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Emotion Regulation and Pain in Borderline Personality Disorder

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BPD is a highly prevalent personality disorder, characterized by instability in affective states, self-image, interpersonal relationships, and behavior. Affective instability seems to be caused by an interaction of limbic hyperarousal along with deficits in emotion regulation. Problematic behavior, such as self-injurious behavior is used by patients to reduce aversive tension. However, neuronal mechanisms underlying this effect of SIB are not clear.

The central aim of this thesis was to enhance the understanding of the interconnection between affective dysregulation and SIB. Therefore, we conducted an experiment inducing negative (vs. neutral) affect through picture stimuli. Adapting an established paradigm from general emotion regulation research, we additionally administered sensory (painful vs. warm) stimuli in order to test the effect of pain on negative affect.

The results of Studies 1 and 3 provide further evidence for disturbed emotional processing in BPD. First, we could replicate limbic hyperreactivity and reduced gray matter volume in the amygdala in BPD patients. Second, DLPFC volume was reduced in BPD patients with co-morbid PTSD, but increased in subjects reporting high levels of trait dissociation. These results corroborate previous findings on structural and functional aberrations in the prefrontal-limbic network in patients with BPD. Most importantly, we found evidence for a specific role of painful stimuli in emotion regulation in BPD. We could replicate the findings of a heightened pain threshold in BPD. However, in Study 1, we obtained mixed results for effects of sensoric stimulation on brain activation levels specific for BPD or for painfulness. Nevertheless, in Study 2, we demonstrated differential effects in BPD with regard to functional connectivity. More specifically, we found that a negative co-variation of brain activity between limbic and prefrontal structures is only evident in BPD when painful stimuli are present during states of enhanced emotional reactivity.

Taken together, we provided further evidence for alterations in the structure and function of limbic regions as well as in the emotion regulation process in BPD. Patients show smaller gray matter volume in limbic regions and enhanced limbic reactivity to emotional stimuli. Furthermore, we provided evidence that pain improves the inhibition of limbic activity by prefrontal areas. It can be concluded that painful stimuli lead to enhanced inhibition of limbic regions by prefrontal control areas in BPD, which may be caused by different appraisal of painful stimuli and attentional distraction by pain. This implies that self-injury reflects a dysfunctional attempt of BPD patients to regulate aversive emotional states.