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Differential neural processing of unpleasant haptic sensations in somatic and affective partitions of the insula in non-suicidal self-injury (NSSI)

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ABSTRACT

Altered perception and neural processing of pain have been observed during non-suicidal self-injury (NSSI). Evidence suggests that this phenomenon could be associated with the affective rather than the somatosensory dimension of pain. Sub-partitions of the insula have been suggested to process these different aspects differentially. In the present study, activation within the posterior, middle, and anterior partitions of the insula upon unpleasant electric stimulation was compared between subjects with a history of NSSI and healthy controls. Using functional magnetic resonance imaging (fMRI), we investigated a sample of 30 subjects, 14 of them with a lifetime history of NSSI. Unpleasant electric stimulation to the dorsum of the non-dominant hand was performed at four levels of increasing intensity. Significantly increasing posterior insula activation, which is likely to reflect the somatosensory aspects of unpleasant haptic sensations, was found upon parametrically increasing electric stimulation in both groups. By contrast, activation of the anterior insula, rather related to the more affective aspects of distressing stimuli, was significantly modulated only in the control group, but not in subjects with NSSI. These findings may support present hypotheses of altered processing of the more affective aspects of unpleasant or distressing experiences in NSSI, as a putatively relevant factor for understanding the etiology of this behavior.

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1. Introduction

Nonsuicidal self-injury (NSSI) denotes the direct and intentional destruction of body tissue without suicidal intent and without social acceptance (Lloyd-Richardson et al., 2007). Self-injurious behaviour commonly starts during adolescence and occurs in up to 28% of European adolescents at least once in their lifetimes (Brunner et al., 2014). A lifetime prevalence of repetitive NSSI (more than four times during the life span) has been described in about 9.5% of adolescents in an international comparison study (Plener et al., 2009). A longitudinal study demonstrated a considerable reduction of self-harm behaviours after adolescence, with prevalence rates of less than 2% within the past 6 months at the age of 20 years (Moran et al., 2012).

While NSSI in adults is predominantly seen as a symptom of borderline personality disorder (BPD), self-injurious behaviour in adolescents usually occurs without association to any other overt psychopathology (Stanford and Jones, 2009) or related to depression (Plener et al., 2009). Recently, these observations led to suggestions to classify NSSI as an autonomous syndrome. Accordingly, research criteria for diagnosing NSSI were integrated into the new DSM-5 (American Psychiatric Association, 2013).

Despite its prevalence and incidence, the neurobiology of NSSI in general and its association to pain processing in particular is not sufficiently understood. Previous studies investigating the mechanisms of NSSI were conducted in adults and were closely connected to BPD psychopathology. Approximately 70–80% of women with BPD report reduced pain perception during NSSI (Leibenluft and Goldberg, 1987). In line with this, Schmahl et al. (2006) reported less overall brain activity as assessed with functional magnetic resonance imaging (fMRI) upon heat stimulation as a potential neural correlate of altered nociception in patients with BPD (Schmahl et al., 2006).

Regarding regular physiological pain processing, the somatosensory cortices, the thalamus, the anterior cingulate cortex (ACC) and the bilateral insula were summarized as parts of the neural

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pain matrix (Wager et al., 2013). Interestingly, next to the processing of literal physical pain (e.g. as a consequence of burning or cutting), the pain matrix has also been demonstrated to be active during the processing of pain in a more metaphorical sense, like social distress or emotionally hurtful situations (Rainville et al., 1997; Eisenberger et al., 2003; Oertel et al., 2008; Kross et al., 2011).

Intuitively, the first dimension of pain is commonly described as a mere somatosensory perception, caused by the stimulation of peripheral nerve fibers. In line with the common understanding of functional neuroanatomy, neuroimaging experiments particularly linked the secondary somatosensory cortex and the posterior insula to these sensory-discriminating, perceptual aspects of pain (Peyron et al., 2000; Eisenberger and Lieberman, 2004; Wager et al., 2013). Secondly, an affective-motivational component of pain, which is associated with the feeling of unpleasantness at various degrees, has been described. Different neuroimaging studies identified the anterior insula and the anterior cingulate cortex (ACC) to play an important role in the processing of unpleasant feelings (e.g., Rainville et al., 1997; Peyron et al., 2000; Price, 2002), or negative and distressing emotional stimuli (e.g., Rainville et al., 1997; Peyron et al., 2000; Bush et al., 2000; Terasawa et al., 2013; Garfinkel and Critchley, 2013).

Previous work in subjects with NSSI reported unchanged processing of the somatosensory component in NSSI with stimulus intensities similarly discriminated by healthy controls and subjects with BPD including NSSI (Schmahl et al., 2004; Cardenas-Morales et al., 2010). Also for other modalities like vibration perception, thermoreception, position and tactile sensibility (Schmahl et al., 2004; Ducasse et al., 2014), no differences were reported between subjects with and without NSSI.

These observations have led to the assumption that differences in the processing of pain or unpleasant sensations in subjects with NSSI may rather be associated with the affective than the sensory dimension of pain. Using an experimental task with parametrically varying levels of unpleasant haptic stimulus intensities, we tested this hypothesis in the present study. We focused on the bilateral insula and the described functional partitions hypothesized to differentially process the different aspects of pain or other distressful stimuli. We hypothesized that NSSI may relate to altered processing of unpleasant stimuli, especially in the anterior insula, reflecting differences in processing the affective aspects of sensory stimulation at varying intensities. By contrast, no differences in modulated brain activity in NSSI subjects compared with healthy controls were expected for the posterior insula, rather involved in the sensory or perceptive aspects of processing unpleasant haptic experiences. To investigate the posterior-to-anterior gradient in the insula (Craig, 2011), as proposed not only for pain but also for other salient stimuli like sudden noise, we defined three subdivisions of the insula (anterior/middle/posterior) according to Kelly et al. (2012).

2. Methods

2.1. Subjects

The subjects comprised 30 young female subjects (mean age=22.4 years, SD=3.37), 27 of them right handed, who were recruited for this study. The project was approved by the Institutional Review Board of Ulm University, Ulm, Germany. Written informed consent according to the Declaration of Helsinki was obtained from each subject.

Fourteen subjects were included with a lifetime history of NSSI and a frequency of at least five episodes in 1 year within the past 8 years. These subjects were randomly recruited from a larger sample of patients that had presented themselves voluntarily with self-injurious behaviour at the Department of Child and Adolescent Psychiatry and Psychotherapy during adolescence. The mean time period of NSSI elapsed since the last episode of NSSI was 2.50 years (SD=2.90). Eight of these subjects had not engaged in NSSI during the past 12 months and six subjects had NSSI episodes within the past year before fMRI scanning. All subjects with NSSI mainly engaged in skin-cutting; other methods (like self-biting or skin scraping) were used additionally, but not consistently, in some cases. Two subjects reported borderline personality disorder (BPD) as a presumptive diagnosis for clinical treatment during adolescence, but they had no current clinical symptoms. Another participant reported symptoms indicative of BPD upon follow-up in-depth diagnostic assessment. A recent history of depression and agoraphobia was found in two other participants, respectively. No other psychiatric diagnoses, current psychiatric symptoms or neurological illness were detected. None of the participants displayed significant functional impairment in daily life due to their current psychiatric status. In one of the subjects with a recent diagnosis, current psychotropic medication status was not obtained, while the others did not take psychotropic medication. Sixteen control subjects were carefully screened to exclude any history of NSSI, psychiatric or neurological disorders, and they were matched to the NSSI subjects on age and education before inclusion in the study. To control for hormonal influences on neural processing, all fMRI data were acquired during the follicular phase of the menstrual cycle within 10 days after menstruation onset or after at least 14 days of continuous intake of oral contraception. Further demographic and clinical characteristics of the NSSI subjects and healthy controls are summarized in Table 1.

Table 1
Summary statistics of demographic, clinical and behavioural data.

	NSSI	HC	Statistic (NSSI > HC)	
Behavioural/Questionnaire data				
Age (years)	21.1 (2.51)	23.4 (3.72)	$t=-1.95$	$p=0.061$
BDI-II – total score	12.92 (9.32)	4.13 (6.00)	$t=3.08$	$p=0.005$
BIS – total score	64.7 (7.01)	57.7 (7.19)	$t=2.64$	$p=0.014$
Hurt-Feeling-Scale – total score	18.5 (2.30)	16.8 (1.77)	$t=2.27$	$p=0.032$
NSSI				
Mean total incidence*	316 (406.75)	–	–	–
Mean age of NSSI beginning (years)	13.5 (2.03)	–	–	–
Mean time since last NSSI (years)	2.50 (2.90)	–	–	–
Electrical stimulation				
Correct responses	15.92 (2.06)	15.81 (2.79)	$t=1.12$	$p=0.906$
Minimal perceptible intensity (mA)	4.49 (1.81)	5.24 (1.37)	$t=-1.28$	$p=0.211$
Maximum tolerable intensity (mA)	16.34 (7.68)	17.84 (6.80)	$t=-0.57$	$p=0.575$
Mean rating	2.42 (0.19)	2.61 (0.28)	$t=-2.05$	$p=0.051$

Values are means with standard deviation in round brackets; statistical values are *t*-values stem from two-sided unpaired *t*-tests with d.f.=28. Abbreviations: BDI: Beck Depression Inventory; BIS: Barratt Impulsiveness Scale; mA=milliampere.

* during the total NSSI-period

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