Child abuse and recovery

Thomaes, K.

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Outline of the thesis

The baseline part of this thesis (Chapter 2, 3, 4) attends to neurophysiologic changes after repeated child sexual and/or physical abuse in patients who are diagnosed with Complex PTSD with high rates of dissociative symptoms and comorbid depressive disorder and personality disorders, compared to non-trauma-exposed healthy controls. Chapter 2 describes a small sample of Complex PTSD patients and controls (9 vs. 9) of our pilot study; other chapters (3, 4 and also 5) refer to a larger patient sample (33 vs. 30) as part of our main (treatment) study.

In Chapter 2 we aimed to investigate declarative memory function and medial temporal lobe activation in 9 patients and 9 non-trauma-exposed healthy controls and to test organizational and methodological issues (pilot study). We focused specifically on the medial temporal lobe (MTL), including the hippocampus and amygdala, to increase power. We performed an event related functional magnetic resonance imaging (fMRI) during a verbal declarative memory task with neutral and negative words and compared performance (reaction times and error rates) and Blood Oxygenation Level Dependent (BOLD) signal changes between subjects with Complex PTSD and controls. Based on the fear conditioning model in 'simple' PTSD, we hypothesized that Complex PTSD patients would show worse declarative memory function with a preference for negative words relative to neutral words, and that this would be reflected by a decreased in BOLD response in the MTL in patients compared to controls.

In Chapter 3 we aimed to investigate further declarative memory function in a new larger sample of 33 patients with child abuse related Complex PTSD and 30 non-trauma-exposed healthy controls, extending the previous pilot study. Specifically, we wanted to investigate how symptom severity and comorbidity affect neurocognitive functioning in PTSD. We simplified and adapted the emotional memory task to increase power and extended our regions of interest to the prefrontal cortex (PFC), considering its role in extinction and emotion regulation. Again we assessed performance and BOLD response (now whole brain) differences. In addition, we explored whether any abnormalities were correlated with PTSD, trauma severity, and comorbid psychopathology, such as depressive, dissociative and borderline personality symptoms. We hypothesized that -as modeled in the fear conditioning model - Complex PTSD patients would perform worse than controls during a verbal declarative memory task, in particular for neutral words, and that this would be reflected by an increased BOLD response to negative words contrasted with baseline in the amygdala, and a decreased BOLD response in the medial PFC, OFC, and ACC, and in the hippocampus compared to controls.

In Chapter 4 we investigated if child abuse related Complex PTSD is associated with regional reduced brain volumes compared to healthy controls in the same sample (33 vs. 30) of Chapter 3, as was described in the PTSD literature. Again we explored whether any abnormalities were correlated with PTSD and/or trauma severity, or rather with comorbid dissociative, depressive and/ or borderline personality symptoms. To this end, we compared regional gray matter (GM) density on a whole-brain voxel by voxel basis in patients with child abuse related Complex PTSD and matched non-trauma-exposed healthy controls and performed regression analyses using these clinical variables as covariates. We

hypothesized that in child abuse related Complex PTSD volumes of MTL regions (hippocampus and amygdala) and ACC are reduced compared to healthy controls.

The second part of this thesis (Chapter 5, 6), investigates recovery of brain changes in (Complex) PTSD after treatment. Chapter 5 shows results of neurobiological treatment effects in a fMRI study (n = 16) using a classic and emotional Stroop task as part of the above mentioned treatment study, i.e. a randomized controlled trial (RCT). We aimed to investigate whether increased activation in brain areas which are associated with error detection and emotional arousal, would normalize after effective treatment. We compared a stabilizing group treatment based on psycho-education and cognitive behavioral therapy (EXP) added to treatment as usual (TAU) with TAU only. Based on the literature, we hypothesized that at baseline, Complex PTSD patients would show increased interference during both classic and emotional Stroop tasks together with increased inferior frontal cortex, decreased ventral ACC, increased dorsal ACC and - because of high levels of dissociation (Bremner et al., 2004a) - decreased insula activation compared to controls. Furthermore, we expected Complex PTSD patients to show normalization of behavioral and neurophysiologic abnormalities following treatment, and to find correlations of change in PTSD severity with change in brain activation in the a priori regions of interest.

In chapter 6 we investigated in a systematic review whether neural correlates of PTSD normalized after successful treatment and if there are differences between adult trauma related PTSD and child abuse related Complex PTSD in this respect. We systematically reviewed imaging treatment outcome studies in adult patients with (partial) PTSD treated with pharmacotherapy, psychotherapy or other therapies in (Randomized) Controlled Trials or pre-post designs, excluding case studies form databases from PubMed, EMBASE, PsycINFO, PILOTS and Cochrane Library. I conclude with a general discussion in Chapter 7.