Review

How understanding the neurobiology of complex post-traumatic stress disorder can inform clinical practice: a social cognitive and affective neuroscience approach

Lanius RA, Bluhm RL, Frewen PA. How understanding the neurobiology of complex post-traumatic stress disorder can inform clinical practice: a social cognitive and affective neuroscience approach.

Objective: In this review, we examine the relevance of the social cognitive and affective neuroscience (SCAN) paradigm for an understanding of the psychology and neurobiology of complex post-traumatic stress disorder (PTSD) and its effective treatment.

Method: The relevant literature pertaining to SCAN and PTSD was reviewed.

Results: We suggest that SCAN offers a novel theoretical paradigm for understanding psychological trauma and its numerous clinical outcomes, most notably problems in emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing. A core set of brain regions appear to mediate these collective psychological functions, most notably the cortical midline structures, the amygdala, the insula, posterior parietal cortex and temporal poles, suggesting that problems in one area (e.g. emotional awareness) may relate to difficulties in another (e.g. self-referential processing). We further propose, drawing on clinical research, that the experiences of individuals with PTSD related to chronic trauma often reflect impairments in multiple social cognitive and affective functions.

Conclusion: It is important that the assessment and treatment of individuals with complex PTSD not only addresses traumatic memories but also takes a SCAN-informed approach that focuses on the underlying deficits in emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing.

Key words: complex PTSD; fMRI; self-reflection; default mode network; emotion regulation; dissociation; medial prefrontal cortex

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Accepted for publication July 5, 2011

Summations

- The social cognitive and affective neuroscience (SCAN) paradigm offers a metatheory for understanding the psychology and neurobiology of complex post-traumatic stress disorder (PTSD) and its effective treatment.
- A SCAN approach may offer new insights into disturbances related to emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing often observed in individuals with complex PTSD.
- A core set of brain regions appear to mediate these psychological functions. These include the cortical midline structures, the amygdala, the insula, posterior parietal cortex and temporal poles.
- Patients with PTSD have been shown to exhibit behavioural and neurobiological impairments impacting emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing as compared to healthy controls.
Introduction

The field of social cognitive and affective neuroscience (SCAN) seeks to understand how brain mechanisms mediate the complex social and emotional functions of the human mind. Topics of investigation within SCAN include the study of motivation and emotion, self-referential processing, empathy and mentalizing (theory of mind), just to name a few. As is well known, disturbances in these psychological functions are variably involved in a number of complex neuropsychiatric disorders, including autism (1, 2), attention deficit hyperactivity disorder (3), schizophrenia (4), anxiety (5) and depression (6–9).

The contemporary discipline of affective neuroscience originated from the landmark publications of Panksepp (10) and Davidson (11). From the beginning, the field of affective neuroscience has been influenced by the basic emotion view that there might be brain systems dedicated to mediating particular types of emotional behaviour (e.g. fear vs. anger), systems that might be relatively preserved across lower and higher species. Increasingly, higher-order explicitly social and metacognitive aspects of emotional processing and behaviour have also been studied in humans and their relationship to psychiatric disorders has been of growing interest (12, 13). In contrast, social cognitive neuroscience from the outset concerned itself with higher-order complex social behaviours including attitudes, empathy and theory of mind (14). The disciplines of affective neuroscience and social cognitive neuroscience are increasingly united under the field of SCAN, although for the purposes of simplicity and clarity, in the present review, we will discuss concepts traditionally falling under the scope of affective neuroscience and social cognitive neuroscience separately.

Social cognitive and affective neuroscience provides a perspective for the understanding of psychiatric disorders that is both scientifically and clinically valuable, as it provides a way to integrate research in emotion and social psychology with research in neuroscience (15). The complexity of psychiatric disorders and the need to understand them at multiple levels of analysis suggest that they can be best understood via a paradigm that integrates different levels. Lieberman [(15), p. 1], as one of the cofounders of social cognitive neuroscience, notes: ‘A full accounting of human biology cannot proceed without incorporating the social and emotional factors that modulate the functioning and health of biological systems’.

In this review, we examine the relevance of the SCAN paradigm for an understanding of the psychology and neurobiology of complex post-traumatic stress disorder (PTSD) and its effective treatment. For the purposes of the present review, complex trauma will be defined as repeated interpersonal trauma occurring during crucial developmental periods, as opposed to simple PTSD that follows more single incident trauma events. Researchers and clinicians who focus on complex PTSD are becoming increasingly aware of the need to account for physiological, developmental, environmental and social factors, suggesting that a SCAN approach may be particularly appropriate for promoting our understanding of PTSD.

Aims of the study

The aims of this study are to survey the literature illustrating how the core constructs and methods in social cognitive and affective neuroscience have been used towards understanding the pathophysiology of complex post-traumatic stress disorder with a particular focus on emotional awareness, emotion regulation, social emotional processing and self-referential processing. We propose that social cognitive and affective neuroscience offers a novel theoretical and methodological paradigm for understanding the psychological trauma and its myriad clinical outcomes, most notably self- and

Considerations

- It will be important that the assessment of complex PTSD includes an examination of the deficits in emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing.
- The treatment of individuals with complex PTSD should take a SCAN-informed approach that directly addresses underlying impairments in emotional/self-awareness, emotion regulation, social emotional processing, and self-referential processing.
- Outcome studies examining the effectiveness of treatments for complex PTSD should not only examine outcomes for PTSD symptoms but also measure the impact of interventions on emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing.

Lanius et al.
affect-dysregulation commonly observed clinically in persons who have suffered chronic interpersonal and developmental trauma.

**Material and methods**

Relevant empirical literature pertaining to SCAN and PTSD was reviewed.

**Results**

**Affective neuroscience**

In this section, we will discuss two concepts particularly relevant to understanding PTSD, namely deficits in *emotional awareness* and *emotion regulation*.

*Emotional/self-awareness.* Theorists have increasingly pointed out the significant role played by emotional processing in the across-species evolution of consciousness (16), and the brain mechanisms underlying how humans become subjectively aware of their emotional feelings and behaviour is a topic of significant interest in affective neuroscience. Emotional awareness refers to the capacity to be aware of and describe emotions in oneself and others and involves the ability to reflect upon internal affective experience (17, 18). As emotional awareness enables increased self-reflection and regulation of affective states, it is often considered a ‘cornerstone’ of emotion regulation. A history of a secure attachment with one’s primary caregivers has been suggested to play a key role in the development of emotional awareness (19, 20).

*The neurobiology of emotional/self-awareness.* Current theoretical models propose a superior–inferior division within the medial prefrontal cortex underlying emotional awareness [see (21–25); also see Table 1 for a description of frequently discussed brain regions and their functions (26–36)], and a distinction between conceptual and embodied forms of emotional awareness has been proposed. Conceptual emotional awareness refers to the ability to reflect on, interpret and make a decision about an embodied sensation or emotion [reviewed by Fogel (21)]. It is based on linguistic forms of expression, is usually considered to be rational, logical and explanatory and tends to transcend the present moment. This form of awareness is thought to be partly mediated by the dorsomedial prefrontal cortex and its connections with the interoceptive and body schema networks. In contrast to conceptual awareness, embodied emotional/self-awareness is based on sensing, feeling and acting, tends to be spontaneous and creative and is usually lived in the present moment. The ventromedial prefrontal cortex has been hypothesized to be a key brain structure underlying embodied emotional awareness. It is also thought to aid in making decisions when one is in the subjective emotional present.

*The impact of early life trauma on emotional/self-awareness.* Early adverse experience can significantly interfere with the development of emotional awareness. Being trapped in a dangerous environment, such as being with a chronically physically or sexually abusive caregiver, prevents individuals from using their emotional responses to guide effective actions and behaviours. For example, if a child is in an abusive relationship with a caregiver and has the impulse to escape, he/she may quickly learn that escape is not possible. A sense of learned helplessness may ensue. Individuals with such experiences therefore learn that emotional responses to traumatic events are futile because there is no escape from the situation and hence become increasingly disconnected from their inner emotional life in an attempt to disconnect themselves from extreme emotions that are out of their control. It is therefore not surprising that individuals with PTSD often exhibit problems being aware of their affective states and have difficulties identifying and labelling these states. Studies have shown

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### Neurobiology of PTSD and clinical practice

<table>
<thead>
<tr>
<th>Brain structure</th>
<th>Social cognitive and affective neuroscience-related functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial prefrontal cortex (26, 27)</td>
<td>Self-referential, reflective awareness</td>
</tr>
<tr>
<td>Anterior cingulate cortex (28, 31)</td>
<td>Monitoring and modulation of emotions through strong connection with emotional limbic system</td>
</tr>
<tr>
<td>Emotional component of the anterior cingulate cortex (28, 31)</td>
<td>Integrates cognitive and emotional aspects of experience</td>
</tr>
<tr>
<td>Cognitive component of the anterior cingulate cortex (31)</td>
<td>Assesses salience of emotional and motivational information</td>
</tr>
<tr>
<td>Posterior cingulate cortex (29, 30)</td>
<td>Regulates autonomic (heart rate, blood pressure) changes to emotional stimuli</td>
</tr>
<tr>
<td>Amygdala (32, 33)</td>
<td>Pain processing</td>
</tr>
<tr>
<td>Insula (34–36)</td>
<td>Self-referential processing</td>
</tr>
<tr>
<td></td>
<td>Episodic memory retrieval</td>
</tr>
<tr>
<td></td>
<td>Fear conditioning</td>
</tr>
<tr>
<td></td>
<td>Modulation of memory consolidation</td>
</tr>
<tr>
<td></td>
<td>Interoceptive awareness of bodily states</td>
</tr>
<tr>
<td></td>
<td>Bodily self-awareness</td>
</tr>
<tr>
<td></td>
<td>Regulation of sympathetic and parasympathetic systems</td>
</tr>
<tr>
<td></td>
<td>Sensation of pain</td>
</tr>
</tbody>
</table>

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**Table 1. Summary of brain structures and related functions relevant to this review**

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3
that these individuals have lower scores on the Levels of Emotional Awareness Scale (19, 37), that they consistently exhibit higher levels of alexithymia (difficulties identifying and labelling emotional states; for a meta-analysis, [see: (38, 39)]) and that they often show intense levels of emotional numbing, i.e. feeling like they cannot experience emotions (40). Below, we describe the studies of individuals with PTSD aimed at uncovering the underlying neurobiological mechanisms mediating such problems, namely emotional numbing and alexithymia (37, 40–42).

**The neurocircuitry underlying emotional numbing in PTSD.** Emotional numbing symptoms have long been recognized as an important symptom cluster in chronic PTSD (43–45) and represent negative prognostic indicators of psychological treatment (46, 47). Our group has recently examined trait emotional numbing symptoms as a predictor of neural activation during script-driven imagery in patients with PTSD related to childhood abuse of standardized positive (receiving others’ affection-praise) and negative (rejection-criticism) emotional scenarios. Patients were instructed to imagine that what was being described in the scripts was actually happening in the present and to pay attention to their emotional responses to each script. In order to measure emotional numbing symptoms, participants were asked to rate how often they had felt: i) ‘emotionally “numb,”’ as if you can’t experience emotions or feelings?; ii) ‘like your emotions and feelings are “frozen,” “sedated,” or “numbed out,”’ so that you can’t physically sense them?; iii) ‘“shut down” in an emotional sense, as if your emotional feelings have been “turned off” or “tuned out” in your body?; and iv) ‘“cut off” from your emotions and feelings, so that you can’t physically feel them, even if you try?’. Their ratings were then correlated with the extent of brain activation during imagery of positive (receiving others’ affection-praise) and negative (rejection-criticism) scripts. In women with PTSD related to prolonged childhood abuse, increased emotional numbing symptoms predicted decreased brain activation within the dorsomedial prefrontal cortex during imagery of both positive and negative social scripts, consistent with a role of the dorsomedial prefrontal cortex in higher-order reflective and metacognitive aspects of emotional functioning (see Fig. 1, left).

It is interesting to note that, within healthy women completing the same task, the more an individual exhibited the trait of ‘mindful observing’, referring to the intentional paying attention to one’s inner and external stimuli and experiences [e.g. ‘When I’m walking, I deliberately notice the sensations of my body moving’; ‘I notice changes in my body, such as whether my breathing slows down or speeds up’ (48, 49)], the more activation was observed within the dorsomedial prefrontal cortex (see Fig. 1, right) (50).

The development of the neural circuitry facilitating the human ability for conscious awareness of our emotional states, including the dorsomedial prefrontal cortex, may therefore be disturbed in PTSD, leading to the difficulties these individuals often have in reflecting on, interpreting and acting in accordance with emotion. A patient suffering from PTSD related to prolonged childhood abuse summarized feelings of emotional numbing in the following statement: ‘It’s like a blank, I think about my kids and I feel nothing for them. I’ll be sitting there feeling confused and numb, and I wonder what I’m supposed to be feeling. It’s like dead space...and when that happens, I have trouble using words, finding my words, I can’t talk’. The inability to describe feelings in words also relates to the concept of alexithymia, which will be described below.

**The neurocircuitry underlying alexithymia in PTSD.** Alexithymia refers to difficulties identifying and labelling emotional states (51, 52). Krystal (43, 44) described alexithymic traumatized persons as being ‘without emotion’, ‘robotic’, and as if ‘living-dead’. These descriptions parallel test items from the Glover (53) Numbing Questionnaire (e.g.
’I act mechanically like a robot’; ‘I feel dead or shut down’; ’My body feels paralyzed’; ‘I feel that I am in a fog’), illustrating the conceptual overlap between the emotional numbing and alexithymia concepts in traumatized subjects (54). Researchers, however, more often assess alexithymia using the 20-item Toronto Alexithymia Scale (TAS-20) (55), which measures difficulties i) identifying feelings and distinguishing them from bodily sensations (e.g. ‘I am often confused about what emotion I am feeling’, ‘I have feelings that that I can’t quite identify’) and ii) difficulties describing and communicating feelings (e.g. ‘It is difficult for me to find the right words for my feelings’, ‘People tell me to describe my feelings more’). The following quote from a traumatized client in psychotherapy with the first author describes the experience of alexithymia and elegantly illustrates the problems identifying and communicating feelings in words which is often so prominent in individuals with PTSD (42): ‘When I get emotions and stuff like that I don’t really feel them. I can say to someone I feel sadness because tears are welling in my eyes, but I do not know what that is really. They are just physical symptoms’. This quote also exemplifies that an individual may exhibit behavioural (e.g. crying) and psychophysiological signs (e.g. tachycardia) of emotional processing without being subjectively aware of his/her emotional state (56). Further evidence for the latter also stems from studies that have observed lower correspondence between self-reported affective experience and objectively coded facial affect in PTSD subjects as compared to controls in response to emotionally provocative stimuli (57, 58).

We have also reported that alexithymia symptoms predict brain activation during traumatic script-driven imagery in individuals with PTSD (42). In these studies, patients construct a narrative of their traumatic experience including as many sensory details as possible. These narratives are subsequently read to patients who are instructed to recall the traumatic memory as vividly as possible during an fMRI scan. Associations between alexithymia and neural activation were particularly marked for regions of the embodied emotional/self-awareness network. Specifically, during traumatic memory recall, increasing severity of trait alexithymia was associated with reduced activation within the ventromedial prefrontal cortex and the anterior insula. As described earlier, the ventromedial prefrontal cortex may be related to embodied emotional/self-awareness that is based on sensing, feeling and acting and may play a role in assigning the emotional value of a stimulus during self-referential processing. In contrast, the anterior insula has been proposed to be part of the interoceptive network. Craig (35, 59) has postulated that the anterior insula of the right hemisphere, possibly uniquely to humans, constitutes a basis for the subjective evaluation of one’s condition, that is, ’how you feel’. It is therefore not surprising that higher levels of alexithymia or difficulties identifying and labelling feeling states are associated with lower brain activation of the anterior insula.

In addition to correlations with the ventromedial prefrontal cortex and anterior insula, increasing severity of alexithymia was associated with reduced brain activation in the right inferior frontal gyrus. The right inferior frontal cortex responds to emotion regulation tasks, and therefore, a relative absence of response in this area as a function of increasing levels of alexithymia is consistent with a view of alexithymia as a disorder of affect regulation (24, 52, 60). These findings are consistent with the notion that the more an individual is capable of emotional/self-awareness and interoceptive monitoring, the less likely that he or she will be overwhelmed by intense emotional experiences and face a loss of executive control during reminders of past traumatic events and other stressful life events (61).

**Emotion regulation.** As described previously, emotional awareness has long been thought to be crucial to the regulation of affective states and is therefore often considered to be the ‘cornerstone’ of emotion regulation. Emerging research has clearly shown that the affective disturbances experienced by many PTSD subjects not only involve just fear, but also include the dysregulation of a variety of emotional states, including fear, anger, guilt and shame (62, 63). This point has been acknowledged in the newly developed provisional criteria for PTSD in the DSM-5 (http://www.dsm5.org). The term ‘emotion dysregulation’ will be used in this review to collectively refer to disturbances in a variety of emotional responses and to ‘the process by which we influence how much emotion we have and when we have it’ as outlined by Gross (64).

**Two pathways to emotion dysregulation in PTSD.** We have recently reviewed two pathways to emotion dysregulation in PTSD (65). The first of these pathways involves understanding emotion dysregulation as a result of fear conditioning through the mechanisms of stress sensitization and kindling. Over time, an individual who is sensitized to subtle reminders of traumatic and related memories may develop a general pattern of
emotion dysregulation, including anger, grief, numbing, and dissociation, in addition to a generalization of the fear response. The mechanism underlying this process is understood as being analogous to kindling, which involves the emergence of generalized seizures in response to repeated, subthreshold electrophysiological stimulation. In PTSD, similarly to kindling of seizures, the progressive augmentation and amplification of symptoms occurs over time, possibly as a result of the neural circuitry associated with the emotional memory response becoming increasingly reactive and also expanding into neighbouring neural circuits (66–68).

The second pathway emphasizes the importance of early childhood environment (69, 70). In individuals whose environment is impoverished owing to the unavailability of a responsive attachment figure or to childhood maltreatment and abuse, the emotional and arousal regulatory neural systems may not develop appropriately. This results in emotion dysregulation, which in turn results in an diminished ability to regulate physiological arousal to threatening or traumatic events. In a cycle of positive feedback, this leads to an exacerbation of emotion dysregulation, including the development of PTSD after exposure to traumatic event(s) later in life.

**Emotion dysregulation in PTSD: behavioural evidence.**

Recall of traumatic memories through trauma script-driven imagery in individuals with PTSD predominantly owing to prolonged childhood maltreatment has been shown to elicit not only fear and anxiety responses but also other negative emotions, including anger, guilt, disgust and shame (71–73). Subsequent studies using standardized stimuli confirm that emotional disturbances in PTSD related to childhood abuse are rarely restricted to fear. Studies have observed elevated self-reported negative affect in response to standardized scripts, the content of which focused on anger or safety concerns (74) or rejection–shame (75). Furthermore, structural equation modelling analyses in a sample of 215 maltreated and 206 non-maltreated children (aged 6–12 years) demonstrated that a history of neglect, physical and/or sexual abuse, multiple maltreatment subtypes, and earlier onset of maltreatment were directly related to symptoms of emotion dysregulation (76). Symptoms of emotion dysregulation are at the core of Developmental Trauma Disorder, a diagnosis suggested to reflect complex adaptations to prolonged psychological trauma in childhood (77). Further support for emotion dysregulation in PTSD comes from a recent study by Cloitre et al. (78) who specifically examined the treatment of PTSD related to childhood abuse. Results from this randomized controlled trial in adults with a history of childhood trauma showed that a phase-based emotion/interpersonal regulation-to-exposure treatment was associated with greater benefits and fewer side-effects than treatment that excluded emotion and interpersonal training, indicating the need to address emotion dysregulation in the treatment (78).

The neural correlates underlying traumatic and non-traumatic emotional processing and emotion regulation in PTSD. Studies examining brain activation in response to trauma script-driven imagery in subjects who experienced a re-experiencing/hyperarousal response to recalling their traumatic memory showed decreased responses in brain regions involved in emotion and arousal regulation, including the ventromedial prefrontal cortex, rostral anterior cingulate cortex and, in some cases, the amygdala [reviewed by Etkin and Wager (79)].

In our own studies, PTSD subjects who reported re-experiencing their traumatic events in the scanner described, for example, feeling that they were ‘being raped all over again. I could feel him holding down my hands’. Another subject stated ‘It felt like I was surrounded by smoke. I could smell and see it.’ The latter responses were usually associated with an increase in heart rate. However, it is important to note that these brain activation patterns strikingly differ from those observed in PTSD patients who exhibited a depersonalization/derealisation response to the traumatic script. For example, one subject stated ‘I was outside my body looking down at myself. It was too overwhelming to recall the traumatic memory’ while she tried to recall her traumatic memory. These dissociative patients had higher levels of brain activation in the rostral anterior cingulate cortex and dorsal anterior cingulate cortex, medial prefrontal cortex and areas in the superior and middle temporal cortices and usually did not exhibit a significant increase in heart rate during the traumatic memory recall (80–83).

Neuroimaging studies in PTSD that examined affective disturbances other than fear or that used stimuli other than exposure to traumatic memories also suggested the importance of the prefrontal/amygdala circuitry. Recall of non-traumatic sad and anxious memories in PTSD was associated with decreased activity in the rostral anterior cingulate cortex and thalamus similar to those observed during recall of traumatic memories (72). Another study using negatively valenced or aversive pictures showed that activity decreased
in the ventromedial prefrontal cortex response and the amygdala in PTSD subjects as compared to controls (84). A more recent study examined emotion regulation more directly. Non-traumatized healthy controls and subjects with a history of sexual assault with and without PTSD were asked to enhance, diminish or maintain their emotional responses to negative pictures. Non-traumatized controls were better able to downregulate their emotional response to negative pictures than were subjects with a history of sexual assault, either with or without PTSD. Successful regulation was associated with greater activity of prefrontal regions in the non-traumatized group (85).

The studies described earlier indicate a role of medial prefrontal/amygdala circuitry in both fear/traumatic and other non-traumatic, negatively valenced emotions. In general, PTSD has been shown to be associated with two different types of emotion dysregulation: i) undermodulation of affect, such as re-experiencing traumatic events, hyperarousal and anger symptoms mediated by failure of prefrontal inhibition of limbic regions, resulting in overactivation of these regions, including the amygdala, and ii) overmodulation of affect, often associated with a feeling of subjective distance from emotional experience such as during acute depersonalization, derealization and analgesia, thought to be mediated by midline prefrontal inhibition of the same limbic regions (86). A recent review proposed clinical and neurobiological evidence for a dissociative subtype of PTSD in an attempt to classify PTSD patients who show significant symptoms of depersonalization, derealization and analgesia in contrast to PTSD patients who exhibit predominantly re-experiencing and flashback-type symptoms (86). It was suggested that individuals with a dissociative subtype of PTSD may be more likely to have experienced a history of repeated interpersonal trauma occurring during crucial developmental periods.

Social cognitive neuroscience

Within the scope of social cognitive neuroscience, we will discuss two concepts we find to be especially important for an understanding of PTSD: disturbances in social emotional processing and self-referential processing. We also discuss the notion of a default mode network (DMN) in the brain and its relevance to self-referential processing and PTSD symptomatology.

Social emotional processing. Current emotion theory suggests a distinction between social and non-social emotions [reviewed by Hareli and Parkinson (87)]. Even though it is recognized that all emotional responses are potentially elicited by social stimuli or take place within a social framework (88), certain emotions are considered as necessarily social [reviewed by Hareli and Parkinson (87)]. Social emotions [e.g. pride, guilt and shame; (87)] require metacognitive processing, specifically, consideration of the self-relevance of stimuli and the appraisal of others’ thoughts and emotional states. Social emotions can be both positive and negative in valence. Positive social encounters, for example, can elicit admiration, appreciation, empathy and pride (89–91), whereas negative social encounters may provoke anger, disdain, envy, guilt or shame (87). In contrast, non-social positive emotions may be elicited by non-social stimuli such as taking a walk in the mountains alone. Non-social negative emotions can be evoked when an individual is confronted with threatening circumstances occurring outside a social context, for example a fear of heights or of certain animals (e.g. spiders).

Processing of the social dimension of emotion exerts powerful effects on brain activation [e.g. (25, 91–95); reviewed by Van Overwalle (96)]. The dorsomedial prefrontal cortex, the posterior cingulate/precuneus, the bilateral temporal poles, the right amygdala and the bilateral temporoparietal junction are brain regions that have been suggested to play a role in social cognition [reviewed by Van Overwalle (96)]. Specifically, the dorsomedial prefrontal cortex and posterior cingulate cortex/precuneus are involved in self-referential processing (25, 29, 97–101). These areas of the brain are also implicated, along with the temporoparietal junction and temporal poles, in the task of ‘mentalizing’ (attending to states of mind in oneself and others)/theory of mind (the ability to attribute mental states-beliefs, intents and desires to oneself and others and to understand that others have beliefs, desires and intentions that are different from one’s own) (27, 95, 100, 102–109). The right amygdala has also been suggested to respond to salient emotional properties that are specifically social (93).

Social emotion processing in PTSD. In PTSD related to prolonged childhood abuse, neural activation patterns to positive standardized social (rejection–shame) and non-social (fear–anxiety) emotional imagery have been examined (75). PTSD subjects showed altered brain responses in brain regions involved in higher-order social cognition (mentalizing and theory of mind), including the dorsomedial prefrontal cortex, temporal poles and amygdala particularly during positive social emo-
tional imagery. These results may have important implications for key social functions in individuals with PTSD and point to important new research questions. For example, to what degree, do deficits in this circuitry affect the ability to engage in psychotherapy? Can activation of the social emotional system in psychotherapy lead to a reversal of the brain activation patterns during social emotional processing in PTSD (110)? To what extent, do the neural networks underlying social emotions need to be intact in order to use available social support before, during and in the aftermath of trauma? Lastly, do deficits in the social emotion neural circuitry affect the ability to parent and thereby facilitate the intergenerational transmission of trauma, i.e. the emergence of psychopathology as a result of emotion processing/regulation deficits in the parent(s)?

Self-referential processing. Individuals with PTSD often exhibit disturbances in self-referential processing [(111–113); also reviewed in Ref. (114)]. Johnson et al. (115) proposed that the ability to reflect upon oneself requires a robust sense of self, which has been described as ‘a collection of schemata regarding one’s abilities, traits and attitudes that guides our behaviors, choices and social interactions’ (115). It has been well described that psychological trauma can undermine the sense of an adaptive and agentive self, challenging one’s sense of identity and life purpose [see (116, 117) for reviews]. Foa et al. (112) capture these symptoms well in the Posttraumatic Cognitions Inventory that includes items such as I feel dead inside; I will never be able to feel normal emotions again; I have permanently changed for the worse; I feel like an object, not like a person; I have no future; I don’t know myself anymore; and My life has been destroyed by the trauma. Disturbances in self-referential processing in PTSD are further apparent through symptoms of identity disturbance (118, 119), dissociation and the related experience of a fragmented sense of self (86, 119, 120) and symptoms of shame (119, 121, 122). Cloitre et al. (122) have further suggested that shame can lead to the experience of the self as inferior, bad, annihilated and/or identified with the perpetrator of abuse.

The neural correlates of self-referential processing in PTSD. Neuroimaging studies suggest that self-referential processing is partly mediated via cortical midline structures, including the medial prefrontal cortex, perigenual anterior cingulate cortex, posterior cingulate cortex, as well as the temporoparietal junction and temporal poles (96–101, 123). One model of self-referential processing proposes that the dorsal medial prefrontal cortex and the posterior cingulate cortex are involved in self-referential processing independent of whether the stimulus or task is emotionally relevant, while the ventral medial prefrontal cortex may be particularly involved in negative emotionally relevant self-referential processing tasks (123–125). A recent meta-analysis has also suggested a unique role of the medial prefrontal cortex in self-referential processing, with response within the posterior cingulate and precuneus occurring also with familiar stimuli that are not necessarily self-referential (126).

Our group has recently examined the neural underpinnings of self-referential processing in patients with PTSD as compared to healthy controls (R.L. Bluhm, P.A. Frewen, N.C. Coupland, M. Densmore, A.N. Schore, T.K. Stevens, R.W.J. Neufeld, R.A. Lanius, unpublished data). In order to examine the brain mechanisms underlying self-reflective functioning, participants were asked to respond via button-press ‘yes’ or ‘no’ to a series of statements regarding their own self-characteristics (e.g. I am a good friend; I learn new things quickly) or general facts (e.g. Paris is the capital of France) while undergoing a fMRI scan. Controls demonstrated significantly greater response within medial prefrontal cortex and posterior cingulate cortex during this self-reflective task as compared to individuals with PTSD.

In addition, a novel cognitive paradigm akin to mirror viewing has been developed to investigate self-referential processing disturbances in women with PTSD predominantly related to maltreatment experienced during childhood (113). This study population was selected because it is particularly vulnerable to negative self-referential processing following trauma, because of the relational nature of childhood abuse [see (127) for review]. The paradigm involved the collection of self-descriptiveness ratings for negatively (e.g. abandoned, unlovable, despicable, and broken) and positively valenced trait words (e.g. lovable, special and adorable) and later exposing participants to pictures of themselves while such words were spoken. Results showed that PTSD patients endorsed more negative and fewer positive trait adjectives as self-descriptive, which support repeated clinical observations that individuals with PTSD especially related to childhood trauma often experience intense negative thoughts and even self-hatred about themselves. For example, responses from individuals with PTSD to viewing their own face paired with negative adjectives included ‘I relate to the negative side’; or ‘I noticed I was agreeing with all the negative words’; or ‘It made me feel bad
about myself'. Responses from individuals with PTSD to seeing their own face paired with a positive word included 'I did not believe it'; or 'I did not mean anything'; or 'I questioned it. I did not feel the confidence'. Brain activation patterns demonstrated that healthy women showed an increased response within the perigenual anterior cingulate cortex when viewing their face and listening to positive trait adjectives, whereas women with PTSD did not show this effect. Interestingly, the perigenual anterior cingulate cortex is one of the most responsive brain regions to emotional manipulations as assessed by neuroimaging (109). It has also been linked to self-referential processing (123, 128) and is more active during negative emotional events in healthy individuals than in individuals with PTSD [see (79) for review).

In contrast to healthy controls, patients with PTSD exhibited an increased response within the right amygdala when viewing their face and responding with positive trait adjectives (113). Although response within the amygdala has more often been associated with negative emotional processing, particularly of threat-related visual stimuli (79), research also implicates the amygdala in positive emotional processing [e.g. see (129) for review]. In this study, right amygdala activation may be a sign of relatively more positive and healthy self-appraisal within women who, as a group, can be characterized by severe negative self-referential processing. It is also interesting to note that the right amygdala has been suggested to be involved in social emotion processing (see earlier section), and more positive and healthy self-appraisal may be related to better social functioning.

The aforementioned findings suggest alterations in brain functioning during self-referential processing tasks in PTSD. Future studies will need to examine the relationship between the intensely disturbed sense of self and brain activation patterns in regions involved in self-referential processing in PTSD and whether psychotherapeutic interventions that are specifically designed to improve the capacity for introspection and self-awareness can restore the functional abnormalities observed in brain regions such as the medial prefrontal cortex and the posterior cingulate cortex (130).

The default mode network. The DMN is one of the main intrinsic or resting state networks in the brain and has been suggested to play an important role in self-referential processing (131–134). The DMN is activated when individuals are engaged in stimulus-independent thought [reviewed by Buckner et al. (135)], and it is thought to aid in serving to consolidate, stabilize and set the context for future information processing (135). A recent meta-analysis demonstrated that autobiographic memory recall, theory of mind tasks and prospection/thinking about the future activate the DMN (136, 137). This may indicate that these functions bear a direct relationship to the connectivity of this network.

The DMN includes several brain regions that have been associated with self-referential processing (138), including the medial prefrontal cortex, posterior cingulate cortex in addition to midline parietal structures, medial and lateral temporal lobes and lateral parietal regions. The regions comprising this network were originally thought to be involved in the maintenance of a ‘default mode’ of brain functioning that occurred in the absence of task-related processes as the regions of this network had a high level of metabolic activity during rest and showed consistent decreases in activity level during the performance of cognitive tasks (139). Later studies showed that these brain regions exhibit positive correlations with each other (140, 141) and negatively correlate with brain areas involved in many cognitive tasks (140, 142). These DMN areas have anatomical connections that may explain their functional connectivity during rest (143, 144).

Given that PTSD has been associated with deficits in self-referential processing, as described earlier, and has been associated with altered activation in areas associated with the default network (e.g. medial prefrontal cortex, anterior cingulate and posterior cingulate cortex) across a variety of paradigms (71, 145–150), our group chose to embark on a study that examined the integrity of this network in PTSD. In patients with chronic PTSD owing to prolonged childhood abuse, significantly reduced resting state connectivity within the DMN was demonstrated (151) (see Fig. 2). The PTSD group showed diminished connectivity between the posterior cingulate seed region and the medial prefrontal cortex, right superior frontal gyrus and left thalamus. Furthermore, the connectivity of the medial prefrontal seed region was strictly limited to adjacent areas in the medial prefrontal cortex. Because autobiographic memory recall has previously been shown to bear a direct relationship to the connectivity of the DMN (152), alterations in brain activation in PTSD during autobiographic memory recall as observed during the script-driven imagery symptom provocation studies may be one underlying mechanism contributing to the altered DMN.
connectivity observed. We have also recently suggested that in adult patients with PTSD related to childhood abuse, DMN connectivity resembles that observed in children aged 7–9, possibly indicating interference with the maturation process because of the toxic effects of stress hormones on the myelination of the corpus callosum (153).

Daniels et al. (154) also studied the ability of patients with chronic PTSD to engage and disengage from a working memory task, as deficits in working memory performance have been well documented in PTSD (155–157). It has recently been shown that failure to suppress activity in the DMN can result in poorer working memory performance (158). In the light of these findings, it is interesting that PTSD subjects showed sustained connectivity in the DMN during the working memory task. In contrast, the control group showed significantly stronger connectivity with areas implicated in salience and executive control, including the right inferior frontal gyrus and the right inferior parietal lobule during the working memory task as compared to the resting condition. These differing patterns of connectivity suggest difficulties with task-induced switches, i.e. engaging and disengaging the DMN, potentially causing the observed reductions in working memory performance as often seen in individuals with PTSD.

Future studies will have to investigate the causal relationship between specific PTSD symptoms experienced during the resting scan and connectivity of the DMN as well as to further elucidate the mechanism underlying switching between resting and cognitive and emotional networks (159). In addition, the relationship between self-referential processing and the altered sense of self in relation to DMN connectivity will have to be explored. We hypothesize that the negatively valenced and often fragmented sense of self that is often experienced by individuals with highly dissociative PTSD will be related to the degree of anterior–posterior integration of the DMN.

**SCAN-informed treatment applications.** As described earlier, a number of recent studies, falling under the scope of SCAN, suggest that patients with PTSD show problems in emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing. Interestingly, a core set of brain regions appear to partly mediate these collective psychological functions, most notably the cortical midline structures, the amygdala, the insula, posterior parietal cortex and temporal poles, suggesting that problems in one area (e.g. emotional awareness) might relate to difficulties in another (e.g. self-referential processing). There is also evidence that these regions are implicated in the pathophysiology of PTSD. In this section, we consider the significance of these findings for the treatment of PTSD.

**Emotional/self-awareness.** Exposure-based treatments of PTSD (160) involve repeated imaginal and in vivo exposure to trauma-related stimuli. Although these treatments have strong empirical support, in order to be successful, it is important for patients to be able to fully engage emotionally with the traumatic material. During exposure

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**Fig. 2.** (a) Default mode network (DMN) connectivity in control (top panel) and post-traumatic stress disorder (PTSD) subjects (bottom panel). Areas of correlation with posterior cingulate/precuneus in healthy comparison subjects ($n = 15$) and in patients with PTSD ($n = 17$), thresholded at $P < 0.05$, corrected using false discovery rate correction. (b) DMN connectivity between group comparison. Areas in which correlation with the posterior cingulate/precuneus is stronger in healthy control subjects ($n = 15$) than in patients with PTSD ($n = 17$), thresholded at $P < 0.05$, corrected using false discovery rate correction. Figure originally published in Bluhm et al. (151) and reprinted with permission.
therapy, patients are usually asked to recall details of their traumatic experience while describing them in the present tense. Exposure sessions are thus designed to overcome the avoidance of such stimuli by providing a safe context in which patients can fully engage with both trauma-related and ‘corrective’ (safety) information. In this way, exposure treatment is designed to overcome and reduce avoidance symptoms, enhance affect management, diminish anxiety and fear associated with the traumatic material and facilitate cognitive restructuring of trauma-related memories. In turn, this should bring about the reduction of re-experiencing and hyperarousal symptoms and, ultimately, lead to a significant reduction of symptoms or elimination of the disorder itself (161).

Teaching PTSD patients with impairments in emotional awareness to identify their affective feelings early on in treatment may make exposure-based treatments more efficient (78, 86, 162, 163) because such therapies are dependent on a patient’s judgment of their internal affect and as well as a reliable report of their symptom presentations. In addition, such treatments may be less effective in the early stages of treatment for patients who are unable to modulate, regulate and engage in the intense affective experiences that may be elicited by exposure-based therapies. Interoceptive awareness training to help patients become aware of and describe bodily sensations and their relationship to emotions is therefore often a crucial part of the first stage of trauma treatment. Such awareness can prevent individuals from entering severe hypo- or hyperarousal states that can prevent emotional and cognitive processing.

During this phase of treatment, patients have to develop an awareness and language that helps them identify bodily sensations and how different bodily sensations relate to different emotional experiences. For example, for one person, tension in the jaw in combination with a feeling of tightness in the throat may correlate with sadness, while in another individual, sadness may correlate with tightness in the stomach and a feeling of heaviness in the chest. By developing emotional awareness, patients can then learn to identify precursors to extreme emotional states and learn to intervene before these states become too intense and overwhelming. Once a patient has mastered these skills, they can be applied during the exposure-based treatments, thereby preventing extreme emotional states that preclude optimal emotional engagement during exposure therapy.

Emotional/interoceptive awareness training has been incorporated into several forms of psychotherapy for PTSD. Dialectical Behaviour Therapy (164, 165), skills training in affective and interpersonal regulation (STAIR) (122) and emotion focused therapy (166) all include a focus on increasing patients’ capacity for introspection and self-awareness, particularly with regard to emotional experiences. In addition, mindfulness-based approaches such as those outlined by Kabat-Zinn and Siegel (167, 168) can be helpful to increase the capacity for emotional awareness. In particular, teaching patients to become aware of physical sensations through engaging in body scans at a pace that feels safe for them can help to increase their introspective ability. Yoga has also been suggested to be effective in treating PTSD, partially by increasing one’s interoceptive ability (169, 170).

Emotion regulation and social emotions. In recognition of complex emotion dysregulation, social and self-dysfunction in patients with chronic PTSD related to childhood abuse, Cloitre et al. (162) developed an empirically validated stage-oriented intervention for PTSD related to childhood abuse. STAIR is a stage-oriented treatment model that uses skills training in emotion and self-regulation and interpersonal effectiveness prior to engaging in exposure-based therapy. Results published by Cloitre et al. (78) support the suggestion that, before beginning exposure therapy, patients need to develop mood regulation skills, allowing them to better identify and modify disordered attachment schemas learned in childhood and enhancing their competence in social interactions.

Early stages of STAIR teach patients how to enhance their capacity to regulate dysregulated emotional states through cognitive strategies (attention shifting, positive self-statements, positive imagery), behavioural strategies (time out, replacement behaviours, engaging in pleasurable activities) and enhancing distress tolerance skills.

Enhancing emotion regulation skills prior to engaging in exposure-based treatment may be particularly important in individuals with emotion regulation problems involving significant symptoms of emotional overmodulation, such as dissociative (e.g. depersonalization, derealization and analgesia) symptoms (86). Such symptoms can reduce or prevent emotional engagement with trauma-related information and thereby reduce treatment effectiveness (171). Further evidence for the latter stems from an investigation by Hagenaars et al. (172) who reported a differential outcome for PTSD patients with high vs. low dissociative symptomatology. Whereas only 10\% of PTSD patients with low dissociative symptoms still met PTSD criteria after a trial of exposure therapy, 69\% of PTSD patients
with high levels of dissociation continued to meet PTSD criteria at follow-up. Therefore, it is crucial, before commencing exposure-based treatments, to assess the levels of emotional overmodulation and provide interventions to reduce such symptomatic responses to trauma-related stimuli (86).

Following the emotion regulation component of STAIR, patients begin interpersonal regulation training that involves understanding interpersonal schemas, changing relationship patterns through role-play using appropriate assertiveness skills and an enhanced understanding of power balances in relationships. In addition, providing a corrective interpersonal experience through a therapeutic relationship can provide a secure base (173), thereby facilitating trust and effective communication is also an important aspect of facilitating enhanced social competence (122, 164, 166).

In the later stages of STAIR treatment, exposure therapy and narrative story telling is designed to help patients reach a stage of resolution of their feelings of shame and grief. This intervention involves both telling the story of the trauma and a related meaning analysis in order to help patients understand who they have come to be as a result of what they have experienced. This understanding may decrease feelings of shame and loss, as well as related experiences of the self as inferior, bad, annihilated, fragmented and/or identified with the perpetrator (122). The resolution of the experience of the self as an extremely negative entity will also likely result in significantly increased social functioning. It is often the experience of the self as bad and inferior that perpetuates individuals being in abusive relationships as they may feel like they deserve to be punished or treated without respect. With regard to the latter, this part of STAIR also involves role-play during which emotions and actions are integrated, thus providing individuals the experience of learning about who they can learn to become through action.

Self-referential processing. Self-referential processing is most closely related to the concept of mentalizing – attending to states of mind in oneself and others (174), and mentalization-based treatments have been shown to be effective in individuals who have suffered from disrupted early attachments and childhood abuse (175). Increased self-referential processing/mentalizing has been suggested to enhance emotion regulation skills as well as to facilitate the process of exposure to traumatic memories. As Jon Allen states: ‘We work clinically [using mentalization-based therapy] with patients who are prone to be swept away by emotion, carried along into impulsive action, without any felt sense of self. We encourage such patients to push a metaphorical pause button by mentalizing’ [(174), p. 11]. Moreover, Allen has stressed that the alternative to traumatic re-enactments is mentalizing, that is developing the capacity to reflect on the relationship between triggering events in the current relationship and previous traumatic experiences. Allen has also suggested that encouraging mentalizing while recalling vivid traumatic memories as is done during classic desensitization procedures allows individuals to take a more active stance in the recall of their traumatic memories, thereby creating an increased mastery experience.

Self-referential processing is also a key component of cognitive restructuring. The latter involves identifying and modifying catastrophic and unrealistic interpretations of one’s traumatic event and future expectations, thereby leading to cognitive schema that will decrease the outcome of psychopathological states with a resulting reduction in trauma-related symptomatology (176). In fact, evidence has shown that optimal treatment outcomes in PTSD are achieved when cognitive restructuring is combined with exposure therapy (176). Cognitive restructuring is also an important component of cognitive processing therapy (177). These findings therefore provide further evidence for the importance of self-reflection in the treatment of traumatized individuals.

Discussion

In conclusion, researchers and clinicians with an interest in complex PTSD have become increasingly aware of the potential effects of early life trauma on neurobiology, symptomatology and prognosis. Yet much remains to be done in developing a concept of PTSD that can account for the far-reaching effects of chronic traumatization. In this review, we have examined the relevance of the SCAN paradigm for an understanding of the psychology and neurobiology of complex PTSD and its effective treatment. We have suggested that SCAN offers a novel theoretical paradigm for understanding psychological trauma and its numerous clinical outcomes, most notably problems in emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing. A core set of brain regions appear to mediate these collective psychological functions, most notably the cortical midline structures, the amygdala, the insula, posterior parietal cortex and temporal poles, suggesting that problems in one area (e.g. emotional awareness) may relate to difficulties in another (e.g. self-referential
processing). We have further suggested, drawing on clinical research, that the experiences of individuals with PTSD related to chronic trauma often reflect impairments in many of these abilities. It is therefore crucial that the assessment and treatment of individuals with chronic PTSD not only addresses the traumatic memories but also takes a SCAN-informed approach that focuses on the underlying deficits in emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing. Finally, treatment outcome studies examining the effectiveness of treatments for complex PTSD will not only need to focus on PTSD symptoms but also ought to assess the effect of the intervention on emotional/self-awareness, emotion regulation, social emotional processing and self-referential processing.

Future research directions

Future studies in complex PTSD should further evaluate social cognitive processes and their shared and non-shared neural correlates, including empathy (178, 179, 180), moral reasoning, theory of mind and emotion comprehension in order to broaden our understanding of the potential deficits in these functions as a result of chronic traumatization. Moreover, the relationship between declines in basic cognitive processes, including working memory, executive functioning and anterograde memory, on the one hand, and deficits in higher-level affective processes such as conceptual emotional awareness and self-referential processes, on the other, will need to be explored. It will also be crucial to develop a detailed understanding of the relationship between specific stress-related symptoms (e.g. emotional numbing) and their precise effects on social cognition. For example, we would hypothesize that significant symptoms of numbing/emotional detachment would substantially interfere with both the capacity for empathy and theory of mind/mentalizing. Research will also have to address to what degree deficits in the neural circuitry underlying social cognition affect the ability to engage in psychotherapy and to what extent do the neural networks underlying social emotions need to be intact in order to utilize social support before, during and in the aftermath of trauma. Furthermore, it will be important to determine whether psychotherapeutic interventions that specifically target interpersonal dysfunction and the capacity for introspection and self-awareness can restore the functional abnormalities observed in the neural circuitry underlying these processes. In addition, the relationship between self-referential processing and the intensely negative and fragmented sense of self often observed in complex PTSD, on the one hand, and DMN connectivity, on the other, deserves exploration. Lastly, it will be important to determine whether deficits in the neural circuitry underlying self-reflection and social cognition affect the ability to parent and thereby facilitate the intergenerational transmission of trauma.

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