The Neurobiology of Posttraumatic Stress Disorder: Recent Advances and Clinical Implications

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Abstract: There have been considerable advances in understanding the neurobiology of posttraumatic stress disorder (PTSD) in the past two decades. This paper gives an overview of the key neurobiological models of PTSD, recent developments in the field, and outlines potential clinical implications of this work. Future research directions are discussed, including the need to explore the specific phenomenology of PTSD and recognise the diverse clinical presentations following trauma, use convergent hormonal, neuroimaging, and cognitive measures, employ longitudinal prospective designs and examine proposed mechanisms before and after treatment, and explore the impact of sex hormones, stress hormones, genetics and age of trauma on these processes.

Keywords: posttraumatic stress disorder, PTSD, neuroscience, neurobiology, trauma

Introduction

In the past two decades, significant advances in clinical and affective neuroscience have led to greater understanding of the neurobiology of posttraumatic stress disorder (PTSD). This paper will review the current neurobiological models of PTSD, the possible clinical implications of this neurobiological research, and highlight some outstanding questions and emerging research.

Neurobiological Models of PTSD

Fear conditioning and extinction.

Given the aetiological role of trauma exposure in PTSD, an early model was that of heightened fear conditioning in which the traumatic experience generates significant arousal that becomes associated (via fear conditioning processes) with previously neutral stimuli present at the time of trauma. Subsequent exposure to trauma reminders then promotes an ongoing conditioned fear response. Although intuitively appealing, there is little empirical evidence for heightened fear conditioning in PTSD in psychophysiological studies (Garfinkel et al., 2014; Milad, Igoe, Lebron-Milad & Novales, 2009). For example, one study employed a fear conditioning and extinction task in which a coloured light was paired with an electric shock (CS+) and a different coloured light was not paired with shock (CS−) in the fear conditioning phase. In the fear extinction phase, both coloured lights were presented without the shock and skin conductance response (SCR) was recorded as an index of conditioned fear. Both the PTSD and control group showed the same amount of acquisition of conditioned fear, reflected by a similar increase in SCR amplitude to the CS+ compared to the CS−. Group differences only emerged in the later extinction phases (Milad et al., 2009). An additional challenge for this model is that if fear conditioning was the aetiological process, it would follow that anyone experiencing a traumatic event would go on to develop PTSD. In fact, resilience and recovery...
are the normative response to trauma. Whilst approximately 70% of us will experience a significant traumatic stressor in our lifetimes, only 10% will develop PTSD (Kessler, Chiu, Demler, Merikangas, Walters, 2005).

The current “fear learning” model proposes that following fear conditioning during trauma exposure, impairments in fear extinction learning and recall (that is, the retention of fear extinction over time reflecting the consolidation of extinction memory) characterise those who develop PTSD (Pitman et al., 2012). For most individuals, conditioned fear responses are present to some degree in the acute aftermath of trauma (indexed by distress to reminders, nightmares, and intrusive memories) and these responses gradually decay in the initial days and weeks post-trauma. This fear extinction process is thought to involve the development of a new inhibitory association between safety (as the trauma is over) and the trauma reminder, leading to a gradual reduction in the fear response. Considerable psychophysiological evidence has revealed impairments in fear extinction learning, and more robustly, in the recall of fear extinction in PTSD. The latter is assessed by repeating the fear extinction phase of the fear conditioning and extinction task 24 hours following the initial task (Milad et al., 2009; Milad et al., 2010; Orr, Lasko, Macklin, Pineles, Chang, & Pitman, 2012; Pitman et al., 2012; Zeidan et al., 2011; Zuj et al., 2016).

There has been a rapid expansion of knowledge of the neurobiology of fear extinction, with animal and human neuroimaging research presenting remarkably convergent findings. Fear extinction learning and recall are mediated by a network comprising ventromedial prefrontal cortical and hippocampal regions interacting to inhibit amygdala-based fear conditioning processes (Milad & Quirk, 2012). The prevailing neurobiological model of PTSD suggests that PTSD is characterised by impaired prefrontal and hippocampal inhibition of amygdala-based fear processing. There is robust functional neuroimaging evidence of reduced activity in ventromedial prefrontal regions (centred in rostral and ventral anterior cingulate cortex) in PTSD (Bryant et al., 2008; Etkin & Wager, 2007; Felmingham, Williams, Kemp, Falconer, Peduto, & Bryant, 2010; Shin & Liberzon, 2010; Pitman et al., 2012; Williams et al., 2006), and of hyperactivity of amygdala response to fearful or trauma-relevant stimuli in PTSD (Bryant et al., 2008; Liberzon & Abelson, 2016).

Clinical implications.

This model of fear extinction learning has significant translational and clinical application as fear extinction learning is proposed as a key mechanism underlying exposure-based therapy (Graham, Callaghan & Richardson, 2014). Trauma-focused cognitive behavioural therapy (TF-CBT), which has both imaginal and graded in-vivo exposure as key elements, is considered the first-line evidence-based treatment for PTSD (Watts, Schnurr, Mayo, Young, Weike & Friedman, 2013). Indeed, functional neuroimaging research has revealed that successful exposure therapy in PTSD is associated with increased activation in ventromedial prefrontal fear extinction networks, and a concomitant reduction in amygdala activity in response to threat stimuli (Felmingham et al., 2007; Helpman et al., 2016; Peres et al., 2011). Eye-movement desensitization and reprocessing (EMDR) therapy is also considered an effective treatment for PTSD (Bisson, Roberts, Andrew, Cooper & Lewis, 2013), and interestingly, has also been associated with reduced limbic and increased prefrontal activation in neuroimaging PTSD studies (Lansing, Amen, Hanks, & Rudy, 2005; Pagani, Hogberg, Salmaro, Nardo, Sunden & Janssen, 2007).
Whilst considerable progress has been made in light of this fear extinction model, it is not necessarily specific to the phenomenology of PTSD and has been criticised for not accounting for symptoms such as spontaneous intrusive memories, hypervigilance, or emotional numbing (Liberson & Abelson, 2016). In this light, further—although not necessarily mutually exclusive—neurobiological models have been developed.

**Memory overconsolidation.**

PTSD is characterised by distressing, intrusive memories of the trauma that can manifest as spontaneous intrusions, or be triggered by trauma reminders. Cognitive models highlight the nature of the traumatic memory, which is fragmented, associated with intense arousal, readily primed and triggered, and poorly contextualised into autobiographical memory (Brewin, 2016; Ehlers & Clark, 2000). Prospective studies reveal that intrusive memories and distress to intrusions in the acute aftermath of trauma are predictive of subsequent PTSD (Ehring, Ehlers & Glucksman, 2008).

Neurobiological models propose that intrusive memories result from memory overconsolidation. In this model, stress hormones (noradrenaline and cortisol) are released at the time of trauma, and this arousal during encoding leads to an overconsolidation of trauma-memories and poorer contextualization of memories, resulting in stronger memory traces which are more readily primed (Pitman & Delahunty, 2005). A wealth of convergent animal research reveals that the interaction of noradrenaline and cortisol in the basolateral nucleus of the amygdala leads to stronger emotional memory traces (Wolf, Atsak, De Quervain, Roozendaal & Wingenfeld, 2016). Recent human memory research has also revealed that stress-induced increases in cortisol and noradrenaline predict greater emotional recall in healthy participants (Felmingham, Fong, & Bryant, 2012; Segal & Cahill, 2009). In the context of PTSD, there is evidence for heightened noradrenergic activity (Pitman et al., 2012), cortisol dysregulation (Lehrner & Yehuda, 2014), and for greater negative intrusive memories (Nicholson, Bryant & Felmingham, 2014). Furthermore, the interaction of noradrenaline and cortisol during encoding negative emotional images predicted negative intrusive memories in individuals with PTSD (Nicholson et al., 2014).

Of note, cognitive models propose a link between intrusive memories and fear conditioning processes. Specifically, the cognitive model proposes that key mechanisms underlying intrusive memories in PTSD are data-driven processing (i.e., processing sensorimotor elements of the experience to the detriment of processing the meaning of the experience), resultant strong perceptual priming and associational learning (fear conditioning; Ehlers & Clark, 2000). The neurobiological substrates involved in emotional memory consolidation (basolateral amygdala and hippocampus) overlap with regions subserving fear conditioning and extinction. Further convergent research reveals that both noradrenaline and cortisol impact on fear extinction learning and recall in healthy controls (De Quervain et al., 2011; Mueller & Quirk, 2012). However, very few studies have examined emotional memory and fear extinction processes within the same individuals and this is an important area for future research.

**Clinical implications.**

Recognition of heightened noradrenergic activity in PTSD, and the role of noradrenaline and cortisol in memory overconsolidation, has led to pharmacological interventions that aim to prevent the development of PTSD following trauma. Several studies have examined the effect of administering propranolol (a beta-receptor adrenergic antagonist) during the initial hours following trauma (the
memory consolidation window) to try to prevent subsequent PTSD. Three studies were conducted that revealed mixed (but largely null) effects of propranolol (Pitman et al., 2002; Stein, Kerridge,Dimsdale & Hay, 2007; Vaiva et al., 2003). However, most studies were limited by methodological issues (Qi, Gevonden & Shalev, 2016).

A more recent focus, with promising early results, is on the role of cortisol in enhancing fear extinction processes. Experimental studies in healthy controls reveal that elevating cortisol levels enhance fear extinction learning (De Quervain et al., 2011). Recent pilot studies have revealed that elevating cortisol levels using hydrocortisone or mifepristone in the early aftermath of trauma led to reduced PTSD symptoms at three months (Golier, Caramanica, Demaria & Yehuda, 2012; Schelling et al., 2004). A recent randomised controlled trial in PTSD has found that increasing cortisol levels with hydrocortisone administration enhanced response to exposure-based therapy in PTSD (Yehuda et al., 2015). Whilst preliminary, these promising initial findings require further investigation.

The emphasis of cognitive models on promoting a coherent narrative of the trauma to de-emphasize data-driven processing and facilitate integration of the memory into autobiographical memory highlights an important role for cognitive therapy, in addition to exposure-based treatments for PTSD. Indeed, there is considerable evidence for the efficacy of cognitive therapy in the treatment of trauma (Bisson et al., 2013; Bryant, Moulds,Guthrie, Dang, Mastrodomenico & Creamer, 2008). One therapeutic approach directly stemming from the Ehlers and Clark (2000) model is a variant of trauma-focused CBT involving both imaginal exposure and cognitive restructuring techniques, in which imaginal exposure is conducted to identify “hot cognitions” during the worst moment of the trauma narrative. Cognitive restructuring is then conducted on these hot thoughts to identify effective challenges, and these are subsequently integrated back into the trauma narrative using a briefer imaginal reliving technique. There is growing evidence for the efficacy of this hybrid technique (Ehlers, Clark, Hackmann, McManus, Fennell, 2005; Ehlers & Clark, 2008; Ehlers, Grey, Wild, Stott, Liness, Deale et al., 2013).

Salience detection and enhanced threat processing.

Other clinical features of PTSD that are not readily explained by the fear learning model are hypervigilance towards threat, and generalised hyperarousal (startle). Functional neuroimaging studies reveal a more extended neural network that is altered in PTSD with evidence of hyperactivity in insula (associated with interoceptive arousal, anticipatory processing), dorsal anterior cingulate (associated with generalised anxiety), and orbitofrontal and striatal regions in PTSD (Aupperle et al., 2012; Felmingham et al., 2008). The salience network (comprising insula, dorsal anterior cingulate cortex [ACC], and amygdala) that governs awareness, vigilance and threat detection (Menon, 2011) has been found to be hyperactive in PTSD (Bryant, Felmingham, Liddell, Das & Malhi, 2016; Liberzon & Abelson, 2016), and this greater salience network activation predicts response to cognitive behaviour therapy (van Rooij, Kennis, Vink & Geuze, 2016). Greater activation in insula and dorsal ACC activity has also been identified in anticipatory anxiety (Aupperle et al., 2012), and cognitive paradigms (Felmingham, Williams, Kemp, Rennie, Gordon & Bryant, 2009), suggesting a neurobiological underpinning to hypervigilance.

Clinical implications.

Identification of dysregulation in salience networks that underpin hypervigilance and generalised arousal suggest that these symptoms may not respond adequately to standard exposure-
based therapy, and may require additional targeted interventions. Indeed, even with effective exposure-based interventions, hyperarousal symptoms (including hypervigilance and startle) are some of the more treatment resistant symptoms.

**Impaired inhibitory control and emotion dysregulation.**

Impaired inhibitory cognitive control and emotion dysregulation are further potential mechanisms involved in PTSD. Neurocognitive deficits in executive functions such as working memory, attentional shifting, and inhibitory control are reliably seen in PTSD (Scott et al., 2015). Neuroimaging studies of emotion regulation, which typically examine cognitive reappraisal (an emotion regulation strategy involving changing emotional responses by reinterpreting the meaning of an emotional stimulus), have identified a network comprising lateral and ventrolateral frontal and orbitofrontal regions (Ochsner, Silvers & Buhle, 2012; Liberzon & Abelson, 2016). To date, only two fMRI studies have examined cognitive reappraisal in PTSD, and both report reduced activation in ventrolateral prefrontal regions (New et al., 2009; Rabinak et al., 2013). Evidence from other inhibitory tasks, such as “Go-No-Go” tasks, also reveal reduced activity in inhibitory inferior frontal networks in PTSD (Falconer et al., 2013).

**Clinical implications.**

Whilst the neurobiological literature in PTSD that directly examines emotion regulation is relatively sparse, the emphasis on emotion dysregulation is important, as it highlights the role of emotions that extend beyond fear. An important aspect of the phenomenology of PTSD is that it is often associated with significant states of anger, shame, guilt and grief. These emotions have not been accounted for in the current fear learning models. Typically, these emotional responses do not respond to fear extinction and exposure-based treatments, but require additional, typically cognitive, interventions. The emphasis on emotion regulation also highlights the importance of the interaction of cognitive control mechanisms and affect regulation as cognitive reappraisal is considered a process integral to cognitive therapy and restructuring (Graham et al., 2014). Therefore, further research should examine the neurobiology underlying reappraisal in PTSD, and how this changes with cognitive therapy.

**Dissociation.**

There has been growing recognition of a specific form of PTSD that typically entails a shutting-down of fear-based symptoms, and is characterised by emotional numbing alternating with intense emotional states, and a sense of depersonalisation and derealisation. Dissociative PTSD is associated with early childhood and cumulative trauma (Lanius, 2015) and has recently been recognised as a subtype of PTSD in DSM-5 (APA, 2013). A growing body of neurobiological research reveals a distinctive neurobiological pattern in dissociative PTSD, with evidence of hyperactivation (rather than typical hypoactivation) in prefrontal regions, an absence of amygdala reactivity to threat and dysregulation in insula and default mode networks (Lanius, Brand, Vermetten, Frewen & Spiegel, 2012; Nicholson, Densmore, Frewen & Lanius, 2015). The underlying mechanisms involved in dissociation, and their associated neurobiology require further research (Lanius, 2015).

**Clinical implications.**

The distinctive neurobiological profile in dissociative PTSD suggests that traditional fear-based extinction therapies may need modification when applied to individuals with predominant dissociative
symptoms. Given that effective exposure-therapy requires activation of emotional and fear-based processing (Foa & Kozak, 1986), individuals with emotional numbing may display a poorer response to traditional TF-CBT, which recent trials reveal (Bae, Kim & Park, 2016; Haganaars, Van Minnen & Hoogduin, 2010). Furthermore, individuals with this presentation may likely need longer, stage-based interventions to develop affect regulation and recognition skills (Cloitre, Petrovsky, Wang & Lu Lassell, 2012) prior to engaging in exposure-based interventions.

**Contextual processing.**

A recent integrative model proposes that impaired contextual processing may underlie many of the processes in PTSD highlighted in previous models, including impaired fear extinction learning, glucocorticoid and hippocampal dysregulation, impaired contextualization of trauma memories, and hypervigilance for threat (Liberzon & Abelson, 2016). The generalised impairment in contextual processing in PTSD may not only impede fear extinction learning by reducing the capacity to discriminate safe from threatening signals, it may also impede the integration of traumatic memories into autobiographical memory stores, leading to intrusive memories (Ehlers & Clark, 2000). Finally, deficits in contextual processing may reinforce a sense of derealisation and depersonalization, and promote hypervigilance for threat due to uncertainty of threat contingencies (Liberzon & Abelson, 2016). Very little research has been conducted in PTSD using explicit contextual processing paradigms, and more research is required.

**Outstanding Questions and Future Directions**

The previous sections have highlighted some recent developments in neurobiological research and models of PTSD, and noted the need for further research examining the specific phenomenology of PTSD that explore convergent cognitive, social, biological and contextual processes. Yet other compelling neurobiological questions with significant clinical implications remain and are emerging areas of research.

**Biomarkers of risk and resilience.**

The key question of what discriminates those who develop PTSD from those who don’t is driving a search for biomarkers of risk and resilience following trauma. Whilst several risk factors have been identified (previous trauma exposure and psychiatric history, catastrophic thinking about the trauma, impaired fear extinction learning, and poor social support), neurobiological markers are being increasingly investigated (see Michopoulos, Norrholm & Jovanovic, 2015, for recent review). The biomarker field has had limited success to date in identifying specific and sensitive markers of different psychiatric disorders, and there are inherent issues of multifactorial complexity and heterogeneity that present significant challenges to this agenda. However, potential biomarkers involving the hypothalamic-pituitary axis, psychophysiological reactivity, and inflammation appear promising in PTSD (Michopoulos et al., 2015).

Given the significant heritability of PTSD, there is an increasing focus on genetic and epigenetic factors interacting with trauma exposure to influence risk for PTSD (see Almi, Fani, Smith & Ressler, 2014, for recent review). Whilst most studies have been candidate genotype studies, which are typically underpowered, there have been a few large-scale genome-wide association studies in PTSD. However, emerging evidence implicates a role for genetic influences that affect glucocorticoid receptor
sensitivity (e.g., FKBP5) and sympatho-adrenal function (e.g., ADRB2) in PTSD (Liberzon & Abelson, 2016; Zuj, Palmer, Lommen & Felmingham, 2016).

A further question remains as to whether these neurobiological changes are premorbid vulnerability factors or if they develop following trauma. Whilst some evidence addressing this question has been derived from an identical twin cohort of Vietnam veterans (Pitman et al., 2012), and there is some evidence that pre-trauma capacity for fear extinction learning is a predictor of subsequent PTSD (Guthrie & Bryant, 2006; Lommen, Engelhard, Sijibrandij, Van Den Hout & Hermans, 2013; Orr et al., 2012), more longitudinal prospective studies are required. It is possible that some neurobiological changes represent adaptive changes to environmental stressors, thus neurobiological markers must be examined longitudinally and in the context of psychological, functional and behavioural outcomes.

**Female vulnerability to PTSD.**

A compelling and under-researched question concerns the female prevalence for developing PTSD. Numerous studies reveal that women develop PTSD at twice the rate of men, despite men being exposed to more traumatic events during their lifetime (Kessler et al., 2005; Tolin & Foa, 2006). Whilst some have argued this discrepancy is due to women being exposed to sexual violence, studies that have examined trauma type as a moderator have found that whilst it is a contributing factor, it does not account for the female vulnerability and in fact, females still develop PTSD at higher rates after experiencing the same trauma (Tolin & Foa, 2006).

Recent evidence has examined the influence of menstrual phase and sex hormones on fear extinction learning, with considerable animal and human evidence revealing that low levels of oestradiol are associated with impaired fear extinction learning and recall in healthy controls and in PTSD (Glover et al., 2012; Graham & Milad, 2013; Milad et al., 2010). This line of research suggests that the variability in oestradiol levels across the menstrual phase, and in particular the low oestradiol phase, may promote greater reactivity to stress or fear signals and an impaired capacity to inhibit fear (Glover, Norrholm & Jovanovic, 2015). Memory research also implicates progesterone in influencing intrusive memories (Bryant, McGrath & Felmingham, 2013; Felmingham et al., 2012; Feree, Kamat & Cahill, 2009), with the midluteal phase and higher levels of progesterone associated with greater memory consolidation (Felmingham et al., 2012; Wassell, Rogers, Felmingham, Pearson & Bryant, 2015). A prospective study that examined individuals in the acute aftermath of trauma, revealed that women who had experienced the traumatic event whilst in the midluteal phase reported significantly more flashback memories (Bryant et al., 2011). Taken together, this evidence suggests that sex hormones may impact on fear extinction and emotional memory consolidation to heighten the risk of developing PTSD following trauma. There are interesting potential clinical implications of this research: should we consider the impact of menstrual phase or use of oral contraceptives when delivering exposure therapy? Or is there the potential of elevating oestradiol levels as an adjunct to exposure therapy?

**Brain stimulation and neurofeedback.**

With the recognition of impaired prefrontal cortical activity in PTSD, there has been recent interest in using brain stimulation and neurofeedback techniques to enhance prefrontal function. Current trials are experimenting with repetitive transcranial magnetic stimulation, transcranial direct current stimulation, and real-time neurofeedback interventions (Gerin et al., 2016; Marin, Comprodos,
Dougherty & Milad, 2014). However, there are very few published studies and findings are preliminary given the limited sample sizes and variable methodologies. The reliability of some brain stimulation methodologies has also been questioned (Horvath, Vogrin, Carter, Cook & Forte, 2015), reinforcing the need for the development of robust stimulation protocols and extensive testing for these procedures to gain traction as clinical interventions for PTSD.

**Memory reconsolidation.**

A novel line of research is examining the efficacy of memory reconsolidation as an intervention for PTSD. It was traditionally thought that emotional memories were indelible, but there has been a renewed recognition that emotional memories can be made labile for a brief period of time when re-exposed to an emotional stimulus (Alberini & Le Doux, 2013; Nader, Schafer & Le Doux, 2000). It has recently been found in animal and human fear conditioning studies that if propranolol is administered during the memory reconsolidation window, when the memory is made labile by re-exposure to shock, then fear responses can be reduced significantly, if not eliminated (Kindt, Soeter & Vervliet, 2009; Soeter & Kindt, 2012). This memory reconsolidation process is distinct from fear extinction learning, and has important implications: if there is a capacity to erase fear responses to memories via noradrenergic blockage and memory reconsolidation, this may reduce the need for exposure therapy.

This research is at an early stage, with only one study translating these findings to a clinical population. In this study, memory reactivation (re-exposure to a spider) was followed by propranolol administration to spider phobics, which significantly reduced behavioural avoidance of spiders for up to one year (Soeter & Kindt, 2015). However, whether this protocol will translate to PTSD remains to be seen, as although an initial pilot trial revealed reduced physiologic arousal to trauma imagery with propranolol and reconsolidation (Brunet, Orr, Tremblay, Robertson, Nader & Pitman, 2008), a subsequent set of three pilot trials failed to replicate this effect (Wood et al., 2015). A known boundary condition for memory reconsolidation is that stronger and older memories are less responsive (Finnie & Nader, 2012). Given the strength and chronicity of trauma memories, memory reconsolidation procedures may prove unsuccessful in PTSD, although this is the subject of ongoing research.

**The impact of trauma and childhood trauma.**

Increasingly, it is recognised that trauma exposure itself (and not specifically PTSD) can impact on neural and emotional functioning (Stark et al., 2015). This highlights the importance of considering the dimensional and heterogeneous nature of trauma exposure, and not restricting ourselves to considering the impact of PTSD alone. Of particular importance is the impact of childhood trauma on neural and emotional function. Early childhood trauma is implicated in structural brain changes, including larger amygdala volumes (Tottenham, Hare, Quin, McGarry, Nurse & Gilhooly, 2010), greater amygdala reactivity to emotional and threat stimuli (Tottenham, Hare, Millner, Gilhooly, Zevin & Cary, 2011), and disturbed hippocampal structure and function (Tottenham, 2010). Furthermore, early childhood trauma is thought to impact the development of the prefrontal cortex and its connectivity with lower limbic structures (Gee et al., 2013). Many more neurobiological studies need to be done to examine the impact of the *timing* of trauma exposure at critical developmental periods and the subsequent effect on neural, hormonal and behavioural responses.

This has important clinical implications, as it is likely that different neural structures and functional connectivity between structures may be impacted if trauma exposure occurs at different critical developmental periods. Furthermore, the impact of the chronicity and cumulative nature of
repeated trauma exposures needs to be examined neurobiologically. Clinically, trauma that occurs in early childhood is associated with greater dissociative and emotional numbing symptoms (Lanius, 2015), and can disrupt attachment and interpersonal development, necessitating a greater focus in therapy on affect regulation, interpersonal function and being able to tolerate and recognize emotional states. Developing knowledge of the neurobiological impact of trauma at different life-periods will be useful in informing clinical practice into the future.

Conclusion

This review highlighted recent neurobiological models that extend beyond traditional fear learning models, and briefly outlined emerging research directions. Despite rapid advancement in neurobiological knowledge, it should be noted that the clinical neuroscience of PTSD is still in early stages, and definitive clinical implications are not yet available due to the preliminary nature of much of the science. With the rapid advances in the field, it is possible that neuroscience will more closely inform clinical understanding and practice in the future. However, the dissemination and implementation of clinical neuroscience into practice presents a considerable future challenge.

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