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**The role of coping with stress and conditioning to trauma reminders in posttraumatic stress disorder: behavioral and neural correlates**

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Posttraumatic stress disorder is an anxiety disorder triggered by one or several traumatic events, which mostly include the experience of threat, fear and uncontrollability. The most potent models our days for the development and maintenance of posttraumatic stress disorder (PTSD) are the fear conditioning model and the stress sensitization model (Foa, Zinbarg & Rothbaum, 1992; Grillon et al., 1996; Siegmund & Wotjak, 2006). In this work associative learning and stress sensitization were investigated in patients with PTSD and traumatized control subjects without a history of PTSD (NPTSD) in two studies that employed functional magnetic resonance imaging (fMRI). The first study investigated stress-induced analgesia. In contrast to previous studies a trauma-unrelated stressor was used to induce analgesia. The results showed enhanced analgesia in PTSD patients as indicated by an increase of pain thresholds and a decrease in pain ratings. PTSD patients showed increased activation of the medial prefrontal and the rostral anterior cingulate cortex, areas known to modulate pain experience and found to be associated with opioid mediated pain reduction and placebo analgesia. In the second study higher order differential fear conditioning was investigated in traumatized individuals with and without PTSD and in never traumatized healthy controls. A trauma-relevant picture was used as unconditioned stimulus and two neutral symbols served as conditioned stimuli. The results indicate enhanced higher order conditioning in PTSD patients compared to NPTSD participants and healthy controls by enhanced differential arousal ratings during the learning phases of the reinforced versus the non-reinforced conditioned stimulus. Contingency ratings revealed deficient extinction of the conditioned response in the PTSD group. The left amygdala was less active during the reinforced versus the non-reinforced conditioned stimulus in the acquisition phase in PTSD. The deactivation of the amygdala interacted with enhanced activity of the dorsolateral prefrontal cortex in the patient group, suggesting anticipatory coping processes, which might regulate arousal and diminish reexperiencing.

These findings show enhanced stress reactivity and reduced pain perception, as well as enhanced higher-order conditioning in PTSD patients in contrast to traumatized participants without PTSD and healthy controls. The results underline the importance of both stress sensitization and associative learning in PTSD and suggest further investigations of reciprocal effects between stress and conditioning as suggested by the dual branch hypothesis (Siegmund & Wotjak, 2006). The reduced analgesic response in trauma-exposed subjects without PTSD and the deficient associative learning might represent resilience markers or adaptive alterations after the trauma experience.