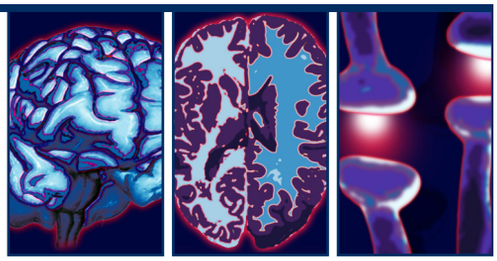

Self-injury and borderline personality disorder

"...a lot of research needs to be carried out to better understand self-injurious behavior in patients with BPD. This will hopefully lead to further destigmatization of this frequent form of psychopathology...."

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Nonsuicidal self-injurious behavior (NSSIB) is a severe form of psychopathology, which a psychiatrist or psychotherapist working with patients suffering from borderline personality disorder (BPD) frequently encounters. We have been working with self-injurious patients for many years and have intensively investigated the origin and the neurobiological background of this behavioral pattern. Still, even if one better understands the motives for self-injury and the underlying mechanisms, this behavior does not lose its embarrassing character. Rather, the suffering of self-injurious patients can be a strong driving force that makes us want to understand its origins and mechanisms better to finally optimize treatment options. This article will first give an overview of what we know regarding NSSIB followed by a section on open questions in this domain.

What do we know?

Nonsuicidal self-injurious behavior in patients with BPD comprises phenomena such as cutting, burning and head-banging, among others, and can be relatively clearly distinguished from suicidal behavior [1]. In patients with BPD, auto-aggression without suicidal intent is usually repetitive, has limited potential for serious or fatal physical harm, and involves a different spectrum of motives than suicidal or ambivalent auto-aggression [2–4]. There is robust evidence that BPD patients use NSSIB to achieve quick release from strong aversive inner tension [2–6]. Release from aversive inner tension by NSSIB can be understood as a dysfunctional coping mechanism of borderline patients when they try to regulate emotions [2,7] and as a negative reinforcer for repetitive dysfunctional behavior.

'Tension release' [3] and relief or escape from emotions [4,6,8] are thought to be the predominant motives for NSSIB, although several studies revealed that motives of NSSIB in BPD are complex and cannot be easily reduced to a single reason. NSSIB is also used to terminate symptoms of dissociation, such as derealization and depersonalization. Further motives comprise self-punishment, feeling physical pain, reducing

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anxiety and despair, emotion generation, controlling others, distraction and preventing oneself from acting on suicidal feelings [2,4,9,10].

We now have some limited understanding of the neurobiological underpinnings of NSSIB. Self-injury in patients with BPD is clearly related to emotion dysregulation as well as disturbed pain processing. We could demonstrate that self-injurious patients with BPD show elevated pain thresholds in relation to emotional stress [11–13], and that reduced pain sensitivity is related to the activation of an antinociceptive network of brain regions in patients with BPD. More specifically, tonic heat pain stimuli that were adjusted for individual pain sensitivity during an functional MRI study elicited higher activity in the dorsolateral prefrontal cortex together with reduced activity in the amygdala, perigenual anterior cingulate cortex and posterior parietal cortex in patients with BPD as compared with healthy age-matched controls [14]. We were also able to show that cessation of self-injurious behavior leads to a normalization of pain sensitivity in patients with BPD [15]. In a recent study, we could demonstrate that heat stimuli – independent of painfulness – led to a reduction of stress-induced amygdala hyperactivity [16]. Also, painful heat stimulation following negative emotional pictures led to more negative coupling of the amygdala with the medial prefrontal cortex, suggesting a strengthening of emotion modulation by pain [Niedtfeld I *et al.*, Manuscript in Preparation]. Using another technique during functional MRI, script-driven imagery, we could demonstrate a breakdown of orbitofrontal cortex activity while BPD patients imagined a situation that led to self-injury [17]. This again underlines the close association of NSSIB with emotion dysregulation and disturbed impulse control in BPD.

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From a neurochemical point of view, the endogenous opioid system appears to play an important role in the context of NSSIB [18,19]. The endogenous opioid system is related to stress-induced analgesia, a mechanism related to NSSIB, as mentioned previously, and dissociation in these patients [20]. NSSIB can be reduced by treatment with the opioid antagonist naltrexone [21]. One potential mechanism, besides blocking opioid-mediated positive-reinforcement processes, is the reduction of stress-related dissociative symptoms by naltrexone [20], therefore reducing the need to terminate dissociative states by NSSIB.

However, the treatment of NSSIB is clearly focused on psychotherapy. Specific treatments have been developed for patients with BPD with dialectical behavior therapy (DBT), schema-focused therapy, mentalization-based therapy and transference-focused psychotherapy having demonstrated efficacy [101]. Among them, DBT has a clear focus on NSSIB and treatment studies have shown that DBT reduces NSSIB rates to approximately 50% [22,23]. DBT teaches specific skills to reduce aversive inner tension and dissociation and uses behavioral analyses to help the patient understand the antecedents and consequences of self-injury.

What do we not know?

Thus far, much of the research on patients' motives for NSSIB has focused on negative reinforcement (reduction of intense aversive emotions). The role of a wider spectrum of motives for NSSIB, including antidissociation, mood enhancing and positive reinforcement (e.g., to reach a kick or high), is as yet inconclusive. It is also unclear whether BPD patients using NSSIB are to be subdivided into subgroups – for example, a group who is primarily positively reinforced (i.e., patients seeking a kick or seeking social attention) versus another group of patients for whom NSSIB is primarily negatively reinforced (i.e., patients seeking relief from negative feelings). Identification of subgroups would be of interest from both a clinical and a neuropsychological perspective. Negative reinforcement requires different treatment strategies (e.g., stress-tolerance skills) than positive reinforcement (e.g., anticraving skills) and it has been speculated that different neuropsychological mechanisms are involved in patients who engage in positively versus negatively reinforced behaviors [24].

Another open question pertains to the specificity of the association between NSSIB and BPD (i.e., whether motives and background of NSSIB are more or less unique to patients with BPD or whether other diagnostic features are also of importance). NSSIB can be found frequently in patients with post-traumatic stress disorder, pointing to a relationship between NSSIB and traumatic life events [25]. A recent meta-analysis, however, only found a moderate correlation between experiences of sexual violence and NSSIB [26]. Motives for NSSIB in patients with post-traumatic stress disorder appear to be related to the termination of intrusive memories [25].

Besides post-traumatic stress disorder, other disorders have also been found to have elevated rates of NSSIB (e.g., dissociative disorders [27] or depressive disorders [28]).

A large amount of research is still needed until firm conclusions on the neurobiology of NSSIB can be drawn. We have some knowledge on the role of disturbed pain processing in the context of NSSIB (as reviewed earlier), but the question remains whether reduced pain sensitivity is a cause or a consequence of NSSIB or whether both are related to a third factor, such as emotion dysregulation [15]. As mentioned earlier, the endogenous opioid system has been the focus of interest in this context, but other systems (e.g., the glutamatergic or the dopaminergic system) have not been investigated so far. From an experimental psychopathology perspective (i.e., modeling pathological behavior under laboratory conditions), several aspects of NSSIB should be considered when designing studies on its neurobiological backgrounds. NSSIB is a complex behavioral pattern, which comprises – besides painful experience – other aspects such as tissue damage or seeing one's own blood flow. To model such complex behaviors under laboratory conditions is a difficult and challenging task. In a first attempt to investigate the role of tissue damage in the context of NSSIB, we recently used an incision paradigm with small cuts into the forearm conducted by an investigator. Preliminary results point to a reduction of subjective and objective measures of stress following the incision in patients with BPD, but not in healthy controls [Reitz S *et al.*, Manuscript in Preparation].

Taken together, a lot of research needs to be carried out to better understand self-injurious behavior in patients with BPD. This will hopefully lead to further destigmatization of this frequent form of psychopathology and to better treatment options for these patients.

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