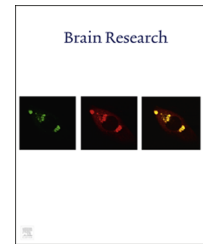


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## Research Report

# Negative emotional distraction on neural circuits for working memory in patients with posttraumatic stress disorder



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## ABSTRACT

**Objective:** To study the neural mechanism for the impact of negative emotional distraction on working memory in patients with posttraumatic stress disorder (PTSD) resulting from exposure to motor vehicle accidents.

**Methods:** Twenty PTSD patients and 20 healthy subjects were recruited. Event-related functional magnetic resonance imaging (fMRI) was used to investigate the effects of negative and neutral distractors on a delayed-response working memory task. All experiments were performed on a 3.0T MRI scanner, and the functional imaging data were analyzed using SPM8 software.

**Results:** The PTSD group showed poorer performance than the control group when the negative distractors were presented during the delay phase of working memory. The functional imaging indicated that, in the presence of negative relative to neutral distractors, the PTSD group showed higher activation in the emotion processing regions, including amygdala, precuneus and fusiform gyrus, but lower activation in the inferior frontal cortex, insula and left supramarginal gyrus than the control group.

**Conclusion:** Based on the results that activation in the PTSD patients in the presence of negative distractors increased in the emotion-related brain regions but decreased in the working memory-related brain regions, we may conclude that the neural basis of working memory is impaired by negative emotion in PTSD patients.

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## 1. Introduction

Posttraumatic stress disorder (PTSD) results from exposure to a traumatic event, such as a fight, violent crime, childhood abuse or motor vehicle accident, and is characterized by unique symptoms such as recurrent, involuntary recollection of the trauma in the form of intrusive thoughts, nightmares, or vivid sensory memories (Blake et al., 1995). It is notable that motor vehicle accidents are the leading cause of PTSD in the general population (Blanchard et al., 1994; Blanchard & Hickling, 2004). More importantly, rather than merely remembering it as a past event, PTSD sufferers seemingly relive the trauma with all its original intensity (McNally, 2006). Meanwhile, the trauma or trauma-related negative emotional stimuli may produce striking disturbances in cognition, especially in working memory. Previous studies have found that PTSD has been associated with marked cognitive deficits including working memory (Vasterling et al., 1998). However, the mechanism for the impact of negative emotional distraction on neural circuits for working memory in PTSD remains largely unknown.

In the recent years, a few functional neuroimaging studies have examined the neural basis of the impact of emotional distractors on working memory performance (Dolcos et al., 2006; McNally, 2006; Wessa et al., 2012; Fonzo et al., 2010). Some studies have shown that presenting emotional distractors during the delay interval evoked strong activity in typical emotion-processing regions of the brain (the amygdala and ventrolateral prefrontal cortex), while simultaneously evoking relative deactivation in the dorsal executive regions (the dorsolateral prefrontal cortex and lateral parietal cortex) and impairing working memory performance (Dolcos and McCarthy, 2006; Dolcos et al., 2006; Pannu Hayes et al., 2009). Aupperle et al. (2012) also found that activation is attenuated in the lateral prefrontal cortex but enhanced in the medial PFC, amygdala and insula during emotional anticipation. Activation was more positively correlated with the level of PTSD symptoms in the ventral frontolimbic regions (notably the ventromedial prefrontal cortex, inferior frontal gyrus, and ventral anterior cingulate gyrus) in the presence of negative stimuli than in the presence of neutral stimuli. Conversely, activation in performing executive tasks was negatively correlated with PTSD symptoms in the dorsal executive network, notably the middle frontal gyrus, dorsal anterior cingulate gyrus, and inferior parietal lobule (Morey et al., 2008). Additionally, the PTSD group showed neural activity markedly different from that of the control group, in response to task-irrelevant visual distractors. To be specific, in the PTSD group, enhanced activity in the ventral emotion-processing regions (the amygdala, ventrolateral prefrontal cortex, and fusiform gyrus) was associated with trauma distractors while activity in the dorsal executive regions (the dorsolateral prefrontal cortex and lateral parietal cortex) was associated with working memory, and attention was disrupted by the distractors independent of their trauma content (Morey et al., 2009). However, some other studies detected enhanced activation in the dorsal executive regions when emotional stimuli were presented as distractors before or during the task, potentially indicating compensatory activation to preserve goal-directed

behavior (Wessa et al., 2012; Blair et al., 2008; Hart et al., 2010; Pereira et al., 2010).

Moreover, the functional neuroimaging studies of working memory have supported that the inferior frontal cortex plays a role in inhibitory processes (D'Esposito et al., 1999; Jha et al., 2004; Dolcos et al., 2006). The activity of the inferior frontal cortex is correlated with subjective ratings of distractibility for task-irrelevant emotional stimuli presented during the delay interval of a working memory task. In a former study, the participants who showed greater inferior-frontal-cortex activity in the presence of emotional distractors also rated themselves as less distracted, possibly as a result of engaging inhibitory processes that reduced the subjective impact of emotional distraction (Dolcos and McCarthy, 2006). Further studies extended the evidence from the studies of cognitive control of emotion that the inferior frontal cortex is involved not only in controlling the emotional response induced by potentially distracting emotional stimuli, but also in diminishing the negative impact of distracting emotions on ongoing cognitive processes (Dolcos et al., 2006). However, the precise role of the inferior frontal cortex in controlling the impact of negative emotional distraction on working memory in PTSD has not been firmly established.

In our previous study, we found significant decrease in cortical thickness in the left medial prefrontal cortex and anterior cingulate cortex in PTSD patients, indicating deficits in the working memory of PTSD patients (Xie et al., 2013). However, little is known about the neural mechanism for the impairment of working memory by negative emotional distraction. The mechanism has important implications for PTSD, as PTSD is characterized by increased susceptibility to emotional distraction. Therefore, the aim of the present study was to investigate the impact of negative emotional distractors on working memory in healthy controls and PTSD patients resulting from motor vehicle accidents using functional magnetic resonance imaging (fMRI) and to explore the neural mechanism for the impairment of working memory by negative emotional distractors in PTSD. We hypothesized that (1) the negative (relative to neutral) distractors would lead to increased activation in both groups, when more neural resources are devoted to working memory performance; (2) the negative emotional distractors would impair working memory performance in the PTSD group; (3) the negative emotional distractors would cause greater activation in the emotion-processing brain regions in the PTSD group than in the control group and impair the function of the inferior frontal cortex in the PTSD group.

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## 2. Results

The PTSD patients and the control group were matched with respect to age, gender, and education duration; there was no significant difference in IQ between the two groups. The patients with PTSD had significantly higher CAPS scores than the control group (Table 1). According to the SCID, 3 subjects in the PTSD group met the DSM-IV diagnostic criteria for the depressive disorder. Among the control subjects, the SCID did not reveal any psychiatric disorders.

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