



**The Role of Social Cognition in Post-Traumatic Stress Disorder (PTSD): A  
Structured Literature Review**

Master Thesis Neuropsychology

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## Abstract

Post-traumatic stress disorder (PTSD) is a psychiatric disorder which can significantly impair individuals in their daily functioning. Not every person who experiences a traumatic event however develops pathology. Research supports the availability of social support, individual adaptive coping styles and effective implicit emotion regulation as resilience factors against PTSD development. Risk factors include specific features of an event, pre-existing attributes and lack of post-traumatic social support. Furthermore, social cognitive impairments are common and debilitating manifestations in PTSD patients, leading to reduced social support. The acceptance of social support is known to be of great importance for the processing of the traumatic experience. Thus, exploring the association between PTSD and different social cognitive domains seems to be worthwhile. The current literature review mainly considers cognitive empathy processes, including theory of mind (ToM), emotion recognition and social perception, but attributional styles are also discussed. Although its limitations, insights in the neural correlates of social cognitive processes may be a valuable, non-invasive manner in trying to understand dysregulated emotional processing in PTSD patients. Considering brain regions, the amygdala, hippocampus, and prefrontal cortex appear to play an important role in the pathophysiology of PTSD. No consensus yet is reached whether amygdala activity is heightened in PTSD or not, which might be attributed to comorbidity problems and heterogeneity of study designs. However, decreased hippocampal volume is repeatedly shown. FMRI studies also show reduced hippocampal activation. Lastly, studies often show less activation or even deactivation in ventral medial PFC regions, including the anterior cingulate cortex. However, other studies have undermined this. A mutual consensus whether vmPFC activity is decreased in PTSD is therefore not yet agreed on. In conclusion, social cognition plays an important role in PTSD. Systematic assessment of social cognition in clinical practice is of the essence, in order to enhance treatment decision-making and outcomes, to ultimately provide the best care for the patient.

*Keywords:* PTSD · Risk/Resilience · Social Cognition · Cognitive Empathy · Neuroimaging

## **Introduction**

Post-traumatic stress disorder (PTSD) is a psychiatric disorder that follows from exposure to a traumatic event (Couette, Mouchabac, Bourla, Nuss, & Ferrer, 2019). The main symptoms are persistence of intrusive stressors associated with the traumatic event (e.g., distressing memories or dreams), persistent avoidance of stimuli associated with the traumatic event, negative alteration of cognition and mood, an on-going feeling of imminent threat, hypervigilance and disturbance of sleep. In the newest version of the DSM (DSM-V), PTSD is no longer described as a subtype of anxiety disorders, but as a new category; Trauma- and Stressor-Related Disorders (American Psychiatric Association, 2013). Although most people will experience a traumatic event during their lifetime, estimates are that only 6 to 10 percent are truly diagnosed with PTSD (Stevens & Jovanovic, 2019). Thus, there appear to be resilience- and risk factors that play a role in determining whether or not someone develops pathology after experiencing a traumatic event.

## **Resilience- and risk factors**

As noted, not every person who experiences a traumatic event will develop pathology. Resilience levels vary between individuals; some can adapt more effectively in the face of stress and adversity. It is worth noting that resilience is a multifaceted concept which may be influenced by several (epi)genetic, psychosocial, developmental and neurochemical factors along with neural circuits and molecular pathways that underlie the development of resilience (Horn, Charney, & Feder, 2016). The current review will focus on psychosocial factors, where three essential components can be identified: social support, coping strategies and emotion regulation.

First of all, social support is associated with higher resilience levels. High levels of social support has been linked to better health outcomes and higher psychological well-being following stress (Horn, Charney, & Feder, 2016). Contrary, low levels of social support has been linked to higher PTSD risk in soldiers (Tsai, El-Gabalawy, Sledge, Southwick, & Pietrzak, 2015), hurricane-exposed adults (Kilpatrick et al., 2007), and earthquake survivors (Feder et al., 2013). Social support might enhance resilience by reducing loneliness and has been linked to decreased threat appraisal related to illness (Horn, Charney, & Feder, 2016).

Besides social support, individual coping strategies also appear to be of importance in the aftermath of trauma. Thompson, Fiorillo, Rothbaum, Ressler, and Michopoulos (2018) examined the relationship between resilience, coping strategies, and the development of PTSD symptoms in a longitudinal study. They found that resilience was predictive of increased social

support seeking, an active coping strategy, and inversely related to social withdrawal, an avoidant coping strategy. Thus, resilient individuals have been found to employ greater amounts of active coping, which in turn is negatively associated with PTSD development (Li, & Nishikawa, 2012). Furthermore, the use of humour is another adaptive coping strategy which seems to alleviate tension and attract social support, as shown in studies with war veterans, firefighters, and terminally ill patients (Sliter, Kale, & Yuan, 2014).

Lastly, emotion regulation is another protective factor linked to psychological resilience. Effective implicit emotion regulation supports cognitive flexibility, cognitive control, and adaptive coping, which in turn mediates for PTSD resilience (Horn & Feder, 2018). Cognitive reappraisal, an emotion regulation strategy involving the ability to monitor negative thoughts and replace them with more positive ones, is often used by resilient individuals (McRae, Ciesielski, & Gross, 2012). In trauma-exposed samples, those who made use of positive cognitive reappraisals had lower reported stress-related and anxiety symptoms, in contrast to individuals who had to suppress their expressions (Moore, Zoellner, & Mollenholt, 2008). Hence, enhancing cognitive reappraisal skills could help in building resilience in PTSD patients.

Apart from resilience factors, which may act as a safeguard in the development of PTSD, risk factors can be identified as well. First of all, it should be emphasized that there is growing evidence that associations between risk factors and PTSD may vary depending on the particular study population or other study attributes (Vogt, King, & King, 2014). These findings suggest that PTSD is unlikely to develop based on a single cause, but should be approached more holistic, including multiple causal pathways. Nevertheless, it is widely agreed upon that a key role is assigned to psychosocial factors. These factors may be categorized into features of the event, the victim's pre-existing attributes or experiences and posttraumatic circumstances (Vogt et al., 2014).

First, considering features of the event, Brewin, Andrews, and Valentine (2000) conducted a meta-analysis and found a modest association between event severity and individuals developing PTSD. Additionally, Ozer, Best, Lipsey, and Weiss (2003) found that PTSD was associated with the degree of life threat experienced during the event. Also, events that involve subjective distress, events that include injury or events where the subject is directly involved rather than a witness were associated with PTSD risk (Vogt et al., 2014). Thus, it may be concluded that (subjective) intensity is correlated with higher risk of developing PTSD. Next, regarding pre-existing attributes, a meta-analysis by Brewin et al. (2000) found modest risk effects on the following attributes: younger age at the time of the traumatic event, female

gender, lower SES/education/intelligence and belonging to an ethnic minority group. As far as pre-existing experiences, individuals with a history of childhood trauma are more likely to be exposed to traumatic events in adulthood and subsequently develop PTSD (Spinhoven, Penninx, van Hemert, de Rooij, & Elzinga, 2014). Post-traumatic circumstances can be divided into individual factors and environmental factors. Considering individual factors, comorbid psychological obstacles and maladaptive coping styles appear to be distinct risk factors (Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). Environmental factors mostly include social ecological factors, which for instance consist of family of origin, poverty rate and social support (DiGangi et al., 2013).

The acceptance of social support is known to be an important aspect for the processing of traumatic experiences (Schnurr, Lunney, & Sengupta, 2004). Yet, PTSD patients often show difficulties in the maintenance of relationships and social interactions (Cook, Riggs, Thompson, Coyne, & Sheikh, 2004). Often, relationships with close relatives and intimate partners are especially challenging and conflicted after the traumatic event (Riggs, Byrne, Weathers, & Litz, 1998). Whilst the role of social support in traumatized individuals is well-established, it is less well understood if and how PTSD in turn influences the use of social support (Janssen et al., 2022). It might be plausible that symptoms of PTSD, such as a negative shift in mood and cognition, influence the ability to perceive, interpret and respond to others. These abilities are collectively falling under the term ‘social cognition’. A valuable question therefore will be whether traumatic events not only lead to PTSD symptomatology, but whether they also contribute to changes in facets of social cognition which are important for successful social interactions. These interactions facilitate social support and consequently are important for the patient’s recovery and quality of life (Schnurr, Lunney, & Sengupta, 2004). Hence, it is essential to better understand the association between PTSD and different social cognitive domains – which is the primary aim of this research paper.

### **Social cognition in PTSD**

Social cognition refers to the ability to perceive, process, and understand social information and to respond adequately (Couette et al., 2020). One component of social cognition, cognitive empathy, might be of particular importance. Cognitive empathy refers to the capability to understand and explain the others’ mental state (Nietlisbach, Maercker, Rösler, & Haker, 2010). Impairment in cognitive empathic abilities can lead to poorer social interactions, subsequently accounting for less social support, which is an important element in the recovery of PTSD symptoms (Gros et al., 2016). Therefore, it will be discussed how three domains of cognitive

empathy, theory of mind (ToM), emotion recognition and social perception (Green et al., 2008), are modified in PTSD patients. Lastly, the effects of one's attributional style will be debated as well.

### *Theory of mind*

Theory of mind (ToM) describes the interpretation of behaviour of others based on their mental states (Janssen et al., 2022). It refers to our ability to make inferences regarding others' beliefs and is synonymously called 'mind-reading' or 'mentalizing' (Harari, Shamay-Tsoory, Ravid, & Levkovitz, 2010). At around four years, humans develop the ability to mentalize (Nietlisbach & Maercker, 2009). At this age, children begin to realize that the same events or situations may be presented in different, or even contradictory ways to others. Although scientific understanding of children's ToM abilities is growing, relatively little is known about the development of ToM in adults (Nietlisbach & Maercker, 2009). No consensus has been reached yet on how, and if at all, ToM capacities change over time (Brüne & Brüne-Cohrs, 2006). ToM abilities are often tested with the 'Sally and Anne' test (Wimmer & Perner, 1983), recognition of 'faux pas' situations (Brüne & Brüne-Cohrs, 2006), and a 'Reading the Mind in the Eyes' (RMET) test (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). The ToM deficit hypothesis has mostly been applied to schizophrenia, autism and personality disorders, specifically in psychopathy (Brüne, 2001). Its relation to other psychopathology and psychiatric disorders remains less clear (Brüne & Brüne-Cohrs, 2006).

Little research has been conducted on the effects of ToM in the context of PTSD. Four studies (Nietlisbach, Maercker, Rossler, & Haker, 2010; Mazza et al., 2012; Mazza et al., 2015; Palgi, Klein, & Shamay-Tsoory, 2017) were found to have specifically explored the ToM component in PTSD. First, the study of Nietlisbach et al. (2010) compared empathic abilities in PTSD individuals compared to non-traumatized controls. Using the RMET test, no difference in scores on mental stage matching were found, although they did observe a positive correlation between scores and time since trauma, suggesting that a preliminary deficit may increase over time. Participants also took the 'Faux pas' test, where they had to read short stories in which a situation where one person unintentionally hurts another person (faux pas) was made. Participants had to identify these and explain why it was a faux-pas and how the victim felt. No difference between controls was found. Mazza et al. (2012) administered an emotion attribution task where participants had to read stories describing an emotional situation and identify what the main protagonist felt. PTSD patients had difficulty properly describing an emotional situation, especially in situations of sadness, embarrassment or happiness. They also used a

strange stories task, where participants had to read short stories depicting social situations including lie, white lie, joke, pretend, misunderstanding, and contrary emotion. Patients' comprehension of the situation ('Was it true, what X said?') and a further explanation of the strange story were assessed. Patients with PTSD performed like controls on this task. In a follow up (Mazza et al., 2015), a modified version of the Multifaceted Empathy Test (MET) was used, where participants had to assume the valence of the mental state of a person shown in a picture. They found that patients with PTSD displayed disruptions on the affective dimension, but not on the cognitive one. Lastly, Palgi et al. (2017) used a biological motion task where participants had to attribute an emotional state to a human body expressing anger, disgust, fear, sadness, or happiness, but with no facial clues. It was found that PTSD patients were less accurate than controls in recognizing emotions. Participants also engaged in a Yoni's task, in which a cartoon face looks at a stimulus and smiled in some trials, indicating that he likes something. Participants had to point to the correct answer, which is the image looked at by the cartoon, based on a sentence at the top of the screen and the cartoon's cues. Patients with PTSD were less accurate than controls.

### *Emotion recognition*

Another part of the cognitive empathy spectrum is emotion recognition, which explains the identification and recognition of emotional states from social stimuli (Janssen et al., 2022). Overall, research has shown that individuals with PTSD show abnormalities in higher-order emotional processes, including emotion recognition (Passardi, Peyk, Rufer, Wingenbach, & Pfaltz, 2019). When the ability to recognize these expressions is diminished, which is the case with PTSD, factual knowledge that is associated with certain types of facial expressions is lost. (Adolphs, Damasio, Tranel & Damasio, 1996). Deficits in facial emotion recognition have been reported, especially for the emotions fear and sadness.

To illustrate, a study by Poljac, Montagne, and De Haan (2011) showed a reduced accuracy and a decreased sensitivity for these emotions in PTSD patients compared to healthy controls. Furthermore, a study by Ashley and Swick (2019) have extended this knowledge to angry facial expressions. Their results suggest that PTSD patients may be more vigilant for or primed to respond to the appearance of angry faces. Furthermore, a study by Castro-Vale, Severo, & Carvalho (2020) found an impaired ability to identify facial expressions of emotions, independent of the type of emotion in a sample of war veterans with lifetime PTSD. Strikingly, their results show that also their offspring showed a reduced ability to identify facial emotional expression both in general, and specifically for the emotions of disgust and happiness. Thus,

these results suggest that normal emotion regulation is disrupted, which may act as a mediator for troubled social interactions. This consequently may lead to less social support seeking, resulting in reduced resilience and a decreased quality of life.

### *Social perception*

Social perception, another cognitive empathy process, is considered the recognition of social rules, regulations and goals (Janssen et al., 2022). Research shows that social perception is often disturbed in PTSD. Here, a negative attention bias towards social threat might hinder the interpretation and processing of several contextual cues (Bomyea, Johnson, & Lang, 2017; Naim et al., 2015), which is believed to be essential in social perception. This is relevant, since a lack of perceived social support is shown to be related to symptom severity (Hofman, Hahn, Tirabassi, & Gaher, 2016; Jankowski et al., 2004). A recent study by Janssen et al. (2022) have conducted a meta-analysis reviewing the effects of PTSD on different domains of social cognition, including social perception. They indicated that only a single study have explored this subject, which is the study by Nazarov et al. (2014). Compared with healthy controls, women with PTSD related to childhood abuse were slower to identify prosodic cues conveying fear, happiness, and sadness. Thus, they have found a diminished social perceptive ability in PTSD. However, since this domain only includes one study, the results must be interpreted with caution. Nevertheless, their findings indicate that problems in perceiving and understanding one's social context is often experienced in traumatized individuals, emphasizing the importance of research on social perception and PTSD.

### *Attributional style*

One's attributional style illustrates the explanation of social situations (Janssen et al., 2022). Attributional style measures aim to determine which explanation the participant will favour when explaining a certain (social) event (Plana, Lavoie, Battaglia, & Achim, 2014). These explanations can be negative, positive or ambiguous. Subsequently, different kinds of biases can be evaluated depending on the type of event presented in the task. For instance, a negativity bias can be inferred based upon how often a participant favours negative causes for ambiguous events (Franklin, Huppert, Langner, Leiber, & Foa, 2005). No difference in attributional style between a control group and PTSD group was discovered in studies that mainly examined causal explanations of *general* events (Janssen et al. 2022). However, when investigating *trauma-specific* causal attributions, results differed (Gonzalo, Kleim, Donaldson, Moorey, & Ehlers, 2012). Compared to a broad dispositional attribution group, trauma-specific attributions were more predictive of PTSD symptoms (Gray, Pumphrey, & Lombardo, 2003). Especially



global, stable and internal attributions of particular traumatic events were linked with PTSD symptoms (e.g., Gray et al., 2003). For instance, internally focused, self-blame attributions after sexual assault were associated with PTSD symptom development (Berman, Assaf, Tarrasch, & Joel, 2018; Najdowski & Ullman, 2009). Hence, only a single study has reported a negative attributional bias in PTSD with a large effect size, as this was the only one using a PTSD specific attributional style measure (Boffa, Norr, Tock, Amir, & Schmidt, 2018).

### **Neural mechanisms in PTSD**

Insights in the neural correlates of social cognitive processes may be a valuable, non-invasive manner in trying to understand dysregulated emotional processing in PTSD patients. Nevertheless, PTSD is also often comorbid with other disorders (Henigsberg, Kalember, Petrović, & Šečić, 2019). Therefore, the distinction between cognitive processes in PTSD and other psychopathology is challenging. Though, studies in PTSD have made an effortful contribution and found that, considering brain regions, the amygdala, hippocampus, and prefrontal cortex are all important structures in the pathophysiology of PTSD.

#### *Amygdala*

Animal studies have showed that the amygdala is a core structure involved in the regulation of stress and fear, and increased amygdala activity is ought to play an important role in a wide variety of anxiety disorders (Simmons & Matthews, 2012). Findings on amygdala activity in PTSD are mixed, with studies pointing to increased amygdala activity, and studies that show no difference in amygdala activity with healthy controls (Henigsberg et al. 2019). To illustrate, an fMRI study by St. Jacques, Botzung, Miles, & Rubin (2011) showed that PTSD patients have higher right-amygdala activation during the construction of negatively intense autobiographical memories compared to controls. Etkin and Wager (2007)'s meta-analysis showed an increased amygdala activity in PTSD cases; however, the activation was smaller for the PTSD group than for the social anxiety and specific phobia cases. The activity level was found to relate to symptom severity, with different and more complex emotional dysregulation observed in individuals with PTSD. While others also have shown that activation of the amygdala positively correlates with symptoms of PTSD (e.g., Shin et al., 2004), however, a few studies have not found/ found limited amygdala activation during symptomatic conditions in PTSD.

For example, Stevens et al. (2017) examined associations between data-driven symptom dimensions and episodic memory formation. Five symptom dimensions were defined, re-experiencing, somatic symptoms, reflecting negative effect, hyperarousal and numbing. Only

re-experiencing was associated with an increased amygdala activation. Contrary, negative affect predicted decreased activation for recalled scenes. Additionally, Britton, Phan, Taylor, Fig, & Liberzon (2005) found that there was no increased amygdala activation among the PTSD patients compared to veterans without PTSD during the exposure of subjects to emotional aversive stimulus. The control group, contrary, showed increased left amygdala activation in response to aversive stimuli. Difference in results might be assigned to the heterogeneity of study designs, outcome measures and sample sizes. Also, as mentioned above, it is important to emphasize that hyperactivity of amygdala is also associated with other anxiety disorders, and therefore a solid distinction between cognitive abnormalities in PTSD and other psychopathology is difficult to make.

### *Hippocampus*

The hippocampal structure has a crucial role in the control of stress response, declarative memory and fear conditioning (Henigsberg et al., 2019). Stress and trauma induce synaptic degeneration, and neuronal atrophy in the hippocampus (Popoli, Yan, McEwen, & Sanacora, 2011), and many studies have shown decreased hippocampal volume in PTSD (e.g., Sussman, Pang, Jetly, Dunkley, & Taylor, 2016). To illustrate, a research group called “ENIGMA” (Enhancing Neuroimaging Genetics through Meta-Analysis), which works within the field of PTSD, compared hippocampal structural volumes between PTSD patients and control subjects in the largest PTSD neuroimaging study so far. The study included data from 1868 participants and showed the connection between PTSD and lowered hippocampal volume (Logue et al., 2018). Moreover, in a comparative study by Apfel et al. (2011), smaller hippocampal volumes were found in people with chronic PTSD compared to acute PTSD and depressive patients. Nevertheless, such research is often criticised by the fact that it is still not possible to claim with certainty whether decreased hippocampal volume can be considered a cause or a result of PTSD. There is also always a possibility that both are correct, so a diminished hippocampal volume represents an increased risk for PTSD, whereas it could also be a consequence of trauma exposure.

Regarding hippocampal activity, a series of fMRI studies reported reduced activation of the hippocampus during both conditioning and extinction learning in PTSD (Shvil, Rusch, Sullivan, & Neria, 2013; Rauch, Shin, & Phelps, 2006; Sripada, King, Garfinkel, Wang, Sripada, Welsh, & Liberzon, 2012). In addition, a meta-analysis by Etkin and Wager (2007) of emotional processing studies reached similar conclusions, finding that patients with PTSD showed hypoactivation of different regions, including the anterior hippocampus, when

compared with control subjects (with social anxiety disorder or specific phobia) during a variety of emotional processing tasks. Hence, deviant hippocampal activity seems to play a part in PTSD, however, the question whether this is a cause or an effect of PTSD remains unanswered.

#### *Prefrontal cortex*

Functional neuroimaging studies have typically reported less activation or even deactivation in ventral medial PFC regions, including the anterior cingulate cortex (ACC), during traumatic script-driven imagery in PTSD (Hughes & Shin, 2011). These regions have been frequently associated with emotional arousal (Taylor, Phan, Decker, & Liberzon, 2003), emotional conflict (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006), anticipation of aversive events (Kalisch et al., 2005) and autonomic activity (Critchley, 2005). To illustrate, Shin et al. (1999) found less right anterior cingulate cortex (rACC) activation in response to traumatic versus neutral scripts in survivors of childhood sexual abuse with PTSD versus those without. Furthermore, Lindauer et al. (2004) found lower activation in the medial frontal gyrus in response to traumatic versus neutral scripts in trauma-exposed police officers with PTSD compared to those without. Dorsomedial prefrontal cortex activation was also found to be negatively correlated with PTSD symptom severity (Hughes & Shin, 2011).

Research have also probed ventromedial prefrontal cortex (vmPFC) function in PTSD using trauma-related stimuli other than scripts. By using images, Yang, Wu, Hsu, & Ker (2004) presented trauma-related and neutral pictures to adolescents with PTSD and trauma-exposed control subjects and found a decreased ACC activation in the PTSD group when viewing trauma-related pictures compared to neutral pictures. The same results were found by Hou et al. (2007) in mining accident survivors with PTSD.

Decreased vmPFC activation have been demonstrated on emotional stimuli as well. Using facial stimuli, Kim and colleagues used a same/different judgment task with task-irrelevant neutral and emotional face distracters on subway fire survivors with PTSD. In the PTSD group, a significantly diminished rACC activation was found compared to the control group in the fearful versus neutral face condition. Also, using non-facial emotional stimuli resulted in less ACC activity. Lanius et al. (2003) found decreased ACC activation in PTSD patients in response to scripts describing anxious and sad personal events.

Even though many studies have found evidence opting that regions of the vmPFC are hyporesponsive in PTSD, several other studies have not. For example, Bonne et al. (2003) found no difference in resting ACC perfusion between PTSD, trauma-exposed non-PTSD, and trauma-unexposed groups. Zubieta et al. (1999) used SPECT with combat sounds and found

increased medial prefrontal cortex (mPFC) activation in combat PTSD patients relative to combat and healthy control subjects. Bryant et al. (2008) found greater vmPFC activation in response to unconsciously processed fearful versus neutral faces in a PTSD group compared with a healthy control group.

A mutual consensus whether vmPFC activity is decreased in PTSD is therefore not yet agreed on. Differences in findings seem mainly to be caused by methodological differences (Hughes & Shin, 2011). Nevertheless, these neurobiological alterations might be involved in the maintenance of PTSD-related symptoms, such as higher emotional arousal, dysfunction of autonomic activity and, above all, anticipation of aversive events strictly connected with the avoidance of trauma-associated stimuli.

## **Discussion**

The primary aim of this literature review was to better understand the association between PTSD and different social cognitive domains. Social cognitive impairments are common and debilitating manifestations in PTSD patients, leading to reduced social support. The acceptance of social support is known to be of great importance for the processing of the traumatic experience (Gros et al., 2016), emphasizing the relevance of social cognition and PTSD. The result of this paper suggests that social cognition is predominantly disturbed in PTSD. Studies on ToM in PTSD patients have shown varied results. It seems that the affective and emotional aspects of ToM are frequently altered, whereas the cognitive dimension is found to be less disturbed, or even preserved. Nevertheless, firm conclusions on the effects of PTSD on ToM are hard to make, since only a few studies have explored this specific relationship and there are large differences in study design and outcome measures. Thus, more research is needed to systematically explore how ToM is affected in PTSD patients.

The association between another social cognitive domain, emotion recognition, and PTSD has been more established. Overall, research has shown that PTSD patients show abnormalities in higher-order emotional processes, including emotion recognition (Passardi, Peyk, Rufer, Wingenbach, & Pfaltz, 2019). Deficits in facial emotion recognition have been reported, especially for the emotions fear and sadness. This may be due to lost factual knowledge that is associated with certain types of facial expressions. (Adolphs, Damasio, Tranel & Damasio, 1996). Disrupted emotion recognition possibly hinders successful social interactions, which consequently may lead to less social support seeking. Again, social support is of importance in symptom reduction (Gros et al., 2016). Therefore, it could be interesting for future research to investigate this correlation further.

Research on PTSD and social perception is lacking, and more research should be conducted to make firm conclusions. Nevertheless, traumatized individuals often experience problems in perceiving and understanding their social context. These problems might arise because a negative attention bias towards social threat might hinder the interpretation and processing of several contextual cues (Bomyea, Johnson, & Lang, 2017; Naim et al., 2015). This bias might not only lead to anxious individuals, but also hinder effective communication with others.

Furthermore, identifying neural mechanisms and structures that play a part in PTSD is interesting and potentially valuable, however, neuroimaging studies do not yet add much to the understanding of PTSD. Since PTSD is often comorbid with other disorders, the distinction between cognitive processes in PTSD and other psychopathology is challenging, and it is hard to define whether deviant brain activity is a cause or an effect of PTSD. Nonetheless, looking at underlying neural substrates can offer a powerful and non-invasive means to understand any dysregulated emotional processing in PTSD patients.

As can be concluded, social cognitive skills are vital for successful communication and, consequently, mental health and wellbeing. Its assessment in clinical practice is, therefore, of paramount importance. Systematic assessment of social cognition in clinical practice is of the essence, in order to optimize the process and to ultimately provide the best care for the patient. This importance is now formally recognized in the last edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-5), which includes social cognition as one of six core neurocognitive domains (American Psychiatric Association, 2013). Standardized tests are essential for objective quantification of the severity and extent of impairment, yet the DSM-5 does not name any proprietary tests. Suggestions of suitable tests in different social cognitive domains are presented by Henry, von Hippel, Molenberghs, Lee, & Sachdev (2016). To assess ToM, they suggest either false-believe tasks, the Awareness of Social Inference Test (TASIT), the Strange Stories test, the Faux-Pas test or the RMET. To assess emotion recognition abilities they suggest the Ekman Faces test or the Facial Expressions of Emotion test. Lastly, to assess social perception they suggest two test batteries; the Comprehensive Affect Testing System and the Florida Effect Battery. Yet, they argue that these tests should be seen in a broader context: clinical assessment should ideally be a combination of standardized self-report, informant rating and ability-based assessments, in addition to clinical observation. Thus, when used in combination with more standard assessments to inform treatment efforts, these measures have the potential to enhance treatment decision-making and outcomes. Identified resilience and risk factors could additionally facilitate recovery by administering early interventions.

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