al., (Weiss, Williams, & Connolly, 2015) also report that emotion dysregulation emerged as a significant mediator of negative affect and the urge to engage in risky behaviours in veterans seeking treatment for substance abuse. Additionally, altered emotion regulation in PTSD in general has been noted in the non-dissociative and dissociative subtype of PTSD (Ehring & Quack, 2010; Lanius, et al., 2010; Nicholson, et al., 2017; Powers, Cross, Fani, & Bradley, 2015; Rabinak, et al., 2014; Seligowski, Lee, Bardeen, & Orcutt, 2015). Thus, we predicted that increased levels of emotion dysregulation would correlate negatively with neural activation in regions involved in emotion regulation (e.g. dorsal ACC and medial PFC) across all memory types.

Methods

Participants

Thirteen military members and veterans between 24 and 59 years of age with a confirmed diagnosis of PTSD (DSM-IV diagnostic criteria using the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997) and Clinician Administered PTSD Scale (CAPS; Blake, et al., 1995) with a score >50) were included in the military PTSD group. The TEMC group was comprised of nine military members and veterans between 27 and 54 years of age. Participants in the TEMC control did not have a current or past diagnosis of PTSD, as confirmed by the SCID-I. Military members and veterans with a lifetime history of bipolar or psychotic disorder, current or past Axis II disorder, current alcohol/substance abuse or dependence within the last 6 months, history of pain disorder, history of obsessive-compulsive disorder, and/or a history of electroconvulsive therapy or trauma-focused psychotherapy (e.g. exposure therapy, trauma EMDR) were excluded. The HC group consisted of 14 individuals between 18 and 39 years of age. HCs with a current or past Axis-I or Axis-II disorder(s) were excluded.

Any participants with implants, etc. that do not comply with 3T fMRI safety standards; history of head injury, any loss of consciousness (or a Glasgow Coma Scale Score < 15 at the time of incident assessed retrospectively by participant); significant untreated medical illness; history of neurological disorder; or history of any pervasive developmental disorder were excluded.

Participants were recruited through advertisements and posters from the general community in London, ON, from within the Department of Psychiatry at the University of Western Ontario (UWO) at the London Health Sciences Centre (LHSC; i.e., patients of UWO-LHSC), from family physicians, from mental health professionals, psychology/psychiatry clinics (including the Student Development Centre, UWO), and from community programs for traumatic-stress survivors. Participants were also recruited from Canadian Forces, US military, and Veterans Affairs healthcare systems in Canada and USA. This study was approved by and complied with the local research ethics board. All participants provided written and informed consent.

Materials

Clinical measures

Axis I diagnoses were assessed with the full SCID-I (First, et al., 1997). PTSD symptomology was further assessed with the CAPS (Blake, et al., 1995), which includes assessments of symptom severity and current (past month) and lifetime PTSD diagnostic status. Depression and anxiety severity were assessed using the Beck Depression

Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and the Beck Anxiety Inventory (BAI; Beck & Steer, 1993) respectively. Dissociation was examined using the Multiscale Dissociation Inventory (MDI; Brier, 2002). The MDI is a self report questionnaire comprised of 30 items that assess dissociative responses. This study focused on MDI subscales of depersonalization and derealization as they compose the basis of PTSD+DS. A history of childhood trauma was determined using the Childhood Trauma Questionnaire (CTQ; Bernstein, et al., 2003). The CTQ is a 28-item self report questionnaire that assesses emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Emotion regulation was assessed with the Difficulties in Emotion Regulation Scale (DERS; Gratz, & Roemer, 2004). The DERS is a 36-item self report questionnaire with 6 subscales: 1) emotional awareness, 2) non-acceptance of emotional responses, 3) goal directed behaviour, 4) impulse control, 5) emotion regulation strategies, and 6) emotional clarity. The Repeatable Battery for Assessment of Neuropsychological Status (RBANS; Randolph, 1998) was used to assess global neuropsychological functioning. The RBANS includes 12 subsets which culminate in 5 index scores and a total score: 1) immediate memory, 2) delayed memory, 3) visuospatial/constructional, 4) language, and 5) attention. The National Adult Reading Test (NART; Nelson, 1982) was used to test premorbid intellectual functioning. The NART includes a list of 50 words that do not comply with common rules of pronunciation to be read aloud by the participant. Scores on the NART provide indices of verbal IQ, performance IQ, and full-scale IQ. All participants completed each of these measures with the following exceptions: one TEMC did not complete the MDI or the

DERS; four HCs did not complete the RBANS; one TEMC did not complete the RBANS or NART.

Pre-scan interview

Following completion of the clinical assessments, participants completed a pre-scan interview to identify an emotionally neutral, positive, negative and traumatic/ most stressful AM that would be recalled during the fMRI scan. In lieu of a traumatic memory, HCs were instructed to recall a highly negative or most stressful event. Each memory identified needed to represent an event specific in time and place participants were personally involved in, and recall being personally involved in.

Following the identification of the events, each event was given a brief title. Participants were instructed to segment each event into five chronological parts that were subsequently formatted as brief titles to serve as memory retrieval cues in the fMRI scanner (e.g. event title: “Injury of a fellow soldier”: i) hearing gun shots; ii) running to that area; iii) firing gun; iv) comrade shot; v) evacuation). To avoid rehearsal and re-encoding effects, no further details of the memories were discussed in the interview or leading up to the MRI scan, which was performed no more than a week after the clinical assessments and pre-scan interview.

fMRI scan

Event titles created in the pre-scan interview were used to cue retrieval during the fMRI scan. E-Prime software (https://www.pstnet.com/eprime.cfm) was used to program and present visual stimuli in the fMRI scan; stimuli were presented as black text on a grey screen.

Participants were cued to recall a memory with a 7.5 s presentation of the event title (e.g. “Injury of a fellow soldier”). Segments of the event were then presented for 15 s each (e.g. “hearing gun shots”). Participants were instructed to recall thoughts, feelings, physical sensations, sounds, and visual images associated with each segment. Each 15 s memory segment presentation was followed by a subjective rating consisting of: i) a 10 s rating interval where subjects rated the degree of autobiographical re-experiencing they experienced during retrieval and; ii) a 10 s rating interval where subjects rated the degree of emotional change they experienced during retrieval. Both subjective ratings were rated on a Likert type scale from 1 to 7, where 1 represented no re-experiencing/emotional change and 7 represented the highest degree thereof. A 10 s fixation block followed the subjective rating.

Following the 5 segments of an event and the associated subjective ratings, participants completed an odd number detection task which been shown to reduce medial temporal activation (Stark & Squire, 2001). In this task, single digit numbers were presented in quick succession (1.25 s duration, 0.25 s ISI) and participants were instructed to indicate through the press of a button whether the number was odd or even. Participants were cued with a 2.5 s introduction to the task, followed by five 25 s intervals of the task. A 15 s fixation block preceded the odd number detection task. A 20 s fixation block followed the odd number detection task. Stimuli were presented in a blocked design, with one run per memory, each memory including 5 stimuli corresponding to 5 previously identified memory segments. The blocks were presented in random order with the exception of the last block: the traumatic/ most stressful memory was always presented last to avoid any carry-over effects into non-traumatic/ most stressful memory retrieval.

fMRI data acquisition

A 3.0 T scanner (Magnetom Tim Trio, Siemens Medical Solutions, Erlangen, Germany) with a 32-channel phased array head coil was used to collect whole brain functional images. Functional images were collected using an interleaved gradient-echo planar imaging pulse sequence (single-shot, blipped-EPI, 3D PACE) with TR = 2500 ms, TE = 20 ms, voxel size = 2 × 2 × 2 mm3, Field of view (FOV) = 192 × 192 × 112 mm3 (96 × 96 matrix, 56 contiguous slices), and Flip Angle (FA) = 90°. This study also collected high-resolution T1-weighted anatomical images (MPRage: 192 slices, voxel size = 1 × 1 × 1 mm3).

Data processing and statistical analysis

Neuroimaging data

Images were processed (motion correction, spatial normalization, and smoothing) using standard procedures in SPM 12 (https://www.fil.ion.ucl.ac.uk/spm/software/spm12/) in MATLAB 8.4 (MathWorks Inc.). Mean functional volumes were created once individual functional images were realigned with the first volume in each session to correct for motion and resliced. The images were then co-registered to the imaging template found in SPM 12 (standard echo-planar template in MNI space). A deformation matrix was created and then applied to all functional volumes which were subsequently smoothed using a full-width half-maximum gaussian filter of 6 mm.

Before first-level statistics were carried out, single-subject global signal artifacts were controlled for using Artifact Detect Tools software (ART: https://www.nitrc.org/projects/artifact_detect/). Additional to the six regressors from the realignment step, this software identifies outliers in the functional data for each participant to be included as covariates in the first level general linear model (GLM). These analyses were carried out for the following blocks: neutral memory blocks, positive memory blocks, negative memory blocks, trauma/most stressful memory blocks, and odd number detection task blocks. The following contrasts were carried out: i) neutral vs. fixation, ii) positive vs. fixation, iii) negative vs. fixation, iv) trauma/most stressful vs. fixation, v) positive > neutral vs. fixation, vi) neg > neutral vs. fixation, vii) trauma > neutral vs. fixation. Second level analyses were completed by running a 3 x 4 ANOVA and then examining the following contrasts: within group and within memory, within group and between memory, between group and within memory, between group and between memory.

Region of interest (ROI) analyses were completed on ROIs identified using the Talairach Daemon atlas in WFU PickAtlas (https://www.nitrc.org/projects/wfu_pickatlas/) thresholded at $p \leq .05$ (FWE-corrected, peak level) with a 10 mm radius sphere from initial whole brain analyses thresholded at $p \leq .001$, k=10. A single ROI was used in this study, centered on the left lingual gyrus.

Subjective Ratings

For each participant, within each memory type, ratings of re-experiencing given after each memory segment were averaged to create a singular rating of re-experiencing for each memory type. The same method was applied to ratings of emotional change, resulting in a single, averaged rating of emotional change for each memory type. Multiple regression analyses via second-level analyses were used to examine the relation between subjective ratings given in the scanner regarding re-experiencing and emotional change during retrieval and brain activity in each condition for all groups. A one-way ANOVA was used to determine group differences among ratings. Where group differences were found to violate the assumption of homogeneity, a Welch's correction was used. Bonferroni corrections were also used to correct for multiple comparisons.

Clinical variables

Multiple regression analyses via second-level analyses were used to examine the relation between scores on the MDI, CTQ, and DERS and brain activity in each condition for all groups. A one-way ANOVA was used to determine group differences among scores on the MDI, CTQ, and DERS. Where groups were found to violate the assumption of homogeneity, a Welch's correction was used. Bonferroni corrections were also used to correct for multiple comparisons.

Results

Demographic data

Refer to Table 1 for demographic data and scores on clinical measures. The HC, PTSD, and TEMC groups did not differ in sex distribution or premorbid intellectual function. The groups did differ significantly in age ($p < .00$). Generally, groups did not differ in current global neuropsychological function, with the exception the visuospatial/constructional item of the RBANS where a significant difference between groups emerged ($p = .04$). Among the TEMC group, one met criteria for a history of MDD and was currently in remission; given the difficulty of recruitment in this group, this participant was included in analysis.

Clinical variables

As expected, the groups significantly differed in CTQ total scores ($p = .04$) where a Bonferroni correction revealed significantly higher CTQ total scores in the PTSD group compared to HCs ($p = .01$). Additionally, a significant difference was found between groups on the physical neglect item of the CTQ ($p = .03$) where a Bonferroni correction revealed significantly higher scores for physical neglect in the PTSD group compared to HCs ($p = .01$).

Groups also significantly differed on DERS total scores ($p < .00$) where a Bonferroni correction revealed that the PTSD group had significantly higher total scores than the TEMC ($p < .00$) and HC ($p < .00$) group. Specifically, groups significantly differed on the following five items of the DERS: 1) non-acceptance of emotional responses ($p < .00$) where a Bonferroni correction revealed significantly higher scores of non-acceptance in the PTSD group compared to HCs ($p < .00$); 2) goal directed behaviour ($p = .01$) where a Bonferroni correction revealed significantly higher scores in the PTSD group compared to the TEMC group ($p = . 01$); 3) emotional awareness ($p < .00$) where a Bonferroni correction revealed significantly higher scores in the PTSD group compared to HCs ($p = .02$); 4) emotion regulation strategies ($p < .00$) where Bonferroni correction showed significantly higher scores in the PTSD group compared to HCs ($p = < .00$); and

5) emotional clarity ($p < .00$) where a Bonferroni correction revealed that the PTSD group had significantly higher scores than both the TEMC ($p < .00$) and HC ($p < .00$) group.

As expected, participants also differed significantly on total MDI scores ($p = .01$) with a Bonferroni comparison reporting significantly higher scores in the PTSD group compared to HCs ($p < .00$). Specifically, groups differed on the following four items of the MDI: 1) disengagement ($p = .01$) where a Bonferroni correction showed that the PTSD group had significantly higher scores than HCs ($p = .01$); 2) derealization ($p = .02$) where a Bonferroni correction showed significantly higher scores in PTSD group compared to HCs ($p = .01$); 3) emotional constriction ($p < .00$) where a Bonferroni correction revealed significantly higher scores in the PTSD group compared to HCs ($p < .00$); and 4) memory lapse ($p = .02$) where a Bonferroni correction showed significantly higher scores in the PTSD group compared to HCs ($p = .02$).

Subjective ratings

Groups differed significantly in reported emotional change during neutral memory retrieval ($p = .01$) where a Bonferroni correction revealed that the PTSD group reported increased emotional change compared to HCs ($p = .01$).

Neuroimaging results

Among the TEMC group, participants showed increased left frontal operculum (FO) activation during negative memory retrieval as compared to baseline condition ($t = 5.63$, p-FWE $< .00$) (Fig. 1). Furthermore, increased bilateral FO activation was found in negative memory compared to neutral memory retrieval in the TEMC group (left: $t =$

5.42, p-FWE = .01; right: t = 5.40, p-FWE = .01) (Fig. 1). No other differences in patterns of activation were identified.

ROI analysis revealed left lingual activation during traumatic memory retrieval as compared to baseline condition in the PTSD group (t = 3.82, p-FWE = .04) (Fig. 2). Heightened left lingual activation was also observed in the PTSD group as compared to TEMC group during traumatic memory retrieval (t = 4.06, p-FWE = .02) (Fig. 2).

No other regions differences in activation were identified through second-level contrasts.

Correlations between hemodynamic activity and subjective ratings

Ratings of re-experiencing or emotional change did not correlate with patterns of neural activity across study conditions.

Correlations between hemodynamic activity and clinical variables

Given the relatively small sample size, and the heterogeneity of military-related trauma exposure among these groups, the TEMC and PTSD groups were combined for correlation analyses concerning clinical variables, creating a trauma-exposed military group.

Dissociation

The depersonalization and derealization indices of the MDI were collapsed for analyses, creating a general index of dissociation consistent with DSM-5 diagnostic criteria for PTSD+DS. Several brain regions were correlated positively with trauma memory retrieval in the trauma-exposed group and dissociative behaviour (Fig. 3): right lateral orbital gyrus (t = 7.08, p-FWE = .033), right supramarginal gyrus (t = 6.89, p-FWE

= .044), left superior frontal gyrus (t = 7.06, p-FWE = .034), left precentral gyrus (t = 6.85, p-FWE = .047), right lingual gyrus (t = 6.98, p-FWE = .038), and right middle cingulate gyrus (t = 6.95, p-FWE = .04). The left lingual gyrus (t = 5.15, p-FWE = .015) was also identified through ROI analysis (Fig. 3).

Childhood trauma and emotion regulation

Correlation analysis did not reveal an association between patterns of neural activation and responses on the CTQ or DERS across study conditions.

Discussion

The main objective of this study was to examine the neural correlates of emotional memory retrieval in a military-related PTSD population compared to a trauma-exposed military population and healthy controls. We predicted that, relative to trauma-exposed military members and veterans without PTSD and healthy controls, military members and veterans with PTSD would show decreased activity in areas related to cognitive functioning and increased activity in areas related to emotional processing during recall of positive, negative, neutral and traumatic memories. The findings of this study modestly supported our initial hypotheses. Specifically, we found increased left lingual activation in the PTSD group during trauma memory retrieval as compared to: i) baseline function and ii) TEMC trauma memory retrieval. Due to lingual involvement in implicitly cued attentional and memory processes, we posit that the lingual activation observed here during trauma memory retrieval in the PTSD group is consistent with the flashback-like recall observed in this disorder, pointing towards a potential neural substrate of this implicitly cued memory recall in military PTSD. In addition, we found increased FO activation in the TEMC group during negative compared to neural memory retrieval and baseline function. Given the role of the FO in conceptual processing and in the integration of information, these findings point towards the utilization of cognitive regulatory strategies in the TECM group during strategically guided recollection of negative AMs, with the additional benefit of dampening down emotionally driven responses (e.g., flashbacks).

The secondary objective of our study was to examine the relation between childhood trauma, dissociative symptoms, and emotion dysregulation and the neural correlates of emotional AM retrieval in a military-related PTSD group. Although we failed to identify a relation between childhood trauma or emotion dysregulation and the neural correlates of AM retrieval in this trauma-exposed military population, we did find a clear association between dissociative symptoms and patterns of neural activation during traumatic memory retrieval in the combined trauma-exposed military sample. Here, we found right lateral orbital, right supramarginal, left superior frontal, left precentral, bilateral lingual, and right middle cingulate activation correlated with dissociative symptoms during traumatic memory retrieval in the trauma-exposed military group. To the best of our knowledge, these findings are the first to demonstrate an association between activation of these frontal and posterior regions and trauma AM retrieval in a trauma-exposed military population. We discuss these findings in greater detail below.

Our work further corroborates previous research that shows increased rates of childhood trauma in military-related PTSD as compared to controls. Interestingly, our results also demonstrated that military members with PTSD self-reported higher levels of emotional change during neutral memory retrieval than HCs, suggesting that ostensibly "neutral" memories may convey emotion/ be arousing in this disorder. As previously discussed, non-dissociative PTSD-is characterized by emotion undermodulation where hyperactive limbic structures (e.g. amygdala) and hypoactive emotion regulation areas (e.g. medial PFC) are hypothesized to give rise to, in part, symptoms of hyperarousal (Lanius, et al., 2010; Liberzon & Martis, 2006), a pattern consistent with the symptom profile of this disorder (Amstadter & Vernon, 2008; Boden, et al., 2013; New, et al., 2009; Seligowski, et al., 2015; Shepherd & Wild, 2014; Tull, Barrett, McMillan, & Roemer, 2007) including meta-analytic work reporting large effects sizes for general emotion dysregulation, rumination, thought suppression, and experiential avoidance in relation to post-traumatic stress symptoms (Seligowski, et al., 2015). Taken together, these symptoms of hyperarousal and emotion dysregulation may underlie, in part, the greater degree of emotional change experienced by the PTSD group during neutral memory retrieval. Individuals with PTSD also show altered responses to positive, negative and neutral material (e.g., a compliment is hurtful; a neutral memory may trigger recollection of another negative event), a pattern that may also account for the increased emotional change observed in the PTSD group during neutral memory recall. Although our imaging findings did not show a difference in neutral memory retrieval between the PTSD and HC groups, this difference in subjective ratings warrants further investigation. For example, future research should examine neutral emotions in PTSD to determine if hyperarousal, emotion dysregulation, or other factors (e.g. trauma type; interpretation) alter the experience of neutral emotion. These findings may also pertain to cognitive functioning in PTSD, where cognitive and emotional processes are inextricably intertwined (Hayes, VanElzakker, & Shin, 2012).

The lingual gyrus has been connected to attention and memory processes through implicitly cued networks. Attention has been established to play a significant role in memory retrieval and is posited to consist of two networks: 1) a dorsal frontoparietal network involved in the selection of stimuli driven by controlled processes such as a memory search, a monitoring strategy, or internal goals and; 2) a ventral frontoparietal network involved in the detection of pertinent stimuli and is related to memoires retrieved spontaneously, rapidly, and with a high degree of confidence (Burianová, Ciaramelli, Grady, & Moscovitch, 2012; Ciaramelli, Grady, Levine, Ween, & Moscovitch, 2010). Occipital areas, including the lingual gyrus, are associated with the ventral frontoparietal network, suggesting the regions are involved in less conscious processing compared to the dorsal counterpart (Ciaramelli, et al., 2010). Similarly, implicitly cued memory function represents unconscious processes (Roediger, 1990) that have been shown to also rely on ventral posterior regions (Vuilleumier, Schwartz, Duhoux, Dolan, & Driver, 2005). Considering the overlap between the ventral frontoparietal network of attention and implicitly cued memory function, it is possible that the observed lingual activation indicates unconscious processing that does not reflect effortful or controlled retrieval. Here, we argue that intact implicit function found in the PTSD group during trauma memory retrieval may indicate flashback-type retrieval where flashbacks are described as intrusive and occurring outside of an individual’s conscious control (McNally, 1997).

As noted above, FO activation observed in the TEMC group is likely reflective of enhanced regulation of negative affect in this group. Specifically, inferior frontal regions, including the FO, are hypothesized to play a role in conceptual processing and determining meaning, functions critical to appraisal processes (Pulvermüller, 2013; Karama, et al., 2011). Furthermore, in a study of source memory episodic retrieval, the left FO was strongly associated with the integration of information (Lundstrom, Ingvar, & Petersson, 2005). Given these suggested functions of the FO, it is possible participants in the TEMC group may have been engaging frontal regions during negative memory retrieval in an unconscious effort to regulate the retrieval of a negatively-valenced, emotionally arousing memory. This proposal of enhanced frontal regulation is supported by the well-established role of the frontal regions in emotion regulation (Green, & Malhi, 2006). As this finding is specific to the TEMC group, it may point further to compensatory brain function and/or behaviour in trauma-exposed military members/veterans who do not develop PTSD. However, the nature of such unique functioning remains inconclusive; further research is needed to understand the differences between those who have experienced trauma and develop PTSD versus those who do not.

Three frontal regions were identified in our correlation analysis examining the association between dissociative symptoms and trauma memory retrieval in a trauma-exposed military group, such as the right lateral orbital gyrus. Aberrant orbitofrontal cortex (OFC) activation has been reported in the PTSD literature regarding emotional responses (Bremner, et al., 2003; Liberzon, & Martis, 2006) and right OFC activation has been positively correlated with the suppression of sadness (Lévesque, et al., 2003). As the current orbital finding pertains to dissociative behaviour, it may reflect a significant association of this region with emotion regulation processes characteristic of excessive corticolimbic inhibition. Another frontal area identified in the current correlation analysis is the left superior frontal gyrus. Bilateral superior frontal gyrus activity has been shown to be negatively correlated with flashback intensity in PTSD, suggesting this region is not involved in high degrees of re-experiencing (Osuch, et al., 2001). Our results corroborate these findings as they link superior frontal activation with dissociative responses; dissociative symptomology is not associated with flashbacks due to heightened corticolimbic inhibition (Lanius, et al., 2010). The remaining frontal region revealed in our dissociation correlation analysis is the left prefrontal gyrus. Thorne and colleagues (2017) have demonstrated abnormal resting-state motor cortex connectivity indicative of aberrant motor readiness in PTSD. The authors discuss the possibility that sensorimotor activation may help to regulate the stress response in PTSD (Thorne, et al., 2017). Taken together with the current pattern of findings, the observed precentral gyrus activation may reflect the high levels of emotional regulation observed in the dissociative subtype. Further research is urgently needed to determine the association precentral gyrus activation with dissociation.

Parietal lobe function is also dysfunctional in PTSD. A review of emotional responding in PTSD reported an association between abnormal right parietal activation and emotion dysregulation in PTSD (Liberzon, & Martis, 2006), a pattern that aligns closely with research that implicates right parietal activation in the dissociative response (Lanius, et al., 2002).Notably, this review includes a study that demonstrated an association between right supramarginal activation and reappraisal processes serving emotion regulation during exposure to highly negative scenes (Ochsner, Bunge, Gross, & Gabrieli, 2002).

In the present study, right supramarginal activity correlated with dissociative behaviour during traumatic memory retrieval in the trauma-exposed group, perhaps indicating regulation typical to the dissociative response. Posterior activation was also reported in this analysis, as observed on the association of increased bilateral lingual activity with dissociative symptoms during trauma memory retrieval. Although the above discussion of the lingual gyrus emphasizes its role in bottom-up processes rather than the top-down pattern characteristic of dissociation, a study has linked left lingual activation with dissociative states in dissociative identity disorder (DID; Reinders, et al., 2014). The authors conducted this study to examine whether DID patients were similar to PTSD patients in their dichotomous emotional profiles; they were able to find support of their initial hypothesis wherein some DID patients exhibited symptoms of hypoarousal and some hyperarousal, depending if trauma identity states were induced (Reinders, et al., 2014). Reinders and colleagues (2014) caution against the application of their results to PTSD patients as it remains unclear the exact similarities and/or differences between dissociation in DID and PTSD. Currently, research may be inconclusive regarding the association of the lingual gyrus and dissociative symptoms.

The final region identified in our dissociation correlation analysis is the right middle cingulate cortex (MCC). Classically, the ACC is shown to be highly related to the dissociative response; it is a key agent of emotion modulation as it plays a critical role in arousal modulation and emotion regulation (Lanius, et al., 2002). The ACC has been described as a unique structure as it has connections to both the limbic system and prefrontal cortex, necessitating a role in both emotional and cognitive processes (Stevens, Hurley, & Taber, 2011). The MCC does not share the same characteristic as it has connections to cognitive areas (lateral PFC), motor areas (primary motor cortex), and pain- and motor-related thalamic nuclei (Stevens, et al., 2011). The current analysis shows a positive correlation between MCC activation and dissociative symptoms in a trauma-exposed population during trauma memory retrieval. The MCC may relate to dissociation through its connections to other areas associated with dissociation, such as the dorsolateral PFC (Lanius, et al., 2010) and the motor area discussed above. Furthermore, MCC activation has been shown to increase during the perception of a nonpainful stimuli as painful when pain was initially expected, demonstrating how expectation of a certain stimulus may influence neural activity (Ochsner, & Gross, 2005). This finding could also apply to an association between MCC and dissociative symptoms as dissociation represents a psychobiological response to a perceived threat (McKinnon, et al., 2016). MCC activation could potentially indicate the expectation of a threat and/or pain through trauma memory retrieval, which might be an indication of a failure of fear extinction seen in PTSD (Maren, Phan, & Liberzon, 2013). Further research would be required to confirm any proposed association and/or role regarding the MCC and dissociation. When considering the results of our correlation analysis between dissociative symptoms and traumatic memory recall in military-related trauma exposure, it must be noted that it remains unclear how military-related trauma exposure and PTSD are related to the results. Although the demographic data shows significantly increased dissociative symptoms specific to the PTSD group, no firm suggestion can be made regarding whether military-related trauma exposure and/ or PTSD is driving the response.

The results of the current study were in keeping with current evidence of altered AM function in PTSD. Moreover, our results suggest a unique correlate of aberrant AM function in a trauma-exposed military population with PTSD, pointing toward a role of altered implicit functioning in this group. These findings further affirm the inclusion of the trauma memory as treatment target in PTSD (e.g. Bisson & Martin, 2009). Importantly, our work expands on current research regarding implicit functioning in PTSD. The PTSD literature consistently describes an implicit bias for trauma-related information where those with PTSD demonstrate increased implicitly cued memory for trauma-related cues such images, sounds, and words compared to non trauma-related cues (Amir, Leiner, & Bomyea, 2010; Amir, McNally, & Wiegartz, 1996; McNally, 1997; Van der kolk, 1998; Zeitlin & McNally, 1991). Research posits that the observed phenomenon may contribute to the maintenance of PTSD symptomology wherein trauma cues are overrepresented in implicit systems rendering them easily accessible, possibly leading to the intrusive symptoms seen in the disorder (Buckley, Blanchard, & Neill, 2000; Van der kolk, 1998; Zeitlin & McNally, 1991). In keeping with related investigation, a recent review argues that implicit and automatic hypermnesia of fear memories plays a significant role in altered memory processes in PTSD, recommending exposure-based treatments that work to restore a normal representation of traumatic memories via reprocessing of the traumatic information (Desmedt, Marighetto, & Piazza, 2015).

Despite clear knowledge of altered implicitly cued memory function in PTSD, little work has sought to examine implicit function in personal, emotional AMs. Further research therein could provide key findings regarding the extent of implicit dysfunction in memory processes, potentially identifying mechanisms which affect altered self perceptions accompanying altered AM function in the disorder.

Our results also demonstrate key neural correlates for dissociative symptoms and their association with trauma memory retrieval in a military trauma exposed population. These findings address a pressing need to understand dissociative symptomology in military-related trauma exposure as both are established as factors related to a higher illness burden in PTSD, as noted above. To date, little work has addressed dissociative symptomology in trauma-exposed military populations specific to AM function. Our results strongly suggest an association between dissociation and AM function in a trauma-exposed military population, suggesting dissociation as a therapeutic target in the treatment of PTSD. Research has shown that the presence of dissociative symptoms affects treatment response, leading to differential response to treatments such as eye movement desensitization and reprocessing and cognitive processing therapy (Bae, Kim, & Park, 2016; Resick, Suvak, Johnides, Mitchell, & Iverson, 2012). However, these findings are not specific to a military population; future research should endeavour to examine the effect of dissociative symptoms on treatment in a trauma-exposed military population as their presentation of PTSD has been demonstrated to have an increased severity.

The current study suffers from limitations, including concerns regarding the samples included. Limited sample sizes may have rendered the analysis weak, resulting in a study design with low power. Future research should complete appropriate power analysis and endeavour to obtain the appropriate data. Furthermore, the sample of trauma-exposed military members with PTSD did not separate between those with the non-dissociative and dissociative subtype of PTSD. Going forward, research should include these two separate groups to provide an accurate assessment of AM as non-dissociative PTSD and PTSD+DS have been shown to have contrasting patterns of symptomology and emotion modulation. Additionally, a comparison between a trauma-exposed military groups with PTSD+DS and civilian PTSD+DS may provide critical knowledge regarding differing effects of these respective index traumas on dissociative symptomology and the resultant effect on cognitive function. Additionally, the rehearsal of AMs found in our study design may also present as a limitation. Our design required participants to identify and discuss memories before measuring brain function, possibly resulting in the re-encoding of events. Although many studies in the literature employ such a paradigm, few studies examine spontaneous AM retrieval (e.g. St. Jacques, et al., 2011). Future research could compare both paradigms and determine their respective validity. Lastly, it is important to note that our study did not include an age-matched healthy control group where the HCs were significantly younger than the PTSD and TEMC groups. Episodic memory function has been shown in the literature to be susceptible to age-related cognitive decline (Head, Rodrigue, Kennedy, & Raz, 2008; Rossi, et al., 2004; Small, 2001) with one study reporting that neural (e.g. structure volume) and cognitive (e.g. inhibitory control) factors mediated age differences in episodic memory (Head, et al., 2008). As evidence suggests age is related to AM function, the between groups comparisons in our study may be influenced, in part, by age differences in AM function. It is therefore important for future AM research to include age-matched groups to provide a true comparison of AM function between groups, avoiding the potential effect of age on AM function.

Our study sought to examine the neural correlates of emotional AM retrieval in military-related PTSD and to determine the relation between clinical variables and the neural correlates of emotional AM retrieval in military-related PTSD. The results of this work provide significant evidence of altered AM function in a trauma-exposed military population with PTSD, implicating implicitly cued processes previously shown to be altered in PTSD. Here, our research was able to contribute an increasingly comprehensive view of cognitive function in PTSD through a discussion of the relation between altered implicit and explicit cognitive processes in PTSD. Additionally, we were able to demonstrate a provocative association between dissociative symptoms and the neural correlates of AM function in a trauma-exposed military population. As a result, we suggest both AM function and dissociative symptoms should be included as targets for treatment in a trauma-exposed military population. We particularly urge treatment of dissociative symptoms in military-related PTSD as military-related PTSD and dissociative symptoms have individually been shown to exacerbate the disorder, resulting in increased illness burden. Importantly, the research covered in this study addressed a high-risk population, providing a compelling contribution to current literature aimed at characterizing military-related trauma exposure and PTSD.

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Tables

Table 1. Participant characteristics by diagnostic group.

	Healthy Controls (n=14)	Trauma-exposed controls (n=9)	PTSD Patients (n=13)
Demographics			
Sex	11M/3F	8M/1F	13M
Age	24.9(7.7)	39.4(10.7) *	46.2(9.3) **
RBANS Immediate memory	106.9(7.8)	100.3(20.9)	96.2(13)
RBANS Delayed memory	96.5(9.7)	92.6(8.1)	99.2(12)
RBANS Visuospatial/constructional	90.5(14.9)	88.4(19)	104.5(13)
RBANS Language	98.8(21.5)	102.8(12.6)	99.4(6.9)
RBANS Attention	93(19.6)	96.9(14.8)	103.6(17.9)
RBANS Total	95.8(11)	94.1(10.3)	100.7(12.4)
NART Verbal IQ	107.1(8)	105.7(10.9)	104.8(10.1)
NART Performance IQ	109.1(4)	108.5(5.1)	108.1(4.7)
NART Full scale IQ	108.6(7.4)	107.6(9.6)	106.9(8.8)
CTQ Emotional abuse	5.9(1.4)	6.1(1.2)	8.9(5.2)
CTQ Physical abuse	5.6(1.2)	6.3(1.8)	8.7(5.1)
CTQ Sexual abuse	5(0)	5(0)	5.6(2.2)
CTQ Emotional neglect	6.5(2.5)	7.3(2.7)	10.2(5.4)
CTQ Physical neglect	5.2(0.6)	5.7(1.1)	6.6(1.7)*
CTQ Total	28.1(4.5)	30.4(5.2)	40.1(15.4)*
MDI Depersonalization	5.1(0.4)	5.6(1.4)	6.9(3.6)
MDI Derealization	5.1(0.3)	6(1.6)	8.6(4.4)*
MDI Disengagement	7.9(2)	8.8(2.4)	12(4.2)*
MDI Identity dissociation	5(0)	5(0)	4.9(0.9)
MDI Memory Lapse	5.3(0.6)	7(4.9)	9.5(4.7)*
MDI Emotional Constriction	5.1(0.3)	7.6(3.3)	11.2(5.1)**
MDI Total	33.5(2.6)	40(12.6)	52.9(18.6)*
DERS Non-acceptance of emotional responses	7.9(2.1)	13(7.8)	17.8(5.5)**
DERS Goal directed behaviour	11.5(5.1)	8.9(2.6)	15.3(4.6)*
DERS Impulse control	7.6(2.1)	8.9(5.1)	12.9(6.5)
DERS Emotional awareness	10.8(3.8)	14.3(5.3)	19.2(5.6)*
DERS Emotion regulation strategies	10.2(2.7)	12.1(4.8)	19.2(7.4)**
DERS Emotional clarity	6.8(2.6)	7(1.9)	13.9(4.3)**
DERS Total	54.8(12.7)	64.1(21.7)	98.2(19)**
Subjective Recall Ratings During fMRI Scan			
Neutral memories: Re-experiencing	4.8(1.8)	4.9(1.5)	4.6(1.3)

Neutral memories: Emotional change	1.6(0.4)	2.2(1.2)	2.8(1.2)*
Positive memories: Re-experiencing	5.3(1.3)	5.6(1.5)	5.2(1.2)
Positive memories: Emotional change	4.5(1.2)	4.5(1.5)	4.6(1.1)
Negative: Re-experiencing	5.1(0.9)	5.9(1.4)	5(1.4)
Negative: Emotional change	3.8(1.5)	4.6(1.5)	4.3(1.7)
Trauma/most stressful memories: Re-experiencing	5.4(1)	6.3(0.7)	6(1)
Trauma/most stressful memories: Emotional change	4.4(1.3)	4.9(1.3)	4.8(1.6)

Note: values are *n* or means (standard deviation)
Abbreviations: RBANS, Repeatable Battery for the Assessment of Neuropsychological Status; NART, National Adult Reading Test; CTQ, Childhood Trauma Questionnaire; MDI, Multiscale Dissociation Inventory; DERS, Difficulties in Emotion Regulation Scale.
*Significance $p < .05$
**Significance $p < .001$

Figures

Figure 1. Increases in hemodynamic activity among the TEMC group during negative memory retrieval. a) Within the TEMC group, left frontal operculum showing increased activity during negative memory retrieval > baseline activation, b) Within the TEMC group, left frontal operculum showing increased activity during negative memory retrieval > neutral memory retrieval, c) Within the TEMC group, right frontal operculum showing increased activity during negative memory retrieval > neutral memory retrieval. Bar represents *t* scores. Significant one sample *t*-test presented at alpha < .05 (FWE-corrected) in whole brain analysis.

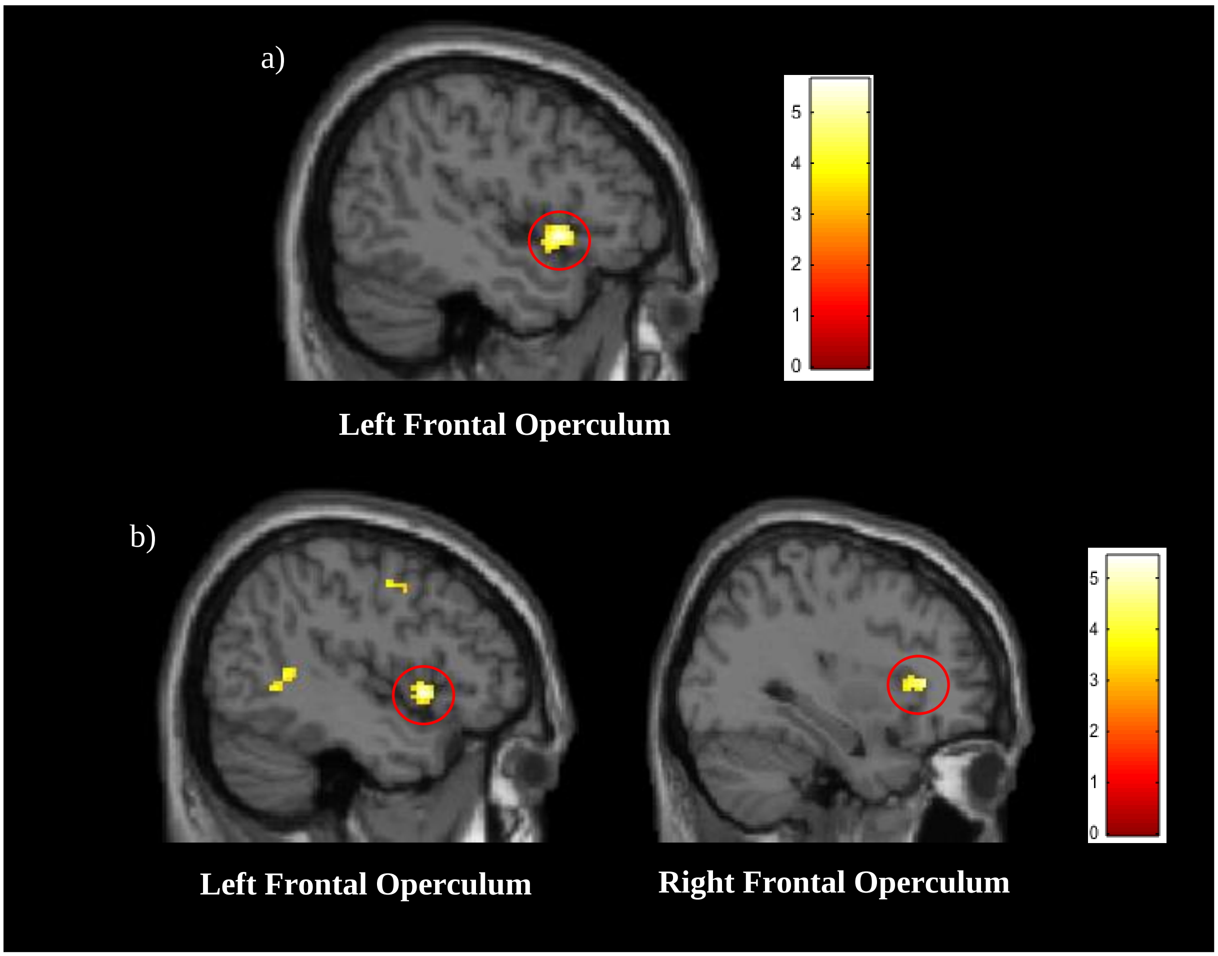


Figure 2. Increases in hemodynamic activity among the PTSD group during traumatic memory retrieval. a) Within the PTSD group, left lingual gyrus showing increased activity during traumatic memory retrieval > baseline activation, b) left lingual gyrus showing increased activity among PTSD > TEMC traumatic memory retrieval. Bar represents *t* scores. Significant one sample *t*-test presented at alpha < .05 (FWE-corrected) within a ROI.

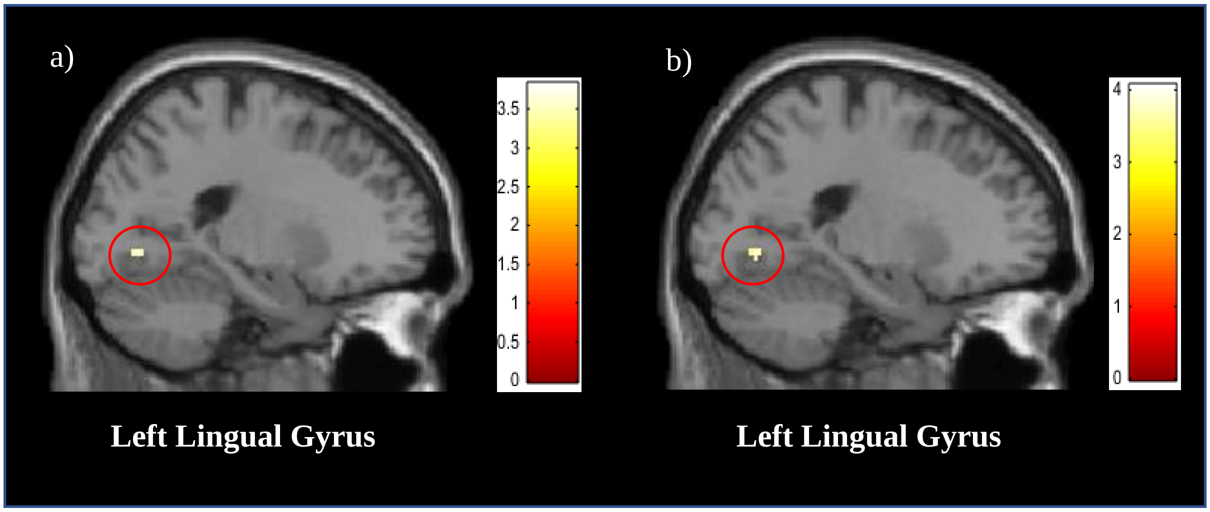
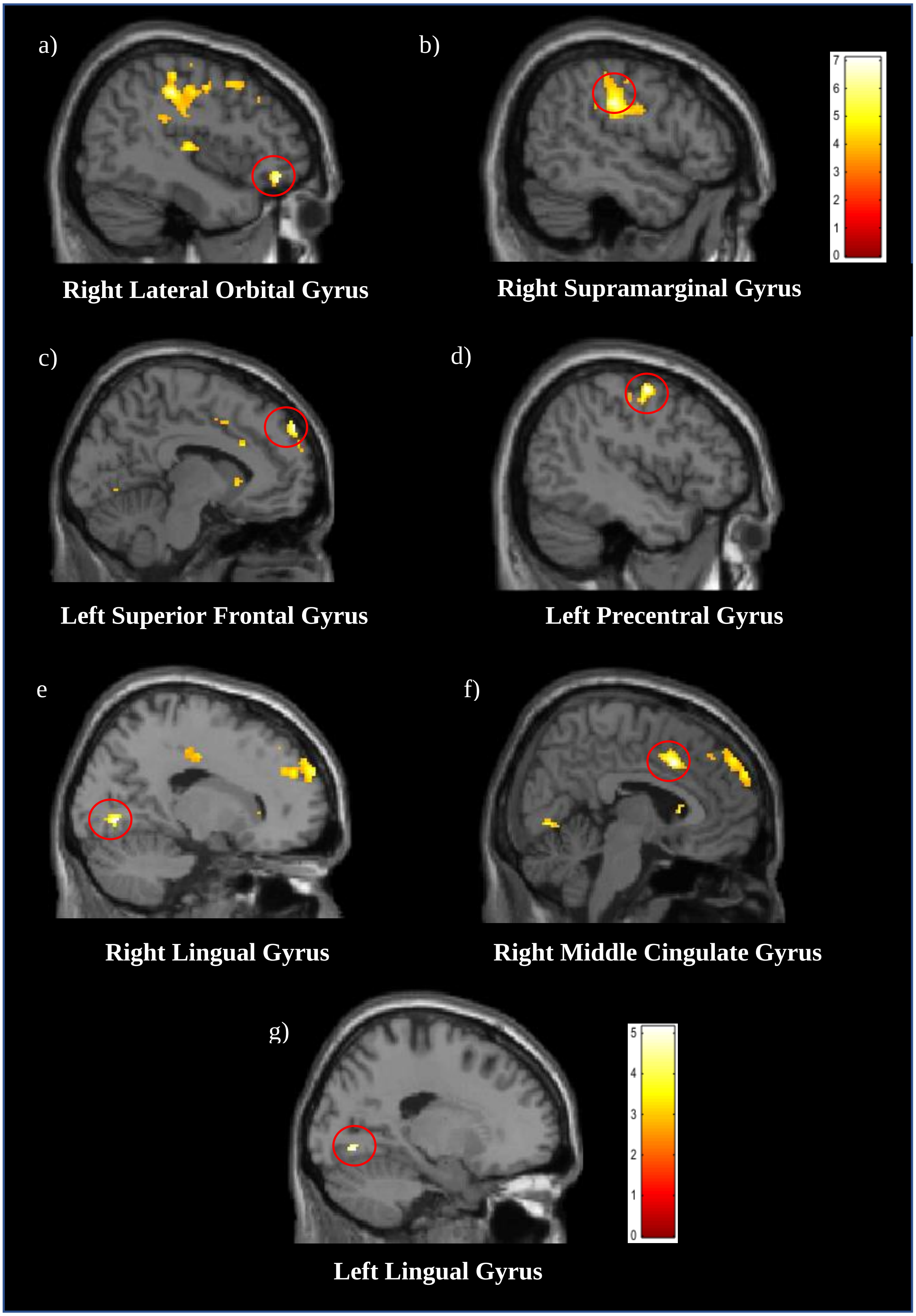


Figure 3. Correlation between hemodynamic activity and dissociative symptoms among trauma-exposed group during traumatic memory retrieval. Within the trauma-exposed group, activation in the a) right lateral orbital gyrus, b) right supramarginal gyrus, c) left superior frontal gyrus, d) left precentral gyrus, e) right lingual gyrus, f) right middle cingulate gyrus, and g) left lingual gyrus* is associated with dissociative symptoms. Bar represents *t* scores. Significant one sample *t*-test presented at alpha < .05 (FWE-corrected) in whole brain analysis and within an ROI* at alpha < .05 (FWE-corrected).


a)
b)
7
6
5
4
3
2
1
0
Right Lateral Orbital Gyrus
Right Supramarginal Gyrus
c)
d)
Left Superior Frontal Gyrus
Left Precentral Gyrus
e
f)
Right Lingual Gyrus
Right Middle Cingulate Gyrus
g)
5
4
3
2
1
0
Left Lingual Gyrus

Chapter 3

Afterword

In chapter two, we presented evidence of altered neural activation during AM retrieval in military members and veterans with PTSD. Specifically, we reported increased left lingual activation during trauma compared to baseline activation in the PTSD group and compared to trauma memory retrieval in the trauma-exposed military members and veterans without PTSD (TEMC) group. We argued the observed lingual activation may indicate flashback-type trauma memory retrieval in the PTSD group. The lingual gyrus is hypothesized to be a part of frontoparietal attentional processes important to memory that are separated into a 1) dorsal network that selects stimuli via controlled memory processes such as memory search, monitoring strategies, internal goals and a 2) ventral frontoparietal network that detects pertinent stimuli and is associated with spontaneous and rapid retrieval completed with a high degree of confidence (Burianová, Ciaramelli, Grady, & Moscovitch, 2012; Ciaramelli, Grady, Levine, Ween, & Moscovitch, 2010). The ventral frontoparietal network is associated with occipital regions, such as the lingual gyrus, which may indicate that the lingual gyrus is in involved in a degree of subconscious processing (Ciaramelli, et al., 2010). Furthermore, ventral posterior regions are also shown to contribute to implicitly cued memory processes which represent unconscious memory function (Roediger, 1990; Vuilleumier, Schwartz, Duhoux, Dolan, & Driver, 2005). As both the ventral frontoparietal network of attention and implicitly cued memory function rely on ventral posterior areas, it may be concluded that the lingual gyrus is implicated in unconscious processing. It is in keeping with this proposed role of the lingual gyrus that we posit the PTSD group experienced flashback-like retrieval of their traumatic memories. It is well established that flashbacks may occur as intrusive memories, outside of the conscious control of patients with PTSD (McNally, et al., 1997). Here, our research substantiates the need for trauma memories as therapeutic targets in the treatment of PTSD; indeed, trauma-focused therapies, such as trauma-focused cognitive behavioural therapy, have been shown to be more effective in the treatment of PTSD as compared to non-trauma focused treatments (Bisson, & Martin, 2009). Furthermore, our findings suggest abnormal implicit functioning specific to military-related PTSD and trauma memory retrieval where lingual dysfunction could represent a neural correlate underlying increased illness burden in this population. However, future research would be required to corroborate this claim and provide further detail.

We also found increased frontal operculum (FO) activation in TEMC negative memory retrieval compared to their baseline function and neutral memory retrieval. We posit this observed FO activation may indicate regulation of negative affect. The FO is hypothesized to contribute to functions important to appraisal processes, notably conceptual processing and determining meaning (Pulvermüller, 2013; Karama, et al., 2011). Moreover, the FO has been implicated in the integration of information observed in a study examining source episodic memory retrieval (Lundstrom, Ingvar, & Petersson, 2005). Taken together, the function of the FO can be aligned with the general notion that frontal areas are involved in emotion regulation (Green, & Malhi, 2006). In keeping with the proposed function of the FO, its activation during TEMC negative memory retrieval may then suggest that individuals in the trauma-exposed group were engaging in regulation-related activity, possibly in an effort to regulate negative affect associated with the memories. Furthermore, the specificity of this finding to the TEMC group may indicate activation unique to trauma-exposed AM retrieval and could therefore represent an effect of military-related trauma exposure alone. Further research is required to substantiate

these findings and to determine the difference between trauma-exposed military members/veterans with and without PTSD.

Our research was also able to report significant findings regarding dissociative symptoms, operationalized as a combination of depersonalization and derealization items on the Multiscale Dissociation Inventory (Briere, 2002). Here, we reported a positive correlation between frontal and posterior regions and dissociative symptoms during trauma memory retrieval in a trauma-exposed group, suggesting a significant association between dissociative symptomology and AM function in a trauma-exposed military population. These findings emphasize a consideration of dissociative symptomology in those exposed to military-related trauma, advocating for dissociative symptoms to be included as treatment targets. In fact, current research highlights the need for a tailored treatment approach when treating those experiencing dissociative symptoms as they may demonstrate a decreased response to psychotherapy (Lanius, et al., 2012). Furthermore, our findings demonstrate a crucial need for dissociative symptoms to be addressed in military-related trauma as both dissociative symptomology and military-related trauma have been shown to exacerbate PTSD symptomology.

3.1 Future directions

This thesis has maintained a focus on AM in PTSD and has endeavoured to reveal an association between clinical variables and AM in PTSD. Beyond measuring task-positive activation of regions, such as the original research presented in chapter two, research has turned to the examination of task-negative functional connectivity and task-negative large-scale brain networks that support cognitive function to determine the effects of PTSD on brain structure and function. For example, an investigation of the resting-state functional connectivity of amygdala subregions has revealed differences between amygdala connectivity in PTSD-DS and PTSD+DS. Here, a study reported greater functional connectivity of amygdala subregions with prefrontal areas associated with emotion regulation compared to PTSD-DS, suggesting a unique biomarker for PTSD+DS while also providing neural correlates for the symptom profile of PTSD+DS (Nicholson, et al., 2015). Similarly, an investigation into the resting-state functional connectivity of subregions of the insula in PTSD also revealed differential connectivity in PTSD-DS and PTSD+DS. Harricharan and colleagues (in press) reported increased connectivity of the bilateral anterior and posterior insula with posterior cortices, including the left lingual gyrus and left precuneus in PTSD+DS compared to PTSD-DS. Furthermore, they reported a positive correlation between state dissociation scores and dorsal posterior insula connectivity with the left fusiform gyrus in PTSD+DS (Harricharan, et al., in press). These authors suggest the observed connectivity of the insula further provides neural markers of the PTSD+DS symptom profile as visual cortex activation is strongly associated with PTSD+DS (Harricharan, et al., in press). Moreover, altered resting-state connectivity between the amygdala and insula in PTSD has been demonstrated by Sripada and colleagues (2012). Here, combat veterans with PTSD exhibited increased positive connectivity between the amygdala and insula compared to combat veterans without PTSD, demonstrating aberrant coupling between areas heavily involved in the emotional response (Sripada, et al., 2012). Sripada et al. (2012) emphasize the possibility that such altered functional coupling of the amygdala and insula may provide a neural basis for emotion-dysregulation often reported in PTSD. These results are corroborated by previous research conducted by Rabinak and colleagues (2011) who also speculate increased connectivity between the amygdala and insula in veterans with PTSD compared to veterans without may underlie, in part, increased psychological arousal reported in PTSD given the role of the amygdala in perceiving and responding to fear and the insular role of monitoring bodily states. Taken together, the above findings describe altered connectivity between regions that may underlie emotion dysregulation in PTSD, a symptom posited to contribute to cognitive deficits in the disorder as discussed in chapter one. Future research could expand to study resting-state connectivity in tandem with neural activation during cognitive function. One such study on veterans with differing levels of PTSD symptoms reported that reduced performance on tasks of inhibition was associated with decreased resting-state connectivity between the rostral ACC and medial PFC, key regions involved in emotion regulation (Clausen, et al., 2017). The authors of this study posit that the observed cognitive deficits may relate to aberrant self-referential processing seen through the decreased connectivity between medial regions involved in self-referential processing. Research examining cognitive function and resting-state connectivity in PTSD is newly emerging and has the potential to provide a comprehensive insight into the effects of PTSD.

Additional to studies of the connectivity of individual regions, the PTSD literature is beginning to examine intrinsic connectivity networks that underlie a broader scope of brain function (Akiki, Averill, & Abdallah, 2017; Yeo, et al., 2011). The following three networks have been identified and are examined in PTSD: 1) the default mode network (DMN), involved in self-referential processing primarily at rest, 2) the central executive network (CEN), involved in goal directed behaviour, cognitively demanding tasks, and emotion regulation, and 3) the salience network (SN), involved in the detection of salient internal and external stimuli (Akiki, et al., 2017; Chand & Dhamala, 2016; Menon, 2011; Yeo, et al., 2011). An extensive review conducted by Akiki and colleagues (2017) suggests large scale functional and structural abnormalities in PTSD seen through an overactive and hyperconnected SN possibly resulting in diminished connectivity and activity of the DMN and CEN. Specifically, increased connectivity within regions of the SN (e.g. amygdala, insula, dorsal ACC) at rest in PTSD may prime the individual for saliency, altering modulation of DMN and CEN activity (Akiki, et al., 2017). Here, regions in the DMN have been shown to exhibit decreased coupling (e.g. the ventromedial PFC and hippocampus) in PTSD which may affect self-referential function such as autobiographical memory (Akiki, et al., 2017). Although research on the CEN in the PTSD literature is limited, extant work describes weak connectivity between known regions of the CEN such as the premotor cortex and dorsolateral PFC which has been associated with increased exposure to trauma (Akiki, et al., 2017). Aberrant CEN functioning could lead to cognitive deficits in patients with PTSD, although further research is required to elucidate the effects of altered CEN function on cognition in PTSD (Akiki, et al., 2017). Taken together, evidence emerges of an overactive SN which may destabilize DMN and CEN function, resulting in widespread changes of connectivity in individuals with PTSD (Akiki et al., 2017). However, the authors of the review highlight the need to examine causal interactions between these intrinsic connectivity networks in an effort to elucidate a potential chain of events that may lead to affective and/or cognitive dysfunction in PTSD (Akiki, et al., 2017).

Identifying cognitive deficits in PTSD is not only important to the characterization of the disorder but is also inherently significant to its treatment. Recent developments in the treatment of PTSD seek to address cognitive deficits through cognitive remediation. Cognitive remediation therapy (CRT) entails learning behavioural strategies that target the development of core cognitive skills and has been shown to be effective in the treatment of schizophrenia, anorexia nervosa, and depression (Bowie, et al., 2013; Brockmeyer, et al., 2014; McGurk, et al., 2007). As the success of CRT has been previously demonstrated in neuropsychiatric illness, it can be suggested that it would be beneficial in the treatment of cognitive deficits seen in PTSD (Lanius, et al., 2015). Although research on CRT in PTSD is limited, the use of goal management training (GMT) in PTSD is emerging. GMT employs a top-down approach to skills training which aims to improve goal-directed behaviour that relies on basic cognitive processes and executive function (Levine, et al., 2000). A GMT outcome study of combat veterans with PTSD and mild traumatic brain injury has demonstrated a decrease in executive dysfunction in patients after 10 biweekly sessions of GMT (Waid-Ebbs, et al., 2014). However, little other work has been completed regarding GMT in PTSD, despite demonstrated efficacy in disorders that include executive function and attentional deficits such as frontal lobe damage and substance abuse (Alfonso, Caracuel, Delgado-Pastor, & Verdejo-Garcia, 2011; Levine, et al., 2011). Currently, our lab is investigating GMT in PTSD, examining performance on a wide range of cognitive functions before and after GMT where a recent feasibility study has demonstrated the efficacy of GMT in treating cognitive deficits (Boyd, et al., 2019).

3.2 Conclusions

The current thesis aimed to examine the functional neuroanatomy of AM in PTSD while also investigating clinical variables and their relation to AM function in PTSD, specific to a military population. We were able to demonstrate altered trauma memory retrieval compared to baseline function in PTSD and TEMC trauma memory retrieval, implicating the lingual gyrus in the altered AM response in PTSD and potentially providing a neural correlate for increased illness burden in military-related PTSD specific to AM function. We were further able to show FO activation in negative compared to baseline function and neutral memory retrieval in the TEMC group, positing unique regulation specific to a trauma-exposed military population without PTSD. Our findings also provide evidence of an association between dissociative symptoms and the functional neuroanatomy of trauma memory retrieval in a trauma-exposed military population, indicating a link between dissociative symptoms and AM function. Cumulatively, our original research has worked to significantly contribute to current AM literature in PTSD and demonstrates a need to identify and treat cognitive deficits in PTSD with concentrated consideration of dissociative symptoms. Importantly, we reported critical findings regarding a high-risk population in PTSD, contributing to the limited current knowledge of military-related PTSD and the neural correlates of AM.

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