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The neural networks of inhibitory control in post-traumatic stress disorder

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Post-traumatic stress disorder (PTSD) is associated with information-processing deficits and a reduction of selective attention, along with hyperarousal and hyper-reactivity to threat- and emotion-related stimuli. PTSD may involve an enhancement in automatic processing and an inability to inhibit automatic processing when required. The predominant neurophysiological model of PTSD has focused on medial prefrontal disruptions during the processing of fear-related stimuli and does not include potential alterations in the explicit inhibitory control of automatic responding. We investigated executive inhibitory control in PTSD during an emotionally neutral (go/no go) response inhibition task in individuals with PTSD ($n = 23$) and matched healthy controls ($n = 23$) using functional magnetic resonance imaging. PTSD was associated with diminished inhibitory control and reduced activation of a cortical inhibitory network (particularly right ventrolateral prefrontal cortex), as well as an increase in areas associated with sensory processing (somatosensory and visual cortices) and increased inhibitory task demand (striatum). These findings are consistent with diminished executive inhibitory control in PTSD and may reflect increased stimulus processing, which undermines cortical control mechanisms.

04-04

Treatment response and post-traumatic stress disorder: neuroimaging findings

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Biological models propose that post-traumatic stress disorder (PTSD) reflects a failure of extinction of the conditioned fear response. Animal models and recent human imaging studies suggest that ventromedial prefrontal (vmPFC) regions inhibit amygdala fear networks during fear extinction (Rauch et al. 2006; Phelps et al. 2004). Accordingly, PTSD is associated with a failure of vmPFC activity in response to threat. Exposure-based treatments are thought to facilitate the extinction of conditioned fear. No imaging studies have examined the neural correlates of symptom improvement following exposure-based treatment in PTSD. Eight individuals with PTSD underwent functional magnetic resonance imaging (fMRI) scanning while viewing fearful and neutral facial expressions in a passive viewing task adapted to a 1.5T scanner. fMRI assessments were conducted before and after treatment. Amygdala and vmPFC (anterior cingulate) activity was examined before and after treatment in a repeated-measures, fixed-effects ANOVA and changes in these regions were correlated with changes in PTSD severity. Consistent with predictions, findings show that anterior cingulate activity increased and amygdala activity reduced to fear following exposure treatment in PTSD, and symptom improvement was correlated with increased anterior cingulate activity and reduced amygdala activity to fear. These findings provide initial support for a role of reduced vmPFC activity in PTSD that recovers following exposure treatment.

04-05

Neural activity in dissociative and nondissociative PTSD: an fMRI analysis of conscious and nonconscious fear processing

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Post-traumatic stress disorder (PTSD) involves variable responses to threat, ranging from hyperreactive to dissociative. While most imaging research has explored hyperreactivity in PTSD, an important but poorly understood subtype of PTSD is dissociation. Recent imaging research suggests that dissociative