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**Sensory alterations and affective-sensory interactions: Implications  
for the psychopathology of Borderline Personality**

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Borderline personality disorder (BPD) is a complex mental disorder characterized by an interplay between disturbed emotional processing and an altered perception of the body that include sensory deficits. However, it is still unclear if these alterations, which occur primarily in aversive affective situations, are a core variable or some type of coping behavior related to the disorder. The present thesis focuses on the characterization of the altered affective-sensory processing and potential explanations of these dysfunctions, considering BPD-specific symptomatic aspects such as dissociation and investigated the neural correlates of affective-sensory interaction in individuals with current and remitted BPD compared to healthy subjects. In the first study, we examined the relationship between stress-induced dissociation and altered heat pain sensitivity using personalized stressful and neutral scripts in patients with current (N=25) and remitted BPD (N=20) as well as 24 healthy controls. In the second study, we investigated 21 current and 15 remitted BPD patients and 22 healthy controls to determine whether a pattern of brain activation during listening to affective auditory stimuli is altered in both BPD groups compared to healthy controls and whether altered activation patterns in the amygdala in response to emotionally valenced sounds are normalized in participants with remitted BPD. The results of the first study demonstrate that current BPD patients compared to healthy controls display significantly increased dissociation and heat pain thresholds in the neutral situation, while individuals with remitted BPD were in-between. After listening to the stress script, both clinical samples showed enhanced dissociation scores. Participants with current BPD exhibited pain hyposensitivity with significantly higher heat pain thresholds, while remitted BPD only displayed a trend in the same direction. Both BPD groups showed significant heat pain hyposensitivity in the stress condition compared to the healthy controls, but did not significantly differ from each other. However, in the stress compared to the neutral condition, remitted BPD participants showed pain hyposensitivity compared to healthy controls. These findings suggest different mechanisms involved in pain hyposensitivity in current and remitted BPD. In the second study, both current and remitted BPD patients compared to healthy controls rated positively valenced auditory stimuli as significantly less pleasant. We found increased frontolimbic activation during processing of negatively valenced but not positively valenced auditory stimuli in current BPD. Regarding amygdala activations, only remitted BPD patients exhibited significantly enhanced amygdala activity during processing of negatively and positively valenced sounds compared to neutrally valenced sounds. The two studies comprising this thesis provide evidence for deficient sensory-affective interaction in various sensory modalities, more specifically dissociation proneness and pain hyposensitivity as well as disturbed emotional reactivity during the processing of auditory stimuli. This altered processing of sensory-affective interaction could be characterized in BPD and these will continue to serve as putative mechanisms that further our understanding of the disorder. Furthermore, altered neural activity with increased amygdala activation during the processing of negatively valenced sounds in BPD patients after remission could explain the unstable state of BPD remission. Prospective studies are required to clarify this mechanism. The clinical value of these findings, as well as their relevance for therapeutic considerations in the aftercare of BPD, needs to be further evaluated in the future.