

Review

The Neurobiology of Non-suicidal Self-injury (NSSI): A review

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Abstract: Non-suicidal Self-injury (NSSI) is a relevant clinical problem with high prevalence rates in adolescence. Despite the high numbers of individuals with NSSI, the neurobiological background is still poorly understood. This review aims to present an overview from different fields of neurobiological research. Results from neuroimaging studies, as well as from studies on neurotransmitters, point to an insufficient stress response. Analgesia and hypalgesia are often reported from individuals with repetitive NSSI, supporting neurobiological alterations. Therefore NSSI can be understood as a coping strategy that serves to down-regulate high experienced emotions.

Keywords: Non-suicidal Self-injury, NSSI, neuroimaging, neurobiology, analgesia

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Non-suicidal Self-injury (NSSI) is defined as repetitive, intentional, direct injury of one's body tissue without suicidal intent, that is not socially accepted (Lloyd-Richardson et al., 2007). In current classificatory systems (DSM-IV-TR and ICD-10), NSSI is coded as a symptom of Borderline Personality Disorder (BPD). Due to its occurrence in individuals without BPD or any other psychopathology, there is an ongoing discussion to integrate NSSI as autonomous diagnosis in the upcoming DSM-5 (APA, 2012). With regards to suicidal intent, a broader "umbrella term" (Cloutier et al., 2010), Deliberate Self Harm (DSH), can also be found in the literature, describing self-harming behaviours including suicidal intent. DSH is widely described as: "An act with a non-fatal outcome in

which an individual deliberately did one or more of the following: Initiated behaviour (for example, self-cutting, jumping from a height), which they intended to cause self-harm, ingested a substance in excess of the prescribed or generally recognised therapeutic dose, ingested a recreational or illicit drug that was an act that the person regarded as self-harm, ingested a non-ingestible substance or object." (Madge et al., 2008). However, since NSSI is a risk factor for subsequent suicide attempts (Nock et al., 2006; Andover et al., 2012), the neurobiological background of these clinical phenomena may be comparable. Studies on NSSI in adolescence suggest prevalence rates between 3.7% one-year prevalence and 37.2% lifetime prevalence (Plener et al., 2010). In a recent review of 53 studies published between 2005 and 2011 on adolescent NSSI, a mean lifetime prevalence rate of 18% was reported (Muehlenkamp et al., 2012). NSSI has to be understood as transient phenomenon, since the first large longitudinal study showed a decrease of self harming behaviour in young adulthood (Moran et al., 2012).

The integrated theoretical model of the development and maintenance of NSSI (Nock, 2010)

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proposes that these behaviours function as a method of regulation of both affective experience and social situations in the occurrence of a stressful event. Although this leads to the assumption that an altered stress response is involved in NSSI, the neurobiological factors are still poorly understood. A better knowledge of the neurobiological substantiation of NSSI could help to develop a new understanding of these behaviours and inform new evidence-based treatment modalities. This selective review presents empirical findings in recent research, focusing on the neurobiological factors of NSSI. Intrapersonal vulnerability factors such as high aversive emotions and cognitions as well as a poor distress tolerance could possibly be moderated by genetic predispositions for high emotional and high cognitive reactivity and by environmental adversities. Different brain morphology and neuronal activity in patients with NSSI or Borderline Personality Disorder (BPD) compared to healthy controls can be linked to divergent emotional and physical pain perception. An involvement of lipids in the occurrence of NSSI has been suggested (Roaldset, 2010; Garland, 2007). Concerning neurotransmitters, a neurobiological model of NSSI (Sher & Stanley, 2009) suggests that abnormalities in the serotonergic, the dopaminergic and the opioid system as well as the hypothalamic-pituitary-adrenal (HPA) axis (cortisol) lead to an increased level of stress vulnerability. In the event of stress, NSSI might therefore be used in order to restore an altered opioid-homeostasis. Analgesia or hypalgesia experienced by individuals with repetitive NSSI strongly suggests a neurobiological involvement.

Methods

For this selective review, the literature search was performed using MEDLINE, including studies published within the last 20 years. Key words used were: “self-injury”, “NSSI”, “non-suicidal self-injury”, “self harm”, “deliberate self harm”, “DSH”, “self-mutilation” and “auto mutilation”. References from textbook articles (Osuch & Payne, 2009; Sher & Stanley, 2009) were also searched for and – if applicable – included into the review. As often terminology is unclear and classification issues are still unsettled with regards to the distinction or possible overlaps between suicidal behaviours and NSSI (Skegg, 2005), we have included papers that also represent acts of suicidal behaviours in addition to NSSI (such as papers on DSH or parasuicidal behaviours). Borderline Personality Disorder (BPD) studies were selected, as up to 70-80% of patients with BPD also show NSSI (Schmahl et al., 2004) and neuroimaging studies (especially involving adolescents) are rare.

Results

Genetic predisposition for high emotional/ cognitive reactivity

Considering the complexity of gene-behaviour relationship and the development of psychopathology, it would be inadequate to consider the existence of one gene for NSSI. Results of studies on the genetic background of NSSI and DSH are mostly linked to genes involved in serotonergic neurotransmission, but findings are still inconsistent.

In a study of N=252 children and adolescents, those with one or two copies of the short allele in the promoter region of the serotonin transporter gene 5-HTTLPR showed the highest levels of BPD symptoms (Hankin et al., 2011). On the other hand, Maurex et al. (2010) did not find a relationship between 5-HTTLPR and suicidal and self-injurious behaviour in women with BPD (N=77). In accordance with this finding, results from a meta-analysis by Lin and Tsai (2004) did not state an association between suicidal behaviour and the 5-HTTLPR serotonin transporter gene.

According to Pooley et al. (2003), the tryptophan hydroxylase (TPH A779C, rs1799913) allele occurred more often among people with DSH (n=129) than people without DSH (n=329; OR: 1.38, 95% CI 1.02-1.88; p=.03). However, no Bonferroni correction for multiple comparisons was performed. The other polymorphisms studied, being within the 5-HT transporter gene (5-HTTLPR S/L), the monoamine oxidase A gene (MAOA G941T, rs1799835), the 5-HT1B receptor gene (HTR1B G861C, rs6296), the 5-HT2A receptor gene (HTR2A T102C, rs6313), and the 5-HT2C receptor gene (HTR2C Cys23Ser, rs6318), were not associated with DSH.

Evans et al. (2000) determined genetic polymorphisms of TPH and the 5-HT_{2c} receptor in N=376 adults. There was a weak correlation between the L allele of TPH in males and impulsiveness, but no correlation between impulsiveness and the TPH intron7. Males, but not females, with higher impulsiveness scores were more likely to have a 5-HT_{2c} serine variant. On the other hand, there were no differences in the level of impulsiveness between subjects with repeated DSH and subjects who did not engage in DSH.

In a sample of N=195 patients with depression (of which n=46 engaged in “self mutilation”), Joyce et al. (2006) found that the T allele of the G-protein β_3 (GN β_3) was a risk-factor for self mutilation (OR: 3.96). The odds ratio increased, if the sample was limited to patients aged 18 to 24 (OR: 9.21). The association between self mutilation and GN β_3 remained valid, when other confounding variables like childhood sexual abuse and BPD were accounted for. Despite this finding, the mechanism of how the T allele of GN β_3 might

influence self mutilating behaviours still remains unclear.

Overall, the results on genetic predisposition for NSSI and DSH are still very inconsistent. Genes involved in serotonergic neurotransmission, like 5-HTTLPR or TPH, were associated with DSH or related psychopathology in some studies, but no association was reported in other publications. An involvement of the T allele of GN β 3 in self-mutilation was found by Joyce et al. (2006), but the influence of this finding on the clinical manifestation can not be explained yet.

Altered physiological reactivity

There is mixed evidence concerning an altered physiological reactivity in individuals with NSSI or related psychopathology. However, a decrease of physiological tension when imagining an act of NSSI has been reported repeatedly.

Nock and Mendes (2008) compared 62 adolescents with a history of NSSI to 30 matched controls without NSSI. Physiological hyperarousal (measured by skin conductance) could be found among adolescents with NSSI, when confronted with a distressing task. Contrary to this, Kaess et al. (2012) did not find differences in the heart rate of adolescents with NSSI (n=14) and healthy controls (n=14) in a standardized psychosocial stress protocol. Also, in a study on parasuicidal adolescent girls (n=23) and n=23 healthy controls, no differences in heart rate or skin conductance were reported; although there was greater respiratory sinus arrhythmia activity (Crowell et al., 2005).

Imagining an act of NSSI can decrease physiological tension, as measured e.g. per skin conductance and heart rate in individuals with self-injurious behaviours (Brain et al., 1998, Haines et al., 1995). A rapidly lowered heart rate was also detected in rhesus monkeys with repeated self-injurious behaviours, after biting themselves (Novak, 2003).

A physiological hyper arousal in individuals with NSSI was found in one study, but could not be replicated in other studies, which could be due to different methods of stress induction and measurements (heart rate and skin conduction). Two studies reported a reduction of physiological arousal when imagining self-injury.

Neuroimaging

Several studies have found abnormalities in the brain morphology and neuronal activity in patients with NSSI or BPD. In particular, hyperarousal in limbic structures, like the amygdala and the anterior cingulate cortex (ACC), seem to be common outcomes of fMRI studies.

Brain morphology

Greater numbers of parasuicidal behaviours were associated with an increased pituitary gland volume (PGV), in adolescent patients with BPD

(Jovev et al., 2008). Patients with increased pituitary volumes showed higher levels of post-dexamethasone cortisol, which in turn could be due to a hyperactive HPA axis (Garner et al., 2005). In a sample of n=15 BPD patients and 15 healthy controls, Whittle et al. (2009) found a decreased left ACC volume. This decrease was positively correlated with the number of parasuicide episodes.

Brunner et al. (2010) compared n=20 adolescents with BPD to n=20 adolescents with a current psychiatric disorder to n=20 healthy controls. Changes were also found in the clinical control group. Nevertheless, BPD patients and clinical controls showed a reduced grey matter in the left orbitofrontal cortex (OFC) and the dorsolateral prefrontal cortex (DLPFC), leading to the assumption that BPD symptoms in adolescence do not lead to specific morphological changes.

Functional abnormalities

In an fMRI study, adult patients with BPD (n=11) showed a decreased activity in the OFC while listening to a standardized script which described a stressful situation leading to NSSI, compared to healthy controls (n=10). An increase in the activation of the DLPFC was described in patients with BPD in stressful situations. During imagining NSSI, activity in the right posterior ACC, which is responsible for emotional response and cognitive assessment, decreased (Kraus et al., 2010). Supporting these findings, when heat induced pain was applied in twelve patients with BPD and NSSI and twelve healthy controls, an increased activity in the DLPCF and a decreased activity in the posterior parietal cortex, the perigenual ACC and the right amygdala were found in BPD patients compared to healthy controls (Schmahl et al., 2006).

Niedtfeld et al. (2010) confronted adults (n=20 with BPD and n=23 healthy controls) with a combination of warm and painful stimuli as well as neutral and negative pictures of the International Affective Picture System (IAPS). fMRI results showed a significantly higher activation in the amygdala, the ACC and the insula in patients with BPD during the presentation of both neutral and negative pictures. This suggests differences in emotional processing in BPD patients. No changes in activation in the amygdala and the ACC were found during the exposure to warm and painful stimuli compared to healthy controls, although activation decreased in all participants when exposed to sensory stimuli. This could be due to a shift in attention from emotional pictures to sensory experience (Niedtfeld et al., 2010). Also, in a study of n=9 adolescent patients with NSSI and n=9 healthy controls, Plener et al. (in press) found an increased activation in the amygdala and the ACC for all emotional pictures being viewed.

In summary, there is only little evidence for changes in brain morphology of patients with parasuicidality or BPD, and these findings are not

specific for NSSI. Results from fMRI studies point to the direction of hyper activation of limbic structures in patients with NSSI and BPD.

Lipids

Low cholesterol concentrations and low levels of essential fatty acids (EFAs) have been associated repeatedly with self-mutilating behaviours. Results on low cholesterol and suicide-attempts are inconsistent.

Garland et al. (2007) found that patients with DSH (n=40) had a significantly lower cholesterol level, lower levels of EFA and lower n-3 and n-6 EFA levels than healthy controls (n=40). In a randomised controlled double-blind study, n=22 patients with repeated self-harm received n-3 EFA for 12 weeks and n=27 controls with repeated self-harm received a placebo. Patients treated with n-3 EFA showed significant improvements regarding e.g. depression and suicidality compared to the control group. Other measures like impulsivity or aggression did not differ (Hallahan et al., 2007).

Soldiers engaging in autoaggressive behaviours (n=33) showed significantly lower levels of total serum cholesterol than soldiers with other psychopathology (n=21) (Florkowski et al., 2001). In a sample of 254 psychiatric patients, Roaldset et al. (2010) found significantly lower triglyceride levels in patients who showed suicidal and self-mutilating behaviour. Low High-Density Lipoprotein (HDL) concentration was a significant predictor for inpatient self-mutilation. However, due to a low positive predictive value and a low sensitivity, these measures cannot be considered worthwhile for clinical screening.

In a sample of N=1770 healthy women, among those with low levels of high-density lipoprotein cholesterol (HDL-C; <40 mg/dl), the prevalence of suicide attempts was significantly higher (OR=2.93, 95% CI=1.07–8.00, p<0.05) than in women with higher levels of HDL-C (Zhang et al., 2005). However, differences became statistically insignificant after multivariable adjustment and no differences were found in men. Similar associations between suicide and low levels of serum cholesterol have been shown repeatedly, although there are also studies reporting contrary findings (e.g. Deisenhammer et al., 2004; for an overview see e.g. Zhang et al., 2005). Several studies (e.g. Atmaca et al., 2003; Deisenhammer et al., 2004) found lower cholesterol levels in patients who used more violent methods to attempt suicide.

In summary, low cholesterol levels and low levels of EFA have been associated with self-injury consistently. Nevertheless, since serum cholesterol levels are quite possibly influenced by factors like age, gender, medication or diet, they might not serve as reliable markers. Inconsistent results concerning low level cholesterol and suicide attempts may be due to those factors.

Neurotransmitters

From a neurobiological perspective, the serotonergic, the dopaminergic, and the opioid system as well as the HPA axis are being discussed to be involved in the development and maintenance of NSSI (Stanley, 2010).

Serotonin

Some, although inconsistent, evidence for a serotonin deficiency in association with self harming behaviours could be found. Overall, serotonin was more related to aggressive or violent behaviours in general than self-injurious behaviours in particular.

Abnormalities within the serotonergic (5-HT) system have been mentioned repeatedly in the context of NSSI (McCloskey et al., 2009; Hankin et al., 2011; Pies & Popli, 1995). There is evidence that decreases in 5-HT are correlated with impulsive and aggressive behaviours, suicide attempts and depression (Witte et al., 2009; Crowell et al., 2005; McCloskey et al., 2009; Hankin et al., 2011). In a recent study (McCloskey et al., 2009), experimentally reduced serotonergic activity led to more self aggression among adults with (n=16) and without (n=16) Intermittent Explosive Disorder. Crowell et al. (2008) reported, that adolescents' peripheral 5-HT levels, in interaction with negativity and conflict in mother-child dyads, explained 64% of the variance of self-injury. 5-HT levels can be affected by diet, which was not accounted for in this study. However, participants were asked to abstain from the intake of food on the day the blood sample was taken. Although peripheral 5-HT levels are not indicative of brain serotonin levels in humans, these findings can be linked to relevant traits like aggressive behaviours, which could point to an involvement of 5-HT in NSSI (Crowell et al., 2008).

Monkeys engaging in self injurious behaviours (self-biting) also showed a decreased function of the 5-HT system (Tiefenbacher et al., 2005). On the other hand, no differences in the serum serotonin levels of bushbabies with self-injurious behaviours and those without could be found (Watson et al., 2009).

Other studies did not find associations between platelet serotonin and NSSI (Garland et al., 2007; Roaldset et al., 2010). No differences in the levels of 5 hydroxyindolacetic acid (5-HIAA), a serotonin metabolite, could be found between patients with and without NSSI (Stanley, 2010).

Dopamine

There is only limited evidence for increased dopamine levels being connected to NSSI. Most conclusions for a correlation of dopamine and NSSI were drawn from studies on populations with BPD or animal studies (Tiefenbacher et al., 2005; Osuch & Payne, 2009). From animal research, an increase of self-biting with dopamine agonists was reported in studies of mice after amphetamine exposure (Kasim

& Jinnah, 2003). No differences in the levels of homovanillic acid, a dopamin metabolite, could be found between patients with NSSI and patients without a history of NSSI who were all diagnosed with a Cluster B personality disorder and had committed a suicide attempt (Stanley, 2010).

Cortisol

Consistent results from studies on cortisol in individuals with NSSI showed a reduced cortisol secretion. The HPA-axis interacts with endogenous opioids and serotonergic mechanisms, and is involved with the level of secretion of cortisol. Experiencing stress is associated with an elevated secretion of cortisol (Heim et al., 2000). According to Heim et al. (2000), post-traumatic stress disorder (PTSD), chronic stress and stress-related bodily disorders are correlated with a low baseline cortisol secretion. In a recent study Kaess et al. (2012) reported, that adolescents who engaged in NSSI (n=14) showed a hyporesponsive HPA-axis in stressful situations, compared to healthy controls (n=14).

These findings are comparable to several animal studies. Monkeys with self-injurious behaviours that were subsequently exposed to stress, showed a hyporesponsive HPA-axis when experiencing acute stress (Tiefenbacher et al., 2005). According to Tiefenbacher et al. (2005) it is still unclear whether a hyporesponsive HPA-axis leads to self-injurious behaviour, or if self-injurious behaviour influences the responsiveness of the HPA-axis. Another study on rhesus monkeys also reported lower serum cortisol in monkeys biting themselves, when compared to monkeys not hurting themselves after stress (relocation). However, this difference was not significant (Davenport et al., 2008). A study of bushbabies showed significantly lower levels of plasma cortisol in animals with self-injurious behaviours (Watson et al., 2009).

Endogenous opioids

Lower levels of endogenous opioids were found in individuals with NSSI repeatedly and could possibly explain an “addictive quality” of these behaviours.

Endogenous opioids are associated with various disorders, like BPD or pervasive developmental disorders (PDD) of which NSSI and self-mutilation are considered to be symptoms. Opioids are involved in pain-perception and addictive behaviours. Addictive qualities of NSSI have been suggested in past studies (Nixon et al., 2002; Resch et al., 1993), thus underlining the possible importance of endogenous opioids in NSSI.

Altered opioid levels can be found in patients with a history of repeated NSSI (Coid et al., 1983; Sher & Stanley, 2008; Sher & Stanley, 2009). According to a homeostasis model of NSSI (Stanley, 2010), childhood neglect and genetic vulnerability lead to a chronically lower level of endogenous

opioids, which in the event of stress can be restored by engaging in NSSI. In a study of N=29 patients who had committed suicide attempts and were diagnosed with a Cluster B personality disorder (n=14 with NSSI, n=15 without NSSI), Stanley et al. (2010) found that cerebrospinal fluid (CSF) β – endorphin and met-enkephalin levels of endogenous opioids were significantly lower in patients with a history of NSSI.

In summary, the most consistent results from studies on the involvement of neurotransmitters in NSSI are reduced levels of cortisol and endogenous opioids, which suggest an altered stress response. Findings on other neurotransmitters like serotonin and dopamine are inconsistent and imply further research in this field.

Analgesia/opiate hypothesis

Analgesia or hypalgesia are common phenomena reported by patients engaging in NSSI repeatedly. An explanation for this phenomenon could be lowered levels of endogenous opioids (as described above).

According to Nock and Prinstein (2005), repetitive NSSI is typically performed in the absence of physical pain. Among patients with BPD and NSSI, 70% to 80% reported hypalgesia or analgesia (Schmahl et al., 2004). This could be related to the altered levels of endogenous opioids, repeatedly described in patients with NSSI (Stanley, 2010; Sher & Stanley, 2009). According to Tiefenbacher et al. (2005), monkeys directed self-injurious behaviours to body areas that are associated with acupuncture analgesia. Schmahl et al. (2004; 2006) found that patients diagnosed with BPD reported lower pain ratings and higher pain thresholds compared to healthy controls when confronted with heat pain stimulation. Ludäscher et al. (2009) compared n=24 patients with BPD who currently engaged in NSSI (nNSSI=13) and patients who used to engage in NSSI (nExNSSI=11) with n=24 healthy controls. Patients who currently performed NSSI showed the highest pain threshold, followed by the ExNSSI group, which was then followed by the healthy control group. Since the pain threshold apparently decreased after stopping NSSI, these findings suggest that hypalgesia is habitual. In contrast, Nock et al. (2006) found a positive correlation between the number of methods used in NSSI and the experience of physical pain in N=89 adolescents engaging in NSSI.

Discussion

The understanding of neurobiological mechanisms with regards to NSSI is still at its very beginning, however within recent years a larger number of studies have been published. We reviewed the existing literature and found studies with regards

to genetic background, altered physiological reactivity, levels of neurotransmitters, lipids and differences in brain activation.

Studies investigating the genetic background of NSSI and DSH primarily showed results in association with genes involved in serotonergic neurotransmission (Hankin et al., 2011, Pooley et al., 2003, Evans et al., 2000). Since these findings are inconsistent, currently, no candidate genes emerge for further studies, thus leaving the field open to a more exploratory approach.

Studies consistently showed altered physiological reactivity in individuals with NSSI (Brain et al., 1998; Nock & Mendes, 2008; Haines et al., 1995). Only recently Kaess et al (2012) had shown no differences in the heart rate in adolescents with NSSI in a social distress task. This is contrary to the findings of Nock & Mendes (2008) (who also enrolled adolescents in their study), and can possibly be explained by the different assessment procedures used. Also, Kaess et al. (2012) reported other parameters (such as cortisol response, see later), that were changed in these adolescents, leading to assume that differences in physiological reactivity are present in NSSI.

Results from fMRI studies point into the direction of a hyper arousal of limbic structures (such as the amygdala and the ACC) (Niedtfeld et al., 2010; Plener et al., in press). Activation of these structures seems to decrease both after induction of painful stimuli (Schmahl et al., 2006), as well as after imagining an act of NSSI (Kraus et al., 2010). This evokes the assumption, that NSSI serves a stress regulating purpose in an otherwise highly activated limbic system.

Findings from research in blood lipid levels point to the fact that lower cholesterol levels can be found in patients with DSH (Garland et al., 2007) and lower levels of triglycerides seem to be predictive of suicidal and self-mutilating behaviour (Roaldset et al., 2010). However, due to an overall low sensitivity, the use of cholesterol levels as a screening tool for NSSI seems not suitable.

Looking at the level of neurotransmitters, there is ample evidence for an association between impulsive behaviours and a lack of serotonin. However, as NSSI can often not be described as a predominantly impulsive behaviour (see Janis & Nock, 2009), there is only little evidence for a supporting mechanism of serotonin deficiency in the maintenance of NSSI, such as the study relating mother-child dyads and peripheral serotonin with NSSI (Crowell et al., 2008). The same holds true for an involvement of dopaminergic neurotransmission in normally developed individuals with NSSI. In a recent study (Stanley et al., 2010) no differences in levels of serotonin or dopamine metabolites could be found in the CSF of patients with NSSI, a finding, that – with regards to dopamine metabolites – was

also described in studies of primates (Tiefenbacher et al., 2005). Findings from studies on cortisol and the HPA axis in individuals (and animals) with NSSI consistently showed an altered cortisol response (e.g. Kaess et al., 2012, Tiefenbacher et al., 2005). Also studies showed changed levels of endogenous opioids, therefore pointing to an altered stress response in individuals with NSSI and possibly explaining an addictive quality of NSSI that has been described in individuals with severe NSSI (Nixon et al., 2002). These endogenous opioids may serve as an explanation for the consistently reported phenomenon of hypalgesia or analgesia in patients with repetitive NSSI, which seems to be reversible after ending NSSI (Ludäscher et al., 2009).

These findings from different fields of neurobiological research can be interpreted in the direction of an insufficient stress response. This is underscored by the most consistent results from neuroimaging studies, as well as studies on cortisol levels and endogenous opioids. Taken together these neurobiological factors support the integrated theoretical model of NSSI proposed by Nock (2010).

At this point, the evidence emerging from genetic studies is still inconsistent, so that the genetic predisposition for high emotional or cognitive reactivity proposed as distal factor by Nock (2010) still needs further exploration. There is evidence for the intrapersonal vulnerability factor of high aversive emotions, as well as the interpersonal vulnerability factor of poor social problem solving (supported by altered neurochemical reactions in social stress tasks).

It has to be kept in mind, that many studies observed NSSI in the context of other psychiatric disorders, such as BPD or depression. This is understandable, given the fact that NSSI is not represented as disorder of its own in the ICD-10 or DSM-IV. As a NSSI-syndrome has been proposed for inclusion in the upcoming DSM 5, this could increase research that is solely focused on NSSI.

A better understanding of the underlying neurobiology of NSSI can help to foster effective treatment, especially with regards to psychopharmacological interventions. There is still no agreed-upon pharmacological treatment of NSSI (Plener et al., 2009), and proposed treatment with serotonergic agents (Roberts, 2003) has shown to be able to increase NSSI (Donovan et al., 2000). There is also a lack of studies regarding the psychotherapeutic treatment of adolescents with NSSI (Wilkinson & Goodyer, 2011). A psychopharmacological treatment directly addressing NSSI should therefore be administered with caution, however it seems helpful to address existing psychiatric disorders (e.g. depression) with a psychopharmacological intervention, based on the respective treatment guidelines.

Conflict of interest

R.G. reports no conflict of interest. P.L.P. has received travel reimbursement from Lundbeck Pharmaceuticals.

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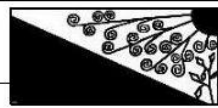
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