The International Journal of Indian Psychology
ISSN 2348-5396 (Online) | ISSN: 2349-3429 (Print)
Volume 12, Issue 1, January- March, 2024
DIP: 18.01.020.20241201, DOI: 10.25215/1201.020
https://www.ijip.in

Review Paper

Investigating the Neural Correlates of Partial PTSD: A Systematic Literature Review

KP Tania Augusta[1]*, Ashwin Shankaran[2]

ABSTRACT

Background: Partial PTSD (PPTSD) describes the presence of clinically significant trauma symptoms that do not meet the diagnostic criteria of posttraumatic stress disorder (PTSD). This study examines the neural correlates of PPTSD by drawing a symptomatic overlap between Generalised Anxiety Disorder (GAD) and Major Depressive Disorder (MDD). **Methods:** Literature databases included Google Scholar, PubMed and Mendeley. Following a preliminary screening, six exclusion criteria (Inadequate data, No neural correlates, Comorbidities, Lifetime prevalence of PPTSD, Inconsistent results, Age criteria) were applied. The reference lists of all selected papers were also screened. **Results:** A total of sixty-three papers were reviewed and critically appraised using the Joanna Briggs Inventory. This study examined the neural networks and dysregulations associated with Partial Post-Traumatic Stress Disorder (PPTSD), shedding light on the intricate interplay of brain regions contributing to the spectrum of PPTSD symptoms. Five key neural networks and regions that play a central role in PPTSD were identified. The Default Mode Network (DMN), Salience Network (SN), Executive Control Network (ECN), Emotion Regulation Network, and the Hypothalamic-Pituitary-Adrenal (HPA) Axis. Along with which the role of the hippocampus in conjunction with these networks was also highlighted. **Conclusion:** These findings emphasise the complex neural landscape underlying PPTSD, with the interplay between these networks and regions contributing to a wide range of neural dynamics. Further research, including neuroimaging and neuromodulatory studies, is needed to explore these interactions in greater depth. Understanding these neural mechanisms is crucial for developing effective interventions and treatments for PPTSD and related disorders.

Keywords: *Partial PTSD, Generalised Anxiety Disorder, Major Depressive Disorder, Neural Correlates*

Partial PTSD or PPTSD describes clinically significant symptoms of PTSD in trauma-exposed individuals who do not meet the full criteria for PTSD (Mylle & Maes, 2004;Weiss et al., 1992). Partial PTSD (PPTSD) is also referred to as subthreshold or subclinical PTSD. The majority of the research references the symptoms of PPTSD using the DSM-III-R and DSM-IV. Some of the earliest papers that describe PPTSD believed that the diagnostic screening criteria for posttraumatic stress disorder were too restrictive for the

[1]Student Researcher, School of Allied Healthcare and Sciences, JAIN (deemed-to-be University)
[2]Teaching Associate, School of Allied Healthcare and Sciences, JAIN (deemed-to-be University)
*Corresponding Author

Received: January 10, 2024; Revision Received: January 23, 2024; Accepted: January 27, 2024

purposes of clinical intervention, resource allocation, and personal well-being. This failed to account for the trauma-exposed (TE) individuals that did not fulfil the DSM criteria. A 2011 study found that in a sample of 34,653 from the wave 2 NESARC data, the lifetime prevalence of PPTSD was 6.6%. Out of the 6.6%, 8.6% of these were women and 4.5% were men. These findings were reported to be statistically significant. (J. Cukor et al., 2010) highlighted the clinical significance and need for treatment of PPTSD. A 2009 study by Pietrzak et al., reported statistically significant psychosocial impairment in individuals with PPTSD. Over 60 publications show the prevalence and morbidity of PPTSD among a wide assortment of traumatised individuals. People with PPTSD also show significant functional impairment in the social and workplace setting (Pietrzak et al., 2011; J. Cukor et al., 2010). Stein et al. (1997) reported comparable help seeking behaviour between PTSD and PPTSD. Studies to further establish the clinical significance of this disorder are needed (Stein, Walker, Hazen, Forde, 1997). Existing studies on PPTSD have studied war veterans, ambulance workers and survivors of toxic chemical exposures, disasters, and various authors have stressed the need to expand the understanding of PPTSD to include other traumas.

There lacks a consensus on a common set of diagnostic criteria with clinical significance. According to Kulka et al., and Pietrzak et al., Partial PTSD is identified when affected individuals meet Criterion B (re-experiencing) and either Criterion C (avoidance) or Criterion D (arousal), or if they meet Criterion B and endorse at least one symptom from Criteria C and D (per the DSM IV) (Kulka et al., 1998; Pietrzak et al., 2009). Mylle & Maes., in their work noticed there to be two sets of viewpoints; one where criteria E and F are included in the diagnostic criteria for PPTSD and one where criteria E and F are excluded. The reasons for this difference in viewpoint were not explained in their work. However, through the extensive literature review, conducted by Mylle & Maes (2004), it can be concluded that PPTSD is indicated by symptoms from clusters B, C, and D per the DSM IV (Mylle & Maes, 2004; American Psychological Association, 1994).

Since most of the research on PPTSD has been done on the basis of symptoms of PTSD in the DSM IV, we have identified the clusters of symptoms that indicate PPTSD (Mylle & Maes, 2004; Pietrzak et al., 2009) and drawn a comparison to the same clusters as it appears in the PTSD symptoms of DSM V. Symptoms under criterion B remain the same in the DSM V as in DSM IV, whereas some cluster C (avoidance) symptoms are now under criterion D. Criterion D symptoms of the DSM IV are labelled under criterion E for the DSM V. A final comparison by the authors of this paper yielded that the symptoms of PTSD in the DSM V that indicate PPTSD can be identified using are criteria B1, B4 and B5 (recurrent, involuntary and intrusive distressing memories of the traumatic event, intense or prolonged psychological distress at exposure to internal or external cues, marked physiological reactions to cues), criteria C (persistent avoidance of stimuli associated to the traumatic event), D (negative alterations in cognition and mood associated with the traumatic event), E (marked alterations in arousal and reactivity) and criteria H (Symptoms attributable to substance, medication, alcohol/other medical conditions). The findings of a study conducted by Mylle & Maes., 2004, was used to establish this. From here on out this paper will refer to the DSM V for the symptomatic presentations of the disorders studied. (American Psychological Association, 2000, 2013; Mylle & Maes, 2004).

In order to extend the diagnostic boundaries of post-traumatic stress disorder (PTSD), and investigate the neural correlates of partial PTSD, the authors decided to map out the symptomatic overlap to well-researched disorders. Upon careful comparison to the DSM IV/V and existing literature for various disorders, a search for overlap of symptoms was

conducted. Generalised Anxiety Disorder (GAD), Major Depressive Disorder (MDD), and posttraumatic stress disorder (PTSD) were chosen as they had the closest symptomatic overlap to PPTSD. This was reviewed further and the details are described below.

Generalised Anxiety Disorder (GAD), characterised by excessive anxiety and worry was chosen due to the symptoms of restlessness, being easily fatigued, difficulty concentrating, irritability, muscle tension, disturbed sleep, avoidant behaviours, hypervigilance, difficulty controlling worry, exaggerated startle responses. The DSM V characterises GAD as excessive worry that interferes with psychosocial functioning. The pronounced and distressing nature of GAD, therefore, sees a lot of overlap with the criteria for PPTSD. Major Depressive Disorder (MDD) sees overlap with PPTSD in terms of the avoidant behaviours, difficulty in concentration, irritability, fatigue or loss of energy, sleep disturbances, diminished interest or pleasure (anhedonia) and reduced motivation. Research has shown the presence of dysphoric (dissatisfaction) moods accompanying trauma responses, whether it be full or partial PTSD. *Diagnostic and Statistical Manual of Mental Health Disorders* (5th ed.; *DSM-5*; American Psychiatric Association, 2013)

In order to be able to hypothetically map the neural correlates for PPTSD based on the proposed diagnostic criteria, the symptomatic presentations and the respective neural correlates of GAD and MDD were used by the authors of this study.

METHODOLOGY

Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, the authors conducted a literature search, data extraction and analysis of articles (Moher et al., 2009).

Eligibility Criteria

The initial criterion was to consider articles since 2010, however, that did not yield sufficient data and the authors had to broaden the search horizon to include articles from 1994 onwards. This also indicated that the current research topic has limited research conducted in the last 10 years. Peer-reviewed articles published in English were considered to be eligible. The authors also considered studies that were conducted in adult (> 18 years) populations. Studies that had points of gender differentiation were also not considered. Exclusion of studies that reflected extensive comorbidities was also excluded. A combination of quantitative and qualitative analyses, and literature reviews/meta-analyses were considered.

Search Strategy

Searches on Google Scholar, PubMed and Mendeley were performed from May to July 2023.

Search terms included *Post-Traumatic Stress Disorder (PTSD), Trauma exposed individuals, Generalised Anxiety Disorder (GAD), Major Depressive Disorder (MDD), neural correlates, neuroanatomy, and brain regions.*

In order to verify the prevalence of subclinical presentations of trauma, the search terms were modified to include *partial PTSD, subclinical PTSD, and subthreshold PTSD* after reviewing *Trauma-Informed Care in Behavioural Health Services* (SAMHSA, 2014) The search terms were used in various combinations as keywords or as subject headings.

Study selection

Figure 1. Study selection flow diagram. Adapted from (Kalvas & Monroe, 2019; Moher et al., 2009) PTSD = post-traumatic stress disorder.

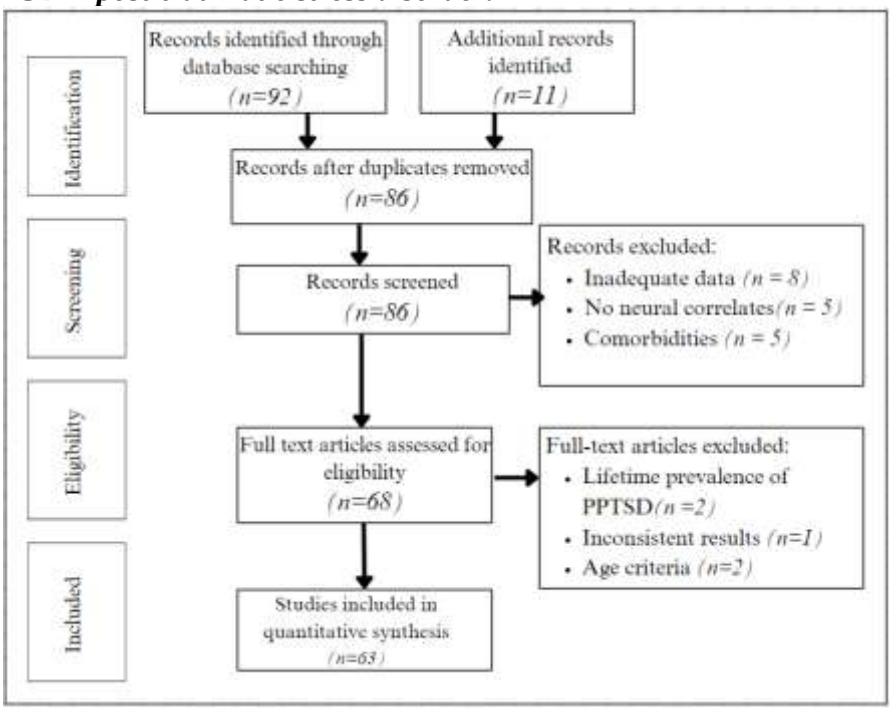

Figure 1 illustrates the process of selection. The database search alone yielded *n=92* records, and a secondary search from reference lists of the articles yielded an additional *n=11* records. A total of *n=103* eligible articles were combed through for the removal of duplicates, leaving *n=86* records for screening. Removal of articles that had incomplete data on the chosen variables (*n=8),* articles that did not include details about neural correlates *(n = 5),* and those that included extensive comorbidities (*n=5*) completed the screening.

The initial screening left (*n = 68)* records for full-text reviews. To narrow it down, the authors looked at articles that specifically mentioned the diagnostic criteria for PTSD and removed the ones that defined a lifetime prevalence of PPTSD (*n =2*). Records that reflected vague or inconsistent diagnostic criteria for partial PTSD were also excluded (*n=1*). A final exclusion of the age criteria *(n=2)*. This left 63 records to include in the literature review.

Data Extraction

The review of the 63 records was done by compiling data into categories of partial PTSD, neural correlates of PTSD, GAD, and MDD. To begin with the review, the co-authors conducted a thorough analysis of all the brain correlates associated with PTSD, GAD, and MDD. Following which, all the records pertaining to partial PTSD, subclinical PTSD, or subthreshold PTSD were reviewed following which the recurring themes in terms of the diagnostic criteria were noted.

The primary interest during data extraction was to focus on understanding partial PTSD and the neural correlates that reflect the same symptomatology as in GAD and MDD. The results have been compiled in a constructive way, after careful review.

Risk of Bias

To critically appraise the articles and aid in the detection of bias, the Joanna Briggs Institute critical appraisal tools (Moola et al., 2017; Kalvas & Monroe, 2019) were used. This included specific criteria that increase the reliability and validity of a research study. Each study design is unique, and the common themes among the criteria include clear descriptions of inclusion criteria, sample demographics, valid reliable measurement of the exposure variable (trauma), appropriate control for confounding factors, valid and reliable measurement of the outcome variable (PPTSD, neural correlates) and the use of appropriate statistical analyses.

The 63 studies were marked as met, not met or not applicable. The two authors agreed on the ratings for each study, the degree to which the chosen studies met or did not meet the criteria determined the overall value of the synthesis of results.

RESULTS

As stated, the neural correlates reported in Generalised Anxiety Disorder (GAD), and Major Depressive Disorder (MDD) were mapped in order to hypothesise the neural correlates involved in PPTSD. A few broad categories of symptoms were created including processing of emotions, fear response, decision making, physiological responses, self-referential processing, and memory consolidation/retrieval. The primary brain regions that notably play a part in these symptoms (processing of emotions, fear response, decision making, physiological responses, self-referential processing, and memory consolidation/retrieval), are the Amygdala, Anterior Cingulate Cortex (ACC), Hippocampus, Prefrontal Cortex and the areas in the Default Mode Network. (Crossman & Neary, 2020a)

In a neurotypical population, the Amygdala is assumed to evaluate sensory information in terms of motivation and affective significance. The Amygdala also plays a role in fear conditioning and learning (Ward, 2015). The Anterior Cingulate Cortex (ACC) plays a host of cognitive functions such as emotional expression, attention allocation, and mood regulation in neurotypical individuals. The subregions of the ACC seem to have distinct roles in combination with other brain regions (Crossman & Neary, 2020a). The hypothalamus plays an important role in the sympathetic nervous system responses, while the brainstem aids in the execution of autonomic function, sleep, and relaxed states. The Periaqueductal Grey (PAG) is associated with stereotyped species-specific behaviour relating to defensiveness. Mobbs et al., 2007 found that activation of the PAG is associated with feelings of dread in healthy control (neurotypical) participants with an impending threat (Charney et al., 2013a; Mobbs et al., 2007)

The Prefrontal Cortex (PFC) is thought to play a critical role in monitoring and regulating emotions. The PFC spans a vast area of the cortex, and is thus broken down into subregions. The *dorsolateral* and *ventrolateral* prefrontal cortices are commonly associated with the regulation of emotions (Gyurak et al., 2011). The Default Mode Network (DMN) contributes to sustained attention during the lack of task performance and maintenance of consciousness. The PFC is seen to function during decision-making, problem-solving, pattern identification, sustained attention, identifying and maintaining sensory stimuli, and emotional stimuli processing. The Thalamus, the brain's relay centre monitors and maintains activity in other brain regions, diverting impulses and information when needed through its "gating" mechanism (Charney et al., 2013a; Crossman & Neary, 2020a).

Post Traumatic Stress Disorder (PTSD)

Individuals with PTSD exhibit chronic hypervigilance, which is in part due to the abnormal basal functioning of the neuroendocrine stress systems, the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis. Overtime, individuals with PTSD may experience an "overload" or allostatic load due to a constant activation of the HPA axis. Abnormal basal functioning is associated with emotional reactivity, sleep difficulties, and exaggerated startles. Evidence suggests HPA blunting occurring in trauma-exposed (TE) individuals, marked by a lower basal and reactive cortisol, which may represent a form of allostatic protection.

The amygdala that evaluates the motivation and affective significance of sensory information. In 2019, Kleshchova et al. hypothesised that resting-abnormalities Amygdala connectivity would persist during affective processing in TE individuals, resulting in an exaggerated and persistent novel neural response to affective information. Including but not limited to hyperarousal, intrusive thoughts, difficulty regulating emotion (Kleshchova et al., 2019). Evidence from animal research and human neuroimaging studies suggests that the Amygdala-vACC (*ventral* ACC) circuit is part of the innate neural alarm system, a subcortico-cortical network involved in automatic threat detection (Charney et al., 2013a; Douglas Bremner, 2002). This affective evaluation is sent to the vACC, which then orients a response to the said stimuli. Evidence suggests that the vACC contributes to the excitation and inhibition of the Amygdala activity. Trauma-exposed (TE) individuals have a greater Amygdala-vACC connectivity at rest. This connectivity persists during affective tasks, symptom provocation and recall of negative memories that are autobiographical. This suggests a sustained activation of the neural alerting response even in the absence of threat, and this activation might be an underlying contributor to chronic trauma-related hypervigilance.

In individuals with PTSD, the *ventral* Amygdala cluster is hyperactive and the *dorsal* cluster is hypoactive. A meta-analysis (Goodkind et al., 2013) speculated that the *ventral* amygdala and Basolateral Amygdala (BLA) in terms of its role in acquiring fear responses and forming emotional memories. The hypoactive *dorsal* cluster is believed to correspond to the Centromedial Amygdala (CMA) assumes a mediating role in autonomic behavioural reactions to threat. This hypoactivation is posited to be related to emotional blunting and numbing seen in PTSD. When the Amygdala-Brain stem connection is hypoactive, it regulates autonomic responses.

Generalised Anxiety Disorder (GAD)

Two sub-regions of the Amygdala have been identified for the context of fear and anxiety, the Basolateral Amygdala (BLA) and the Centromedial Amygdala (CMA). It was found that the BLA responds nonconsciously to fearful emotion expressions (Etkin et al., 2004). The activation of the Amygdala has a positive correlation to the nonconscious processing of negative emotional expressions. In other words, as the Amygdala becomes more active, there is a greater likelihood of processing and reacting to negative emotional cues without conscious awareness. The CMA has subcortical projections to the Brainstem, Hypothalamus and the Periaqueductal Grey (PAG) (Pitakanen, 1997), which may explain the physiological responses associated with fear (increased heart rate, muscle tension, sweating). Additionally, the *anterior* Insula, which is responsible for processing bodily sensations, emotions, and social awareness, also contributes to eliciting physiological responses associated with fear.

The *ventromedial* Hypothalamus is connected to the PAG and Amygdala and is thought to aid in the coordination and modulation of defensive behaviours (Davis, 2002) in response to emotional stimuli. The Thalamus and Sensory Association Cortices (including but not limited to the Striatum (Steinhauser et al., 2023), send sensory information to the BLA, which in turn projects to the thalamic and cortical regions, including those that provide input into the BLA (Davis, 2002). The Amygdala-vACC connectivity that persists at rest has been associated with perceived chronic stress (Taren et al., 2015) and faster orienting to threat (Carlson et al., 2013). The vACC does not activate during implicit regulation tasks in people with GAD. (Heldt et al., 2007) noted a deviation in the normal functioning of the dorsal hippocampus can result in a reduction in the extinction of conditioned fear. *Dorsal* Hippocampus plays a role in constant re-evaluation of stimuli. A meta-analysis by Goodkind, Gyurak, & Etkin in 2013 found that the *anterior* Hippocampal areas adjacent to the Amygdala were consistently activated during emotional tasks in people with GAD.

Activation of the Periaqueductal Gray (PAG) in animals was associated with avoidance, defensive aggression and cardiovascular reactivity (Davis, 2002); activation of PAG in humans was reported with experiences of fear and anxiety, and it also regulates the appropriate behavioural responses. In individuals with GAD, there is a significant activation of the PAG.

The dACC (*dorsal* Anterior Cingulate Cortex)/dmPFC (*dorsomedial* Prefrontal Cortex) is shown to have increased activation during symptom provocation, emotional and resting state tasks (Hoehn-Saric et al., 2004; Paulesu et al., 2010). In anxiety disorders like GAD, the abnormalities in the *posterior* Hippocampus play a role in fear memory and extinction, while the anterior Hippocampus is associated with general fear or anxiety-related behaviours and responses. Hyperreactivity, a symptom of GAD, is regulated by the *ventrolateral* PFC (vlPFC) when viewing emotional stimuli (Monk et al., 2008; Shin and Liberzon, 2010). During a resting state scan, patients with GAD showed greater connectivity between the *dorsolateral* PFC (dlPFC) and Amygdala (Etkin et al., 2009). Activation in the Prefrontal connectivity to the Amygdala has been negatively correlated with symptom severity (Etkin et al., 2009; Shin and Liberzon, 2010).

Major Depressive Disorder (MDD)

The brain regions seen to be major contributors to Major Depressive Disorder (MDD) include but are not limited to the regions in the Default Mode Network, Prefrontal Cortex, Thalamus, Hypothalamus, Brainstem, Amygdala and Striatum.

Limbic areas such as Amygdala, Hippocampus, Basal Ganglia and Nucleus Accumbens aid in mediating raw unprocessed emotions. The areas of the Prefrontal Cortex such as the *subgenual, medial* and *dorsolateral* PFC in combination with the Cingulate Cortex aid in mediating cognitive processing of emotions, they also play a role in the “top-down” inhibition of the limbic system (Mayberg, 2003). The concept of "top-down" inhibition suggests that the Prefrontal Cortex, which is involved in higher-order cognitive functions and emotional regulation, can exert control over the limbic system. This control allows individuals to modulate their emotional responses and regulate their mood. However, in MDD, this top-down control may be compromised. The Prefrontal Cortex may struggle to effectively inhibit or regulate the heightened activity within the limbic system, contributing to the emotional and mood disturbances observed in depression. The disruption in this regulatory system is seen in individuals with MDD. (Charney et al., 2013)

The Default Mode Network (DMN) or the Medial Network comprising of the *Medial* Prefrontal Cortex (mPFC), Posterior Cingulate Cortex (PCC), Precuneus, Lateral Temporal Cortex, *Medial* Temporal Gyrus, *Inferior* Temporal Gyrus, Angular Gyrus, *Superior* and *Inferior* Parietal Lobules, *Subgenual* Anterior Cingulate Cortex (sgACC) is linked to self-referential functions such as recollection, imagination and understanding of others' mental states. In individuals with MDD, overactivation of the DMN is one of the causes of rumination (Charney et al., 2013a).

Neurons in the Orbito-Frontal Network areas respond to stimuli like sight, flavour and texture of food stimuli (multimodal stimuli). The presence of lesions in the Orbital Cortex reflects an inability to use rewards to guide behaviour, contributing to the lack of motivation associated with MDD. Studies seem to converge on the role that the Orbital Network plays in the assessment of the affective value of a multimodal stimulus. However, non-food sensory stimuli seem to be stemming specifically from the vlPFC, as there are no gustatory or olfactory inputs to this region of the Medial Network. The medial network has prominent connections with the Amygdala and other limbic structures, and the outputs to visceral control areas in the Hypothalamus and PAG are also strongly related to emotion and mood (Price & Drevets, 2013). In addition to which, projections from the mPFC to the Amygdala, Hypothalamus, PAG, Locus Coeruleus, Raphe & Brainstem Autonomic Nuclei are reported to play a major role in modulating the visceral and behavioural responses to stressors and emotional stimuli (Charney et al., 2013a).

The vmPFC in humans helps to regulate the automatic visceral responses to emotive stimuli. Limbic inputs encourage the continuation of established patterns of activity between the thalamus, cortex, and back, leading to consistent behaviour. On the other hand, inputs from the pallidum disrupt ongoing patterns, facilitating a shift between behaviours related to mood, evaluating the value of objects, and associating stimuli with rewards (Dean & Keshavan, 2017; Zhang et al., 2018)

Partial Post-Traumatic Stress Disorder (PPTSD)

Partial post-traumatic stress disorder can be characterised by intrusive thoughts, nightmares, dissociative symptoms, emotional reactivity, avoidant behaviours with regard to thoughts, activities, places or people, inability to recall important aspects, diminished interest, feelings of detachment, restriction in expressing affect, foreshortened future, sleep difficulties, increased irritability, concentration problems, hypervigilance, exaggerated startles, physiological reactivity. Partial PTSD is however distinguished from PTSD in a lot of literature by the presence of higher resilience.

As mentioned in the introduction of this paper, these symptoms were observed in MDD, GAD, and PTSD respectively in an overlapping manner. Therefore, it is possible to also map the neural networks involved for PPTSD and its dysregulation based on the established neural networks for GAD, MDD, and PTSD. The consolidated brain areas involved in PPTSD which were corroborated using (Crossman & Neary, 2020; Ward, 2015) and the findings of the neural networks for the other disorders are organised on the basis of the names of the neural networks (Charney et al., 2013; Crossman & Neary, 2020; Ward, 2015)
The first network of note is the Default Mode Network (DMN) comprising the *Medial* Prefrontal Cortex (mPFC), *Posterior* Cingulate cortex (PCC), the precuneus, and the *Inferior* parietal lobule. The next network to note is the Salience Network (SN) which comprises of the *Anterior* Insula, Anterior Cingulate Cortex (ACC), amygdala, the *Dorsal* Anterior Cingulate Cortex (dACC) and *Ventral* Anterior Cingulate Cortex (vACC). The

third network involved is the Executive Control Network (ECN) which comprises the *dorsolateral* Prefrontal Cortex (dlPFC), Anterior Cingulate Cortex (ACC), and the *Inferior* parietal lobule. The fourth network involved is the Emotion Regulation Network which comprises the Amygdala, *ventromedial* Prefrontal Cortex (vmPFC), and the Anterior Cingulate Cortex (ACC).

The final network involved is the Hypothalamus-Pituitary-Adrenal (HPA) axis which as the name suggests comprises the Hypothalamus, Pituitary, and the Adrenal gland. (Charney et al., 2013; Crossman & Neary, 2020; Ward, 2015)

It is crucial to recognise the complex interactions and potential overlap of brain networks, which highlights the complexity of these disorders. Investigations on the precise underlying mechanisms are still ongoing. Research has revealed both common patterns and different subtleties in network interaction across these various situations by using modern neuroimaging techniques.

DISCUSSION

A network of brain regions orchestrates a complicated interplay to give rise to a spectrum of symptoms in Partial Post-Traumatic Stress Disorder (PPTSD). The Default Mode Network (DMN), a group of brain regions that cooperate when resting and thinking inwardly, is one of them. According to research, the DMN is linked to self-referential cognition, memory retrieval, and emotional resonance. Its activity peaks while engaged in self-reflective activities and troughs when engaged in external duties. Rumination, self-blame, and emotional turbulence are examples of symptoms associated with DMN dysregulation (Goodkind et al., 2013). The intrusive thoughts, dissociative symptoms and emotional reactivity present in PPTSD may be related to this.

Parallel to which, the Salience Network (SN) assumes the role of processing pertinent stimuli and the direction of attention. This network guides the responsiveness to internal and external cues. It also processes bodily sensations and emotions, moderates cognitive conflicts and emotions, and orchestrates emotional responses. Dysregulation within the SN can contribute to symptoms of hypervigilance, and avoidant behaviours with regard to thoughts, activities, places or people observed in Partial Post-Traumatic Stress Disorder (PPTSD), potentially leading to heightened vigilance and startle responses due to an elevated focus on potential threats. The amygdala-vACC connectivity (innate neural alarm system) within the SN, at rest seen in TE individuals (Kleshchova et al., 2019) may be noticeable in people with PPTSD that seemingly persists during tasks relating to affect, symptom provocation and recall of negative, autobiographical memories; in individuals with PPTSD, this connectivity may be a contributing element to the intrusive thoughts, and nightmares.

It is our belief that the level of hypoactivation of the Amygdala-Brain Stem Connection would be at an intermediate level (Pietrzak et al., 2009) when compared to the extremes of people with full PTSD and no PTSD. The extent of hyperreactivity and hypoactivity in the ventral and dorsal Amygdala, and their relation to the BLA and CMA respectively, might have a different relationship in individuals with PPTSD than seen in PTSD. Physiological responses associated with fear, such as an exaggerated startle, hypervigilance, and physiological reactivity as seen in PPTSD could be in part due to the subcortical projections extending from the CMA to the brainstem, hypothalamus and PAG.

Concurrently, the Executive Control Network (ECN) governs higher-order cognitive functions. This network enables cognitive control, decision-making, and goal-directed behaviours. It manages attention, impulse control, and complex cognitive tasks. Dysregulation in the ECN can lead to challenges in concentration, decision-making, and cognitive symptoms of restriction in expressing affect, foreshortened future, increased irritability, and concentration problems, as seen in PPTSD. When a person with PTSD experiences a flashback or intrusive thought, the PAG in combination with the amygdala (threat response) and the PAG in combination with the *ventrolateral* PFC and *dorsolateral* PFC sees dysregulation (identification of reality); (Charney et al., 2013a) partial PTSD may reflect the same network.

Further insight into the dimension of emotion emerges from the Emotion Regulation Network. This network guides the processing and modulation of emotional responses. These interactions play a pivotal role in emotional equilibrium. Dysregulations seen in this network may contribute to dissociative symptoms, irritability, diminished interest, feelings of detachment, and mood fluctuations seen in PPTSD.

Along with these networks, the Hypothalamic-Pituitary-Adrenal (HPA) Axis marks its influence as it is the governing centre for activation of the sympathetic and parasympathetic nervous system (Ward, 2015) and choreographs a hormonal response to stress. Dysregulation within this axis can lead to altered physiological reactivity, sleep difficulties, hypervigilance, and exaggerated startles. SNS and HPA dysregulation are associated with trauma-related symptoms like chronic hypervigilance. HPA blunting, characterised by lower basal and reactive cortisol levels, aids in allostatic protection (the physiological and behavioural mechanisms that the body employs to maintain stability and counteract the negative effects of stressors and challenges) (Kleshchova et al., 2019). The role played by the basal functions in resilience formation may be replicated in PPTSD. A tenet of distinguishing partial PTSD from full PTSD is the element of higher resilience when exposed to trauma (Stein et al., 1997). This implies that when environmental challenges exceed an individual's ability to cope, this leads to allostatic load (allostatic load is the cumulative burden of chronic stress and life events) (Guidi et al., 2021; Kleshchova et al., 2019). HPA blunting forms to act as allostatic protection. The resilience seen in PPTSD might also be in part due to the prefrontal activation and prefrontal connectivity to the amygdala, which have been negatively correlated with symptom severity (Etkin et al., 2009; Shin and Liberzon, 2010).

The four networks (DMN, SN, ECN and the Emotion Regulation Network) work in conjunction with the hippocampus (Crossman & Neary, 2020a), as it is the primary information storage centre of the brain, so it would be remiss to mention its role in PPTSD. The posterior hippocampus is implicated in intrusive thoughts and nightmares, the extent and nature of the role played by it in PPTSD would need to be investigated further.

The interaction of these networks and regions, broadly interconnected yet subtly distinct in their contributions, highlights the complex neural landscape underlying PPTSD. The interplay between these networks and their associated symptoms marks a wide range of neural dynamics and necessitates a need to explore the interactions in more depth via neuroimaging and neuromodulatory studies.

Limitations

Our study has several limitations. Firstly, Partial PTSD has been studied across various populations in terms of the symptomatic presentations seen, each of the studies establish slightly differing dimensions for PPTSD, this study attempts to establish a uniform basis for symptomatic identification. The theoretical interpretation established in this paper needs to be validated through further clinical studies. Future research can look into establishing the clinical validity of the proposed symptomatic dimensions.

Further, the theorised neural correlates in the paper are proposed based on the symptomatic overlap seen in GAD and MDD. This is a limitation since further imaging studies will need to be conducted to establish these theorised correlates.

This study paper also does not account for the intensity of symptoms that may be associated with PPTSD. It also does not account for differences in presentations of symptoms seen at different time intervals following a trauma.

CONCLUSION AND IMPLICATIONS

The unified symptomatic dimensions of Partial PTSD can help establish a basis for clinical significance and diagnosis. Our results propose potential neural correlates for the symptoms associated with PPTSD, this can help broaden the current understanding of the condition. The proposed biomarkers and neural correlates can help trauma survivors that are likely to benefit from diagnosis and eventual treatment.

REFERENCES

American Psychological Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders V*.

American Psychological Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders IV*.

Amstadter, A. B., & Vernon, L. L. (2008). Emotional reactions during and after trauma: A comparison of trauma types. *Journal of Aggression, Maltreatment and Trauma*, *16*(4), 391–408. https://doi.org/10.1080/10926770801926492

Balaev, M. (2018). *Trauma Studies* (D. H. Richter, Ed.; First Edition). John Wiley & Sons Ltd.

Bandler, R., & Shipley, M. T. (1994). *Columnar organisation in the midbrain periaqueductal grey: modules for emotional expression?*

Bremner, D. J. (2006). *Traumatic Stress: Effects on the Brain*. www.dialogues-cns.org

Breslau, N., Lucia, V. C., & Davis, G. C. (2004). Partial PTSD versus full PTSD: An empirical examination of associated impairment. *Psychological Medicine*, *34*(7). https://doi.org/10.1017/S0033291704002594

Carlson, E. B., & Dalenberg, C. J. (2000). *A Conceptual Framework for The Impact of Traumatic Experiences*.

Chalton, L. D., & McMillan, T. M. (2009). Can "partial" PTSD explain differences in diagnosis of PTSD by questionnaire self-report and interview after head injury? *Brain Injury*, *23*(2). https://doi.org/10.1080/02699050802635273

Charney, D. S., Sklar, P., Buxbaum, J. D., & Nestler, E. J. (2013). *Neurobiology of Mental Illness*.

Christensen, M. C., Ren, H., & Fagiolini, A. (2022). Emotional blunting in patients with depression. Part III: relationship with psychological trauma. *Annals of General Psychiatry*, *21*(1). https://doi.org/10.1186/s12991-022-00395-1

Crossman, A. R., & Neary, D. (2020). *Neuroanatomy*.

Cukor, J., Wyka, K., Jayasinghe, N., & Difede, J. A. (2010). The nature and course of subthreshold PTSD. *Journal of Anxiety Disorders*, *24*(8), 918–923. https://doi.org/10.1016/j.janxdis.2010.06.017

Dean, J., & Keshavan, M. (2017). The neurobiology of depression: An integrated view. In *Asian Journal of Psychiatry* (Vol. 27, pp. 101–111). Elsevier B.V. https://doi.org/10.1016/j.ajp.2017.01.025

Dell'Osso, L., Carmassi, C., Massimetti, G., Daneluzzo, E., di Tommaso, S., & Rossi, A. (2011). Full and partial PTSD among young adult survivors 10 months after the L'Aquila 2009 earthquake: Gender differences. *Journal of Affective Disorders*, *131* (1–3). https://doi.org/10.1016/j.jad.2010.11.023

Dickstein, B. D., Weathers, F. W., Angkaw, A. C., Nievergelt, C. M., Yurgil, K., Nash, W. P., Baker, D. G., & Litz, B. T. (2015). Diagnostic Utility of the Posttraumatic Stress Disorder (PTSD) Checklist for Identifying Full and Partial PTSD in Active-Duty Military. *Assessment*, *22*(3). https://doi.org/10.1177/1073191114548683

Douglas Bremner, J. (2002). Neuroimaging Studies in Post-traumatic Stress Disorder. *Current Psychiatry Reports*, *4*, 254–263.

European Journal of Psychotraumatology. (2010). European Journal of Psychotraumatology. *European Journal of Psychotraumatology*, *1*(1). https://doi.org/10.3402/ejpt.v1i0.5768

Felmingham, K., Kemp, A., Williams, L., Das, P., Hughes, G., Peduto, A., & Bryant, R. (2007). Changes in anterior cingulate and amygdala after cognitive behaviour therapy of posttraumatic stress disorder. In *Psychological Science* (Vol. 18, Issue 2, pp. 127–129). https://doi.org/10.1111/j.1467-9280.2007.01860.x

Friedman, M. J., Resick, P. A., Bryant, R. A., & Brewin, C. R. (2011). Considering PTSD for DSM-5. In *Depression and Anxiety* (Vol. 28, Issue 9, pp. 750–769). https://doi.org/10.1002/da.20767

Gelkopf, M., Roe, D., Werbeloff, N., Hasson-Ohayon, I., Mueser, K. T., Caspi, A., & Weiser, M. (2013). The prevalence of full and partial posttraumatic stress disorder among people with severe mental illness in Israel. *Journal of Nervous and Mental Disease*, *201*(3). https://doi.org/10.1097/NMD.0b013e3182848d1e

Gerger, H., Werner, C. P., Gaab, J., & Cuijpers, P. (2022). Comparative efficacy and acceptability of expressive writing treatments compared with psychotherapy, other writing treatments, and waiting list control for adult trauma survivors: A systematic review and network meta-analysis. *Psychological Medicine*, *52*(15). https://doi.org/10.1017/S0033291721000143

Goodkind, M. S., Gyurak, A., & Etkin, A. (2013). Functional Neurocircuitry and Neuroimaging Studies of Anxiety Disorders. In K. J. Ressler (Ed.), *Neurobiology of Mental Illness* (pp. 606–620). Oxford University Press. https://doi.org/10.1093/med/9780199934959.003.0045

Guidi, J., Lucente, M., Sonino, N., & Fava, G. A. (2021). Allostatic Load and Its Impact on Health: A Systematic Review. In *Psychotherapy and Psychosomatics* (Vol. 90, Issue 1, pp. 11–27). S. Karger AG. https://doi.org/10.1159/000510696

Hruska, B., Patterson, P. D., Doshi, A. A., Guyette, M. K., Wong, A. H., Chang, B. P., Suffoletto, B. P., & Pacella-LaBarbara, M. L. (2023). Examining the prevalence and health impairment associated with subthreshold PTSD symptoms (PTSS) among frontline healthcare workers during the COVID-19 pandemic. *Journal of Psychiatric Research*, *158*. https://doi.org/10.1016/j.jpsychires.2022.12.045

Jakupcak, M., Conybeare, D., Phelps, L., Hunt, S., Holmes, H. A., Felker, B., Klevens, M., & McFall, M. E. (2007). Anger, hostility, and aggression among Iraq and Afghanistan

war veterans reporting PTSD and subthreshold PTSD. *Journal of Traumatic Stress*, *20*(6), 945–954. https://doi.org/10.1002/jts.20258

Karamustafalıoğlu, O. K., Fostick, L., Çevik, M., Zukerman, G., Tankaya, O., Güveli, M., Bakım, B., Karamustafalıoğlu, N., & Zohar, J. (2023). Ten-Year Follow-Up of Earthquake Survivors: Long-Term Study on the Course of PTSD Following a Natural Disaster. *Journal of Clinical Psychiatry*, *84*(2). https://doi.org/10.4088/JCP.22m14377

Kleshchova, O., Rieder, J. K., Grinband, J., & Weierich, M. R. (2019). Resting amygdala connectivity and basal sympathetic tone as markers of chronic hypervigilance. *Psychoneuroendocrinology*, *102*, 68–78. https://doi.org/10.1016/j.psyneuen.2018.11.036

Kopstein, A., White, K. D., & Currier, C. (2014). Trauma-Informed Care in Behavioural Health Services. In *Behavioral Health Services Recommended Citation Substance Abuse and Mental Health Services Administration. Trauma-Informed Care in Behavioral Health Services. Treatment Improvement Protocol (TIP) Series* (Vol. 57). HHS Publication No. http://store.samhsa.gov.Or,pleasecall

Lai, T. J., Chang, C. M., Connor, K. M., Lee, L. C., & Davidson, J. R. T. (2004). Full and partial PTSD among earthquake survivors in rural Taiwan. *Journal of Psychiatric Research*, *38*(3). https://doi.org/10.1016/j.jpsychires.2003.08.005

Lanius, R. A. (2015). Trauma-related dissociation and altered states of consciousness: A call for clinical, treatment, and neuroscience research. *European Journal of Psychotrauma tology*, *6*. https://doi.org/10.3402/ejpt.v6.27905

Ma, I. C., Chang, W. H., Wu, C. L., & Lin, C. H. (2020). Risks of post-traumatic stress disorder among emergency medical technicians who responded to the 2016 Taiwan earthquake. *Journal of the Formosan Medical Association*, *119*(9). https://doi.org/10.1016/j.jfma.2019.11.021

Marqueses, J. M. S., Sanz, J., García-Vera, M. P., Morán, N., & Eisma, M. C. (2023). Posttraumatic Stress Disorder, Anxiety, and Depression Symptoms after Potentially Traumatic Events: Latent Classes and Cognitive Correlates. *Journal of Nervous and Mental Disease*, *211*(2). https://doi.org/10.1097/NMD.0000000000001580

McEwen, B. S. (2003). Mood disorders and allostatic load. In *Biological Psychiatry* (Vol. 54, Issue 3, pp. 200–207). Elsevier Inc. https://doi.org/10.1016/S0006-3223(03)00177-X

Mclaughlin, K. A., Koenen, K. C., Friedman, M. J., Ruscio, A. M., Karam, E. G., Shahly, V., Stein, D. J., Hill, E. D., Petukhova, M., Alonso, J., Andrade, L. H., Angermeyer, M. C., Borges, G., de Girolamo, G., de Graaf, R., Demyttenaere, K., Florescu, S. E., Mladenova, M., Posada-Villa, J., … Kessler, R. C. (2015). Subthreshold posttraumatic stress disorder in the world health organisation world mental health surveys. *Biological Psychiatry*, *77*(4), 375–384. https://doi.org/10.1016/j.biopsych.2014.03.028

McNally, R. J. (2003). Progress and Controversy in the Study of Posttraumatic Stress Disorder. In *Annual Review of Psychology* (Vol. 54, pp. 229–252). https://doi.org/10.1146/annurev.psych.54.101601.145112

Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., Antes, G., Atkins, D., Barbour, V., Barrowman, N., Berlin, J. A., Clark, J., Clarke, M., Cook, D., D'Amico, R., Deeks, J. J., Devereaux, P. J., Dickersin, K., Egger, M., Ernst, E., Gøtzsche, P. C., … Tugwell, P. (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. In *PLoS Medicine* (Vol. 6, Issue 7). Public Library of Science. https://doi.org/10.1371/journal.pmed.1000097

Motreff, Y., Baubet, T., Pirard, P., Rabet, G., Petitclerc, M., Stene, L. E., Vuillermoz, C., Chauvin, P., & Vandentorren, S. (2020). Factors associated with PTSD and partial PTSD among first responders following the Paris terror attacks in November 2015.

Journal of Psychiatric Research, *121*. https://doi.org/10.1016/j.jpsychires.2019.11.018

Müller, M., Vandeleur, C., Rodgers, S., Rössler, W., Castelao, E., Preisig, M., & Ajdacic-Gross, V. (2014). Factors associated with comorbidity patterns in full and partial PTSD: Findings from the PsyCoLaus study. *Comprehensive Psychiatry*, *55*(4). https://doi.org/10.1016/j.comppsych.2014.01.009

Mylle, J., & Maes, M. (2004). Partial posttraumatic stress disorder revisited. *Journal of Affective Disorders*, *78*(1), 37–48. https://doi.org/10.1016/S0165-0327(02)00218-5

Naylor, J. C., Dolber, T. R., Strauss, J. L., Kilts, J. D., Strauman, T. J., Bradford, D. W., Szabo, S. T., Youssef, N. A., Connor, K. M., Davidson, J. R. T., & Marx, C. E. (2013). A pilot randomized controlled trial with paroxetine for subthreshold PTSD in Operation Enduring Freedom/Operation Iraqi Freedom era veterans. *Psychiatry Research*, *206*(2–3), 318–320. https://doi.org/10.1016/j.psychres.2012.11.008

Olff, M. (2015). Trauma and PTSD: setting the research agenda. In *European Journal of Psychotraumatology* (Vol. 6, Issue 1). Taylor and Francis Ltd. https://doi.org/10.3402/EJPT.V6.28092

Pai, A., Suris, A. M., & North, C. S. (2017). Posttraumatic stress disorder in the dsm-5: Controversy, change, and conceptual considerations. In *Behavioral Sciences* (Vol. 7, Issue 1). MDPI Multidisciplinary Digital Publishing Institute. https://doi.org/10.3390/bs7010007

Papanastassiou, M., Waldron, G., Boyle, J., & Chesterman, L. P. (2004). Post-traumatic stress disorder in mentally ill perpetrators of homicide. *Journal of Forensic Psychiatry and Psychology*, *15*(1). https://doi.org/10.1080/14789940310001630419

Pietrzak, R. H., Goldstein, M. B., Malley, J. C., Johnson, D. C., & Southwick, S. M. (2009). Subsyndromal posttraumatic stress disorder is associated with health and psychosocial difficulties in veterans of Operations Enduring Freedom and Iraqi Freedom. *Depression and Anxiety*, *26*(8), 739–744. https://doi.org/10.1002/da.20574

Pietrzak, R. H., Goldstein, R. B., Southwick, S. M., & Grant, B. F. (2011). Prevalence and Axis I comorbidity of full and partial posttraumatic stress disorder in the United States: Results from Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Anxiety Disorders*, *25*(3), 456–465. https://doi.org/10.1016/j.janxdis.2010.11.010

Posch, L., & Zube, A. L. (2023). Experiences of (Potentially) Traumatizing Deployments and Posttraumatic Stress in Police Officers an Exploratory Survey of Early Career Police Officers. *PPmP Psychotherapie Psychosomatik Medizinische Psychologie*. https://doi.org/10.1055/a-2087-0021

Price, J. L., & Drevets, W. C. (2010). Neurocircuitry of mood disorders. In *Neuropsychopharmacology* (Vol. 35, Issue 1, pp. 192–216). https://doi.org/10.1038/npp.2009.104

Prioux, C., Marillier, M., Vuillermoz, C., Vandentorren, S., Rabet, G., Petitclerc, M., Baubet, T., Stene, L. E., Pirard, P., & Motreff, Y. (2023). PTSD and Partial PTSD among First Responders One and Five Years after the Paris Terror Attacks in November 2015. *International Journal of Environmental Research and Public Health*, *20*(5). https://doi.org/10.3390/ijerph20054160

Radley, J. J., & Morrison, J. H. (2005). Repeated stress and structural plasticity in the brain. In *Ageing Research Reviews* (Vol. 4, Issue 2, pp. 271–287). Elsevier Ireland Ltd. https://doi.org/10.1016/j.arr.2005.03.004

Ranasinghe, P. D., Zhang, X., Hwang, J., Ranasinghe, P. M., Karunathilake, I. M., & Everly, G. S. (2023). PTSD and Depression 8 Years after the 2004 Tsunami in Sri Lanka. *Disaster Medicine and Public Health Preparedness*, *17*(4). https://doi.org/10.1017/dmp.2022.73

Russman Block, S. R., Weissman, D. H., Sripada, C., Angstadt, M., Duval, E. R., King, A. P., & Liberzon, I. (2020). Neural Mechanisms of Spatial Attention Deficits in Trauma. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *5*(10), 991–1001. https://doi.org/10.1016/j.bpsc.2019.05.014

Russo, S. J., Murrough, J. W., Han, M. H., Charney, D. S., & Nestler, E. J. (2012). Neurobiology of resilience. In *Nature Neuroscience* (Vol. 15, Issue 11, pp. 1475–1484). https://doi.org/10.1038/nn.3234

Sarajlić Vuković, I., Boričević Maršanić, V., Aukst Margetić, B., Paradžik, L., Vidović, D., & Buljan Flander, G. (2015). Self-Reported Emotional and Behavioral Problems, Family Functioning and Parental Bonding Among Psychiatric Outpatient Adolescent Offspring of Croatian Male Veterans with Partial PTSD. *Child and Youth Care Forum*, *44*(5). https://doi.org/10.1007/s10566-015-9299-9

Song, J., So, H. S., Choi, H., Choi, J. H., & Lee, S.-H. (2023). The Cognitive Profiles of Partial PTSD in Vietnam War Veterans. *Journal of Korean Neuropsychiatric Association*, *62*(2). https://doi.org/10.4306/jknpa.2023.62.2.78

Stein, M. B., Walker, J. R., Hazen, A. L., & Forde, D. R. (1997). Full and Partial Posttraumatic Stress Disorder: Findings from a Community Survey. In *Am J Psychiatry* (Vol. 154, Issue 8).

Stein, M. B. (2009). Neurobiology of Generalized Anxiety Disorder. In *Neurobiology of Generalized Anxiety Disorder*.

Steinhäuser, J. L., Teed, A. R., Al-Zoubi, O., Hurlemann, R., Chen, G., & Khalsa, S. S. (2023). Reduced vmPFC-insula functional connectivity in generalized anxiety disorder: a Bayesian confirmation study. *Scientific Reports*, *13*(1). https://doi.org/10.1038/s41598-023-35939-2

Stratta, P., Sanità, P., Bonanni, R. L., de Cataldo, S., Angelucci, A., Rossi, R., Origlia, N., Domenici, L., Carmassi, C., Piccinni, A., Dell'Osso, L., & Rossi, A. (2016). Clinical correlates of plasma brain-derived neurotrophic factor in post-traumatic stress disorder spectrum after a natural disaster. *Psychiatry Research*, *244*. https://doi.org/10.1016/j.psychres.2016.07.019

Varela, V. S., Ng, A., Mauch, P., & Recklitis, C. J. (2013). Posttraumatic stress disorder (PTSD) in survivors of Hodgkin's lymphoma: Prevalence of PTSD and partial PTSD compared with sibling controls. *Psycho-Oncology*, *22*(2). https://doi.org/10.1002/pon.2109

Ward, J. (2015). *The Student's Guide to Cognitive Neuroscience*.

Zhang, F. F., Peng, W., Sweeney, J. A., Jia, Z. Y., & Gong, Q. Y. (2018). Brain structure alterations in depression: Psychoradiological evidence. In *CNS Neuroscience and Therapeutics* (Vol. 24, Issue 11, pp. 994–1003). Blackwell Publishing Ltd. https://doi.org/10.1111/cns.12835

Zhen, R., Zhang, J., Pang, H., Ruan, L., Liu, X., & Zhou, X. (2021). Full and partial posttraumatic stress disorders in adults exposed to super typhoon Lekima: a cross-sectional investigation. *BMC Psychiatry*, *21*(1). https://doi.org/10.1186/s12888-021-03528-0

Acknowledgment

The author(s) appreciates all those who helped to facilitate the research process.

Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Author Contribution

K.P.T. Augusta contributed to the design, acquisition, analysis and interpretation of the drafted manuscript; critically revised the manuscript and agreed to be accountable for all aspects of work ensuring integrity and accuracy. A. Shankaran contributed to the conception, design, analysis and interpretation, critically revised the manuscript, gave final approval and agreed to be accountable for all aspects of work ensuring integrity and accuracy.

How to cite this article: Augusta, KP.T. & Shankaran, A. (2024). Investigating the Neural Correlates of Partial PTSD: A Systematic Literature Review. *International Journal of Indian Psychology*, *12*(1), 203-218. DIP:18.01.020.20241201, DOI:10.25215/1201.020