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Research report

Reduced recognition of fear and sadness in post-traumatic stress disorder

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ABSTRACT

Post-traumatic stress disorder (PTSD) is associated with impairments in emotional experience and expression. The current study examined the recognition of emotional facial expressions in PTSD patients and matched healthy controls, both in terms of accuracy and sensitivity. The task involved short video clips of a neutral face changing (morphing) into one of the six basic emotions (happiness, anger, fear, surprise, disgust and sadness). Clips differed in length, with short clips terminating at 20% of maximum emotional intensity, and the longest ones ending with a full-blown expression. We observed a specific impairment in the PTSD group for recognizing the emotions *fear* and *sadness*. This result was observed via a reduced *accuracy* and a decreased *sensitivity* for these emotions. We discuss the observed altered affective processing and its possible clinical implications.

1. Introduction

Correct interpretation of emotional states and intentions of others as well as the expression of our own emotions is essential in social interaction. Facial expressions represent strong cues for emotional states and their perception and interpretation is a human ability crucial for establishing normal interpersonal relationships. A considerable body of research shows that this function is compromised in many psychiatric disorders, such as social anxiety disorder, as well as in a number of neurological disorders, like Korsakoff's syndrome (Lembke and Ketter, 2002; McClure et al., 2003; Green et al., 2004; Venn et al., 2004; Montagne et al., 2006a). Certain aspects of these psychopathological conditions can be understood as a consequence of the altered processing of emotional cues.

Impairments in emotional experience and expression are also seen in individuals with post-traumatic stress disorder (PTSD). Apart from feelings of shame, guilt, sadness, humiliation, anger, and hyper-reactivity to trauma reminders, PTSD is also characterized by inappropriate emotional responses, reduced affect, and emotional numbing (Reynolds and Brewin, 1999; DSM-IV-TR, 2000; Brewin and Holmes, 2003; Dalgleish, 2004). One typical form of emotional dysfunction

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in PTSD is alexithymia, involving difficulties in identifying and labeling emotions (Sifneos, 1973; Taylor and Bagby, 2004).

Several hypotheses have been proposed to account for PTSD-related disturbances in the area of emotional functioning (see reviews by Brewin and Holmes, 2003; Dalgleish, 2004). The dominant neurobiological models attempt to explain the impaired emotional functioning in terms of disrupted neurophysiological patterns of functioning (Frewen and Lanius, 2006; Rauch et al., 2006). These models suggest that heightened amygdala reactivity to fearful, trauma-related stimuli is the result of a decreased medial prefrontal activity. This is due to impaired inhibition of the amygdale by the medial prefrontal cortex (MPFC; Etkin and Wager, 2007). Neuroimaging studies of PTSD support these models as they report different patterns of neural activity in patients, mainly characterized by elevated amygdala reactivity and decreased activity in frontal areas (Bremner et al., 1999; Shin et al., 2006; Williams et al., 2006).

Other studies suggest that decreased hippocampal volume is the most noticeable change in neuroanatomical terms (Bremner et al., 1997; Gilbertson et al., 2002; Villarreal et al., 2002; Shin et al., 2006). Although the hippocampus is a structure that is more associated with memory function, it is also known to be involved in the processing of emotions (Liberzon and Martis, 2006). Other structural abnormalities have also been reported in PTSD, such as reduced volumes of MPFC (Rauch et al., 2003; Woodward et al., 2006), a formation presumably involved in experience and regulation of emotion (Etkin and Wager, 2007).

Support for the notion that there is a neuroanatomical or neurophysiological basis for the deficient emotion processing in PTSD also comes from studies using electroencephalography (Stanford et al., 2001; Felmingham et al., 2003). Felmingham et al. (2003) observed that evoked response potentials to angry and neutral faces in individuals with PTSD display different patterns relative to controls. The authors concluded that their findings reflect adaptive responding to potential threat in the controls, but a reduced capacity to discriminate between non-threat and generalized threat stimuli in their PTSD group. Similar conditions, such as depression or social anxiety, are also known to disrupt the perception and memory of emotional cues, causing a lower sensitivity for the negative facial expressions of anger and disgust, for instance (Montagne et al., 2006b).

The current study investigated whether people with PTSD have difficulties in perceiving and recognizing emotional faces and whether such a problem will be observed across a range of different emotions. The perception of emotions is not a unitary function and recent studies have suggested that, in clinical populations, the perception of specific emotions may be impaired while the recognition of other emotions remains intact (Calder et al., 2000; McClure et al., 2003; Venn et al., 2006). In addition, in healthy subjects a different recognition rate for specific emotion has been observed, e.g., happy faces are recognized faster and better than sad faces (e.g., Montagne et al., 2005a). We examined the perception of what are generally agreed to be the six basic or universal emotions: anger, disgust, fear, happiness, sadness and surprise (Ekman, 1999) in a population of people with PTSD. Given the frequently observed alexithymic symptoms, emotional numbing, as well as the results from imaging studies, we hypothesize a decline in the accuracy and sensitivity of the recognition of these emotions in PTSD population.

2. Methods

2.1. Participants

Twenty patients, all males (mean age 42.05 ± 4.16), diagnosed with PTSD, were recruited from the 'Stećak', a self-help group of war veterans with PTSD, located in Tuzla, Bosnia and Herzegovina. A qualified psychiatrist carried out the diagnostic assessment in accordance with DSM-IV classification, based on the Structured Clinical Interview for DSM-IV, and the Minnesota multiphasic personality inventory (MMPI) (Avdibegović et al., 2008). None of the patients had a diagnosis of alcohol or substance disorder, lifetime history of psychosis, bipolar disorder, or major mood or anxiety disorder. For control purposes, twenty healthy men (mean age 41.65 ± 4.72), matched on age and education level, were also included. All participants, patients and controls, were war veterans exposed to prolonged traumatic events in combat situations during the war in Bosnia (1992–1995). Control participants had no psychiatric history. This study has been carried out in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. Participation was voluntary and all the subjects gave their informed consent prior to the inclusion into the study.

2.2. Materials

2.2.1. Emotion Recognition Task

To evaluate the recognition of emotional facial expressions, we used a computer task developed by Montagne et al. (2007). Color pictures of the faces of four actors (two males, two females), who posed six emotional expressions (anger, disgust, fear, happiness, sadness and surprise) and a neutral face, were utilized to create computer-generated morphs of 19 intermediate images between a neutral face (0% emotion) and a full-blown expression (100% emotion). The task was developed from algorithms designed by Benson and Perrett (1991). In the morphed images, both the dimension of shape and texture underwent gradual transitions. These images were used to construct video clips that incrementally increase in the degree of intensity of the expression by 10% steps. For each actor, and each of the six emotions, there were nine video clips (0–20, 0–30, 0–40, 0–50, 0–60, 0–70, 0–80, 0–90, 0–100%) (Fig. 1).

2.2.2. Benton Facial Recognition Test

The short form of the Benton Facial Recognition Test (Benton et al., 1994) was administered to assess perception of neutral faces and detect possible generalized disorders of face perception. It consists of a booklet with, on each page, a single target face, and below it, a set of six faces. In the first six trials, an identical face has to be selected among five decoys. In the remaining 7 trials, three different views (changed in orientation or lighting conditions compared to the target photograph) have to be distinguished from three incorrect alternatives. The faces are unfamiliar and physically similar, without spectacles or facial hair. The maximum number of correctly identified faces is 27.

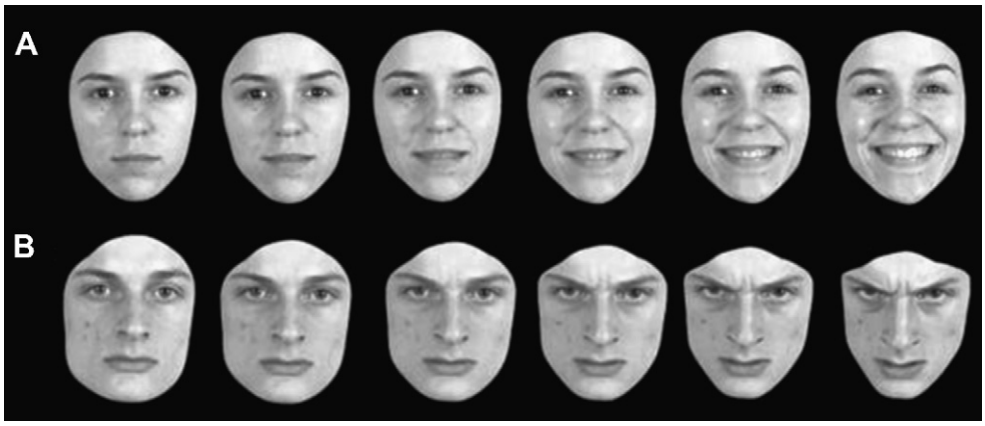

Fig. 1 – The Emotion Recognition Task shows (A) the gradual transition from neutral (0%) to the full-blown emotion of happiness (100%) in steps of 20%, and (B) the gradual transition from neutral (0%) to the full-blown emotion of anger (100%) in steps of 20%.

2.2.3. *Beck Depression Inventory (BDI) scores*

The BDI (Beck et al., 1961), a 21-item self-report questionnaire, was used to measure the presence and the intensity of depressive symptoms.

2.3. *Procedure*

Each participant completed the BDI and the Benton Facial Recognition Test prior to the emotion recognition task. The task started with four practice trials, followed by the main experiment. The clips entailing morphed images of emotional expressions were presented in an incremental order, starting with 20% intensity levels and ending with a 100% expression. Thus, subjects first saw the 24 presentations of the lowest level of emotional expression (0–20%), followed by the second set of 24 trials with video clips from 0% to 30%, and so on until they reached the final sequence of clips in which the neutral face changes into a full-blown expression. Within each level, the order of presentation of emotions and actors was randomized. On each trial, subjects were required to make a forced choice between one of six emotional expression labels that were displayed on the screen during the time of presentation. The duration of the animation in the video clips varied between approximately .5 sec (low intensity) and 2 sec (high intensity). The moving expressions were created in an artificial manner but appear very similar to natural expressions. After the animation, the last frame remained on the screen until the subject made a response, at which time the new trial was automatically initiated. In total each participant performed 216 trials. There was no time restriction. The procedure was identical for the control subjects.

3. Results

3.1. *Demographic data*

Statistical analysis (independent sample *t*-test) revealed no significant differences in age [$t(38) = .27$, $p = .79$] or education [controls mean = 11.05 years, PTSD mean = 11.80 years, $t(38) = 1.10$, $p = .28$] between the groups.

3.2. *Control tasks*

There were significantly higher BDI scores in the PTSD group (mean = 11.50, SD = 3.90) compared to the control group [mean = 3.50, SD = 1.60, $t(38) = 9.3$, $p < .01$]. The short form of the Benton Facial Recognition Test did not reveal significant differences between the PTSD group (mean score = 22.50, SD = 2.20) and the control group [mean score = 23.40, SD = 1.70, $t(38) = .53$, $p = .60$].

3.3. *Emotion recognition*

Two different measures were used; accuracy and sensitivity. *Accuracy* was defined as the percentage of correct answers for each emotional expression at each intensity level. Perceptual *sensitivity* for a specific emotion was defined as the level of emotional intensity that was required for the correct identification of this emotion.

3.3.1. *Recognition accuracy*

Accuracy of recognition of the six basic emotions, anger, disgust, fear, happiness, sadness and surprise, was defined as the total number of correct responses for a specific emotion. Increased intensity of emotional expression resulted in a higher recognition rate, although the slopes differ between emotions (Fig. 2). The percentage of correct answers for each emotional expression at each intensity level was analyzed with a 9×2 repeated measures analysis of variance (ANOVA) general linear model (GLM) with group (PTSD *vs* control) as between-subject variable and intensity level as within-subject variable. Significant differences were observed between the two groups on the recognition of fear [$F(1, 38) = 14.20$, $p < .01$, Fig. 3] and sadness [$F(1, 38) = 3.90$, $p = .05$].

The PTSD group had a higher BDI score than controls. In order to control for a possible confounding effect, we performed an additional analysis with depressive symptoms as

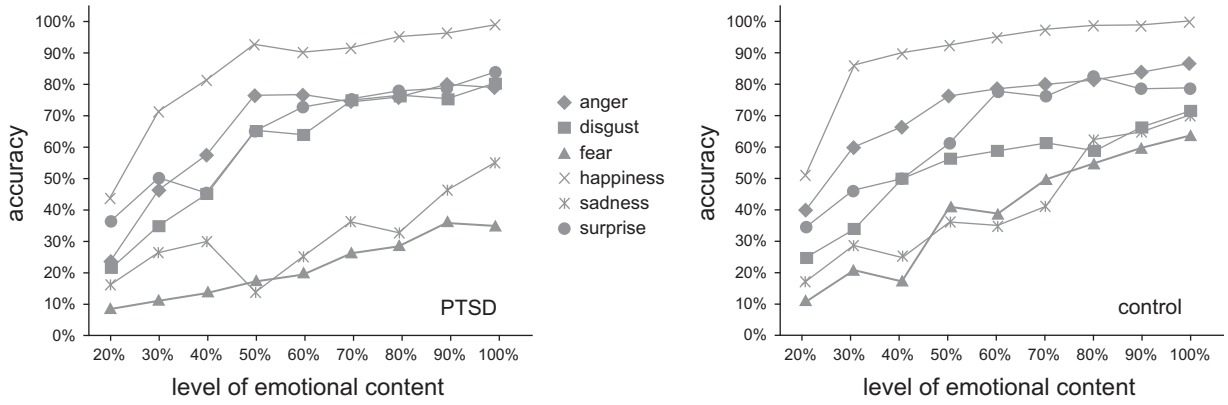

Fig. 2 – The accuracy of recognition plotted for all levels of emotional intensity (*x*-axis, levels 1–9 represent 20–100% emotional content in the face). A general trend for all emotions is an increase in the recognition rate associated with an increase of the emotional content. In the PTSD group, two emotions, sadness and fear, have a clear lower recognition rate compared to other emotions.

covariate. The analysis showed a significant difference between the two groups [$F(5, 33) = 4.24$, $p < .05$]. We did not observe any significant interaction effects.

Overall, the accuracy fluctuated for different emotions [$F(5, 35) = 24.37$, $p < .01$]. Most accurate recognition performance was observed for the emotion happiness (significantly more accurate than all other emotions; all F-values > 19.09), followed by anger, surprise and finally disgust.

3.3.2. *Perceptual sensitivity*

The perceptual *sensitivity* measure was calculated as the percentage of required intensity from which level onwards the emotion was consistently correctly identified.

Statistical analysis (repeated measures ANOVA), with group as the between-subject factor and emotional expression (6 basic emotions) as the within-subject variable, revealed group differences in the level required to correctly identify emotional content [$F(5, 34) = 30.35$, $p < .01$]. In particular, the differences exist for two emotions, fear [$F(1, 38) = 10.31$, $p < .01$] and sadness [$F(1, 38) = 4.60$, $p < .01$]. The PTSD group required on the average more expression in the face in order to correctly identify the emotions fear and sadness (91% and 87% respectively). There were no differences for the emotions anger, disgust, happiness and surprise (all F-values < 2.50).

A separate analysis, which included the BDI scores as a covariate, still showed a significant overall difference between the two groups [$F(5, 33) = 6.71$, $p < .05$], again due to the differences in performance of the perception of fear and sadness.

In addition within each group we observed differences in performance between individual emotions with respect to the level of intensity required for their correct identification [PTSD: $F(5, 114) = 25.56$, $p < .01$, controls: $F(5, 114) = 16.93$, $p < .01$, Fig. 4]. The observed pattern is consistent with the results of the accuracy measure and in line with previous findings. For instance, Montagne et al. (2005a) also found the lowest thresholds for happiness, and the highest for fear and sadness.

4. Discussion

The current study investigated the recognition of facial emotional expressions in people with PTSD. The findings showed decreased *sensitivity* for the emotions of fear and sadness. The patients needed more intense emotional

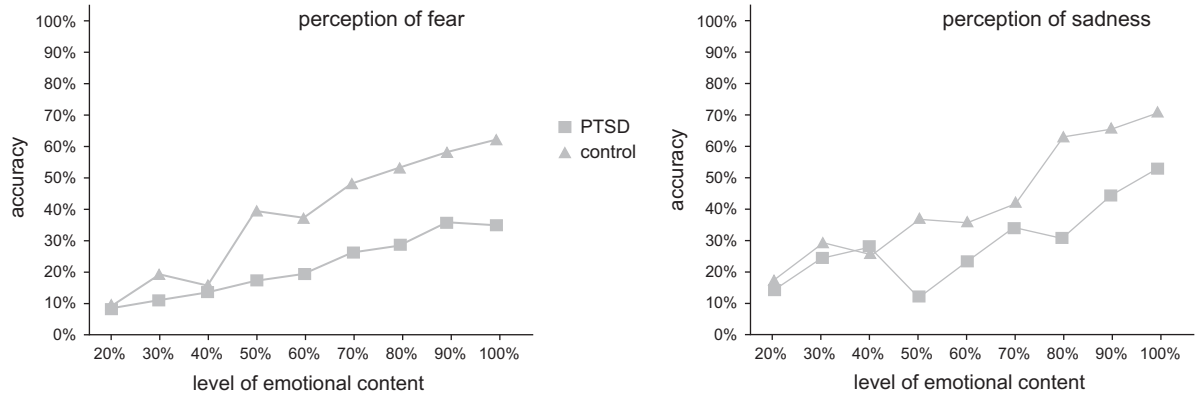

Fig. 3 – Differences in the recognition accuracy for the emotions of fear and sadness in our two groups. The controls gain significantly more from the higher levels of emotional content than our PTSD group.

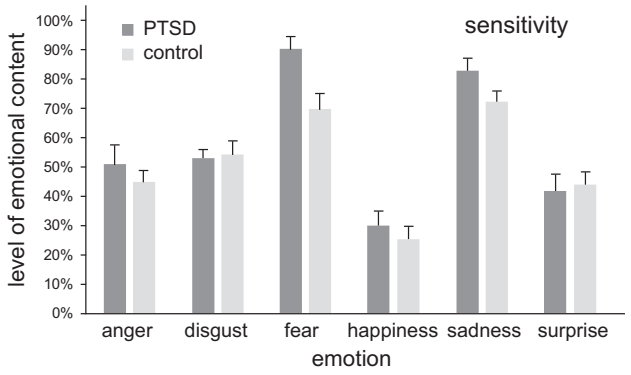

Fig. 4 – The level of emotional content necessary to correctly identify each emotion. The pattern resembles the findings in previous studies, the emotion of happiness requiring lowest amount of emotional content to recognize it correctly while the emotion of fear is most difficult to identify. There are significant differences between the two groups for the emotions of fear and sadness. Error bars represent 1 SE.

expressions that control to correctly identify these facial emotions. In addition, we observed a reduced *accuracy* for the same emotions, fear and sadness, compared to the control group.

These findings are in accordance with earlier studies that showed impaired recognition of fear in other patient populations with disorders in the area of affective functioning (Montagne et al., 2005c, 2006a, 2006b). Our study is also in line with the conclusions of Frewen et al. (2008) in their meta-analysis of the alexithymic symptomatology in PTSD, with particularly large effect size associated with male combat PTSD samples, such as the one in our study.

An explanation for the poor performance in our PTSD patients is not related to basic deficits in the perception of faces, as the performance of the groups did not differ on the Benton Facial Recognition Test. Two alternative explanations for our findings need to be discussed. First, the ability to recognize emotions usually correlates with general cognitive functioning (Mathersul et al., 2009). Montagne et al. (2005b) observed a relationship between cognitive performance and the performance on the emotion recognition task in post-stroke depression (PSD) patients. In addition, recent studies have linked PTSD with impairments in cognitive functioning: attention, working memory, or initial learning (Yehuda et al., 2004; Bremner et al., 2004; Vasterling et al., 2002; Bowler et al., 1998). We have not assessed cognitive functioning in our study, but we feel that this is an unlikely explanation, because our two groups did not differ in their abilities to detect facial identity (a cognitive task) and it is difficult to see why cognitive decline would result in a selective problem for fear and sadness.

Alternatively, our subjects' emotional state might have influenced the result. PTSD is often accompanied by depression (Kemp et al., 2008) and we did observe higher scores on the BDI in our PTSD group. Studies in both patients with mood disorders and healthy volunteers have shown that facial expression perception can vary according to current mood state (Venn et al., 2006). Patients with major depression have been reported to show a negative bias for the interpretation of facial expressions (Feinberg et al., 1986). Kemp et al. (2007) found that PTSD patients with comorbid depression showed reduced activity in the amygdala and medial prefrontal cortex in response to fear, relative to PTSD patients without comorbid depression. As mentioned above, Montagne et al. (2005b) showed decreased overall accuracy in the recognition of facial expressions in people with depressive symptoms after a stroke. However, in a separate statistical analysis that included the BDI score as a covariate, the results remained the same. In addition, the pattern of performance with a selective deficit for fear and sadness has not been reported in depressed patients.

In general, perception of facial expressions is believed to occur independently from other aspects of face processing (De Haan, 2002; Hargrave et al., 2002). Patients with prosopagnosia have difficulties in identifying faces, but their recognition of emotions may remain intact (Stephan et al., 2006). In contrast, other patients experience deficits in the perception of facial expressions, but remain able to recognize familiar faces. We propose that our PTSD patients suffer from a selective impairment in the recognition of emotions. More specifically, they suffer from a recognition disorder that affects certain emotions, i.e., fear and sadness, and not others (surprise, happiness, disgust, and anger).

The selective disruption of the ability to identify certain emotions has been described before (Calder et al., 2000; McClure et al., 2003; Venn et al., 2006). For instance, patients with damage to the amygdale may be impaired in the perception of anger and fear, while insular lesions appear to selectively affect the recognition of disgust (Adolphs et al., 1994; Calder et al., 1996). There is now converging evidence from neuroimaging studies suggesting separate neural substrates underlying the recognition of different facial (emotional) expressions (Phan et al., 2002). Selective deficits have also been observed in psychiatric populations, such as the social anxiety disorder, which is characterized by an attentional avoidance of angry facial expressions (McClure et al., 2003).

We can only speculate with respect to the anatomical substrate of the recognition disorder of fear and sadness in our population. Impaired processing of emotions is commonly attributed to limbic system, in particular the basal ganglia, amygdale, the insula, the cingulate, and the medial frontal lobe (Frewen et al., 2008; Dolan, 2009). Altered amygdala activity has also been observed in other types of disorders, such as psychopathic individuals. Amygdala damage has been associated with a selective impairment of fear recognition (Rauch et al., 2006; Williams et al., 2006), and Venn et al. (2006) refer to a number of studies in support of a possible link between the amygdala and sadness perception. Although we did not have anatomical data of the participants in our study, it is reasonable to suggest a role for the amygdale. This is in line with studies observing altered patterns of amygdala function in PTSD patients (Phillips et al., 1999). In PTSD this is often accompanied by inadequate inhibitory response by frontal areas, MPFC in specific (Shin et al., 2006; Williams et al., 2006). Finally, PTSD is known to impair the functioning of the hippocampus (Brewin and Holmes, 2003), another structure involved in emotional processing (Liberzon and Martis, 2006).

The possibility that these regions are also involved has to be taken into account.

Our findings may have significant clinical implications. The assessment of emotion perception might become a clinically useful addition to neurocognitive assessment of PTSD patients. Impairments in affective processing in Huntington disease (HD), such as deficit in the recognition of disgust, have been observed even in the pre-symptomatic stage when motor signs are not yet present (Speedie et al., 1990). A similar suggestion for the measurement of facial expression perception as a useful tool for assessing the efficacy of antidepressant treatments was made by Venn et al. (2006). Our patients show a very specific emotion recognition deficiency that might be characteristic pattern for PTSD. If this is true, then the emotion recognition task may be a useful tool for assessing the possible existence of PTSD, when other symptoms cannot provide clear answers. In addition, this tool could be used to improve prognostic models for individual differences in treatment outcome of patients with PTSD.

In sum, we have demonstrated that patients suffering from PTSD have a selective deficit in the recognition of the emotions *fear* and *sadness*. This selective impairment might be helpful in trying to understand this form of psychopathology. In addition, it might be used in the clinical practice of assessing PTSD and the development of more detailed prognostic models.

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