BJPsych Open (2025)
11, e163, 1–9. doi: 10.1192/bjo.2025.10765

Differential neural activity associated with emotion reactivity and regulation in young adults with non-suicidal self-injury

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Background

Emotional processing difficulties represent the core psychopathology of non-suicidal self-injury (NSSI), yet the underlying neural mechanisms remain unclear.

Aims

To investigate neural alterations associated with emotion reactivity and regulation in individuals with NSSI and examine whether emotional valence is related to these neural patterns.

Method

During functional magnetic resonance imaging scans, unmedicated young adults with NSSI ($n = 29$) and matched controls ($n = 25$) completed an emotion regulation task in which they viewed pictures of different emotional categories with instructions to either attend to or regulate their emotions.

Results

Individuals with NSSI showed increased neural activation in the right superior temporal gyrus (STG), right parahippocampal gyrus and right supramarginal gyrus during negative emotion reactivity and increased activation in the right middle temporal gyrus and left STG during positive emotion reactivity. Conversely, those with NSSI exhibited reduced activation in the left supplementary motor area, left inferior frontal gyrus, right putamen, right thalamus and right STG during negative emotion regulation and reduced activation in the left ventral striatum during positive emotion regulation. Notably, both hyperactivation of the STG during negative emotion reactivity and hypoactivation of the supplementary motor area during negative emotion regulation were associated with emotion dysregulation in individuals with NSSI.

Conclusions

We observed distinct neural patterns of emotional processing among individuals with NSSI, characterised by hyperactivation during emotion reactivity and hypoactivation during emotion regulation. Our findings provide a neurophysiological basis for therapeutic interventions that facilitate adaptive emotional processing in individuals with NSSI.

Keywords

Non-suicidal self-injury; self-harm; emotion reactivity; emotion regulation; fMRI.

Non-suicidal self-injury (NSSI) refers to deliberate self-inflicted damage to the body without suicidal intent.[1] Meta-analyses have estimated the prevalence of NSSI in community samples as 17.6% in adolescents[2] and 13.4% in young adults.[3] NSSI typically emerges in early adolescence and declines as individuals transition into adulthood. However, a 10-year longitudinal study found that among individuals who reported recurrent NSSI in adolescence, approximately 36% continued engaging in NSSI into young adulthood, and this was associated with tenacious difficulties in managing emotional distress.[4] Furthermore, a recent study suggested a second peak in NSSI onset during early adulthood,[5] a pattern that has been linked to increased suicide risk.[6] These findings underscore the importance of sustained attention to NSSI in young adults in clinical and research contexts, as its persistence beyond adolescence may indicate greater severity and long-term psychological distress.

NSSI has a lasting detrimental impact on an individual's mental health. Cohort studies have consistently found that persistent NSSI is associated with higher risks of emerging adverse clinical outcomes, including mental disorders and impairment in psychosocial functioning.[7,8] Furthermore, although NSSI itself is not intended to end one's life, it is a robust predictor of future suicidal ideation and behaviour.[1,7,8] A recent review of longitudinal studies found that NSSI often preceded suicide attempts, with higher NSSI frequency associated with an elevated risk of subsequent suicide attempts.[9] Despite the growing prevalence of NSSI and its significant impact on mental health, understanding of its fundamental pathophysiology remains in its early stages; furthermore, although several interventions have demonstrated efficacy, their effectiveness remains limited.[10]

Emotion dysregulation is a defining feature of NSSI, contributing to both its onset and persistence. A meta-analysis[11] found that individuals with NSSI exhibited significant impairments across multiple domains of emotion regulation, with all six subscales of the Difficulties in Emotion Regulation Scale (DERS)[12] positively associated with NSSI. Among these, limited access to effective regulation strategies and non-acceptance of emotional responses showed the strongest associations. It was also noted that individuals with NSSI often relied on maladaptive strategies that heightened emotional distress, such as rumination and suppression, while underutilising cognitive reappraisal, a key adaptive strategy that helps individuals to regulate emotion. These deficiencies increase vulnerability to NSSI and reinforce reliance on self-injury as a short-term regulatory mechanism, perpetuating the behaviour over time.[13] Given the critical role of emotion regulation in NSSI, a comprehensive investigation into its neural correlates is essential to provide insight into its underlying mechanisms and guide targeted interventions.

Although numerous studies have proposed a relationship between NSSI and deficits in emotional processing, the neural bases

https://doi.org/10.1192/bjo.2025.10765 Published online by Cambridge University Press

of these alterations in individuals with NSSI have not yet been fully elucidated. A few neuroimaging studies have identified functional alterations of the front-limbic network that is essential for emotional processing. For example, functional magnetic resonance (fMRI) studies found that adolescents with NSSI exhibited hyperactivation of the limbic regions, including the amygdala, hippocampus and anterior cingulate cortex, when viewing emotional images.[14] A previous study also found that individuals with NSSI showed altered activation in the limbic regions and the orbitofrontal cortex in response to both negative and positive images.[15] Similarly, differential frontoparietal activations during implicit response to emotional information in individuals with NSSI were found to be associated with limited emotional awareness.[16]

Previous neurophysiological research on NSSI has focused on emotion reactivity; research on emotion regulation has remained limited. Although closely related, emotion reactivity and regulation are distinct processes. Reactivity reflects immediate emotional responses, whereas regulation involves both automatic and effortful mechanisms to modulate these responses.[17] Investigating emotion regulation deficits in individuals with NSSI is essential for understanding whether these difficulties stem from heightened emotional responses, impaired regulation or a combination of both. Given evidence supporting the role of cognitive reappraisal as a potentially beneficial regulation strategy in this population,[18] examining the neural mechanisms underlying emotion reactivity and regulation may provide valuable insights into the neurobiological processes associated with NSSI. Such findings could contribute to refining intervention strategies to address emotion dysregulation in individuals with NSSI.[19]

In addition, research on positive emotion processing within this population is only beginning to emerge; as such, there is a substantial gap in our understanding compared with that for negative emotions. Accumulating evidence suggests that difficulties in positive emotion regulation contribute to pathophysiology across emotional disorders,[20] indicating a potential treatment target for NSSI. Indeed, evidence indicates that individuals with NSSI may encounter challenges in processing positive emotions as well as negative ones.[21] However, findings on positive emotion processing remain mixed, with some studies reporting no significant differences.[22] These inconsistencies may stem from variations in measurement methodology. Most empirical studies of positive emotional experiences in individuals with NSSI have relied on self-report and behavioural data. Moreover, previous research on the neural mechanisms of positive emotion processing in people with NSSI has been constrained by small sample sizes ($n = 15$) or the absence of control groups.[15,16] Such methodological limitations reduce generalisability and increase susceptibility to confounding factors, such as psychiatric medication use, making it difficult to determine whether observed neural differences are specific to NSSI rather than attributable to broader psychopathology. To address these concerns, a more comprehensive investigation with robust sampling methods and appropriate control groups is essential for understanding emotional processing in individuals with NSSI.

Thus, in the present study, we aimed to investigate neural patterns of emotional processing in unmedicated individuals with NSSI by exploring emotion reactivity and regulation in tandem. Specifically, we examined neural responses during passive exposure to emotional stimuli (i.e. emotion reactivity) and active engagement in cognitive reappraisal (i.e. emotion regulation). In addition, we sought to delineate neural patterns during processing of both positive and negative emotions in individuals with NSSI.

Our initial hypothesis was that individuals with NSSI would exhibit increased neural responses in regions involved in subjective emotional experience, such as the amygdala and insula, during emotion reactivity. Given the established roles of the dorsolateral prefrontal cortex, ventrolateral prefrontal cortex and supplementary motor area (SMA) in successful emotion regulation,[23] we hypothesised that individuals with NSSI would show reduced activation of these brain regions during emotion regulation. We also proposed that these neural changes would be associated with the severity of emotion dysregulation in individuals with NSSI. Last, we hypothesised that individuals with NSSI would exhibit neural alterations during the processing of both negative and positive emotions.

Method

Participants and procedure

Participants were recruited for this study through online advertisements on social media. All participants were required to be between 19 and 29 years old and be native speakers of Korean, fluent in both reading and writing. NSSI participants were included if they had reported a history of at least 5 days of NSSI in the past 12 months. The exclusion criteria for all participants were as follows: (a) any history of psychosis in themselves or first-degree relatives; (b) any presence of a major physical illness or head injury; (c) pharmacological or psychological treatment within the past month; (d) presence of a specific suicidal plan, intense urges to attempt suicide or a suicide attempt within the past week; (e) an estimated IQ of less than 80, as measured by the short form of the Wechsler Adult Intelligence Scale, fourth edition (WAIS-IV);[24] and (f) any contraindications with respect to MRI.

Telephone screenings were conducted by trained research assistants and graduate students to assess inclusion and exclusion criteria. Eligible individuals then underwent clinical interviews by licensed clinical psychologists or supervised graduate students in clinical psychology. Before the interview, participants completed the Korean version of the Inventory of Statements about Self-Injury,[25] and interviewers reviewed their responses to verify eligibility. Diagnostic evaluations were further conducted using the Structured Clinical Interview for DSM-5 (SCID-5)[26] for the NSSI group and the Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-Patient Edition (SCID-I/NP)[27] for the control group. To estimate IQ, the short form of the Korean WAIS-IV[28] comprising the information, matrix reasoning, arithmetic, and coding subsets, was administered by a trained clinical psychologist and graduate students.

The study included 42 unmedicated individuals with NSSI and 43 controls matched for age, gender and handedness. Although participants were matched on these variables, we used a cross-sectional comparative study design rather than a strict case–control framework to assess group differences at a single time point.[29] To ensure data quality and suitability for whole-brain analysis, two authors independently conducted visual inspections of all fMRI scans. Data-sets were excluded only when these authors reached consensus that the image quality or artefact level rendered the data unsuitable for whole-brain analysis. The most frequent reason for exclusion was misalignment of the field of view (FOV), which occurred primarily during the early phases of data collection and resulted in incomplete coverage of dorsal and occipital cortical areas. In addition, participants with excessive head motion (>3 mm translation or >3° rotation) during tasks were excluded. For the NSSI group, participants were excluded owing to incomplete brain coverage ($n = 9$), excessive head movement during the scan ($n = 2$) or failure to attend the scan ($n = 2$). For the control group, participants were excluded owing to incomplete brain coverage ($n = 8$), synchronisation problems ($n = 2$), failure to attend the scan ($n = 4$), reported lifetime episodes of NSSI ($n = 3$) or poor

https://doi.org/10.1192/bjo.2025.10765 Published online by Cambridge University Press

performance on the emotion regulation paradigm (ERP) task due to loss of concentration ($n = 1$). Thus, the final participant group consisted of 54 young adults, including 29 NSSI participants and 25 controls. All participants provided written informed consent before initiating the study and were debriefed upon completion of their participation. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2013. All procedures involving human participants were approved by the Institutional Review Board of Korea University (KUIRB-2020-0031-06).

Psychometric scales

All participants completed a series of self-report assessments. Emotion reactivity was evaluated using the Emotion Reactivity Scale (ERS),[30] which evaluates three components: emotion sensitivity, intensity and persistence. Higher scores on the ERS indicate greater emotion reactivity. The DERS[12] was used to assess the severity of emotion dysregulation across six subscales: emotional awareness, emotional clarity, impulse control, engaging in goal-directed behaviour, non-acceptance of emotional responses and access to effective strategies. Owing to a negative factor loading in the Korean version of DERS,[31] item 17 was excluded from scoring. Higher scores on the DERS denote more severe emotion dysregulation. The Beck Depression Inventory-II (BDI-II) was used to assess depressive symptoms, with higher scores indicating more severe depression.[32]

MRI acquisition and preprocessing

The fMRI data were acquired using a 3T Siemens Magnetom Trio scanner at the Korea University Brain Imaging Center with a 12-channel head coil. For functional scanning, echo planar imaging T2-weighted functional images were acquired using the following parameters: repetition time 2000 ms, echo time 26 ms, fractional anisotropy 77°, FOV 224 mm, 3.5 mm^3 voxel sizes, slice thickness 3.5 mm, 32 slices. T1-weighted structural scans for preprocessing of functional images were acquired using the following parameters: repetition time 2400 ms, echo time 2.19 ms, fractional anisotropy 8°, FOV 272 mm, 0.8 mm^3 voxel sizes, slice thickness 0.85 mm, 224 slices.

Statistical Parametric Mapping (version 12 for macOS; Functional Imaging Laboratory, London, UK; https://www.fil.ion.ucl.ac.uk/spm/) was used for fMRI data preprocessing and analysis. Slice timing correction was applied for interleaved slice acquisition, using the middle slice as the reference. Realignment and unwarping were performed to correct head motion, followed by coregistration of the mean functional image to each participant's T1-weighted structural image for spatial alignment. The resulting images were normalised to Montreal Neurological Institute space (East Asian ICBM template) with a $2 \times 2 \times 2$ mm voxel resolution. Spatial smoothing was applied using a Gaussian kernel with 6 mm full width at half-maximum.

ERP task

During the fMRI scan, participants completed an ERP task according to well-established protocols validated in prior research.[33] Participants were instructed to either 'attend to' or 'regulate' their emotional experiences induced by emotionally valenced photographs according to the respective cues displayed on-screen. In the attend condition, participants were asked to allow their emotions to be experienced naturally without attempting to modify them. Conversely, in the regulate condition, participants were asked to downregulate evoked emotions by cognitive reappraisal, a distancing strategy that involved altering their psychological perspective on the emotional stimulus.[34] Before the scan, participants completed practice trials to familiarise themselves with distancing strategies.

The ERP task included 45 pictures across three emotional categories (18 negative, 18 positive and nine neutral) selected from the International Affective Picture System[35] and the Open Affective Standardized Image Set.[36] The pairing of instructions and emotional category resulted in five conditions: attend-negative, attend-positive, attend-neutral, regulate-negative and regulate-positive. Neutral images were administered solely in the attend condition (i.e. attend-neutral condition), because they induced minimal emotional arousal and were deemed insufficient for regulation purposes.[37]

The ERP consisted of 45 trials across three runs arranged in a pseudo-randomised fashion. Each trial included an instruction cue (2 s), stimuli presentation (8 s), a rating phase (4 s) and a jittered fixation (2 s to 4 s, averaging 3 s). During the rating phase, participants rated their emotional state on a five-point Likert scale (1: very unpleasant, negative feelings; 5: very pleasant, positive feelings). After scanning, participants rated the valence, arousal and intensity of five individual emotions (sadness, fear, disgust, anger and happiness) for each stimulus.

Statistical analyses

A general linear model was used to analyse individual fMRI data, assessing the relationship between brain activity and task conditions. Individual statistical maps were generated to examine blood oxygen level-dependent (BOLD) signal changes across seven conditions: (a) attend-negative, (b) attend-positive, (c) attend-neutral, (d) regulate-negative, (e) regulate-positive, (f) instructions and (g) ratings. Each condition was modelled as a separate regressor and convolved with a canonical hemodynamic response function. To account for motion-related artefacts, six motion parameters from the realignment step were included as nuisance regressors. In addition, instruction cues and ratings were modelled as nuisance regressors to control for potential variance attributable to these elements. A high-pass temporal filter (128 s) was applied to remove low-frequency signal drifts.

Individual contrast maps were generated for each participant using a general linear model approach to identify task-related neural activity. Four key contrasts were defined to examine BOLD signal changes associated with emotion reactivity and regulation. Emotion reactivity was assessed by comparing BOLD responses for the attend-negative and attend-positive conditions with those for the attend-neutral condition (negative emotion reactivity: attend-negative > attend-neutral; positive emotion reactivity: attend-positive > attend-neutral). Emotion regulation was examined by comparing BOLD activity during the regulate-negative and regulate-positive conditions with that for their respective attending conditions (negative emotion regulation: regulate-negative > attend-negative; positive emotion regulation: regulate-positive > attend-positive). For whole-brain analyses, considering the modest sample size, we applied a voxel-level threshold of $P < 0.001$ with a cluster extent of $k = 20$. This approach has been discussed as providing a stringent criterion for minimising type I errors while maintaining sensitivity in whole-brain analyses.[38]

Statistical analyses of demographic and clinical data were performed using SPSS version 29 for macOS (http://www-01.ibm.com/software/uk/analytics/spss/). To examine the relationship between task-related neural activity and clinical measures, beta values were extracted from significant clusters identified in the whole-brain analysis using MarsBaR toolbox (run on macOS; https://marsbar-toolbox.github.io/). Associations between neural

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activity and DERS, ERS and BDI-II scores were assessed using Pearson's correlation. If normality assumptions were violated, Spearman's rank correlation was applied. Given the high comorbidity between depression and NSSI,[5] BDI-II scores were included to explore their potential associations with neural activity and to assess whether the observed neural alterations were specifically related to emotion dysregulation rather than depressive symptoms. To correct for multiple comparisons, the Bonferroni method was applied, setting the significance threshold at $P < 0.017$ (0.05/3).

Results

Demographic and clinical characteristics

There were no significant group differences in age, gender, handedness, education level or years of education (all $P > 0.05$) after participant exclusions. As predicted, individuals with NSSI reported greater emotion dysregulation ($t(52) = 6.71$, $P < 0.001$), heightened emotion reactivity ($t(52) = 3.82$, $P < 0.001$) and more severe depressive symptoms ($t(43.53) = 7.09$, $P < 0.001$) compared with controls. Within the NSSI group, participants reported an average NSSI versatility (i.e. number of NSSI methods used) of 5.21 (s.d. = 2.57). The mean age at NSSI onset was 13.21 years (s.d. = 3.68). Demographic and clinical characteristics of the participants are shown in Table 1.

Table 1 Demographic and clinical characteristics of participants ($N = 54$)

Characteristics	NSSI ($n = 29$)	Control ($n = 25$)	t/χ^2	P
Age, years	21.86 ± 2.46	22.68 ± 2.43	−1.23	0.226
Gender, female/male	25 / 4	21 / 4	0.05	0.820
Handedness, right/left	27 / 2	24 / 1	0.22	0.643
Level of education			3.54	0.315
High school or below	2 (7%)			
Undergraduate	20 (69%)	20 (80%)		
College or university graduate	7 (24%)	4 (16%)		
Graduate or above		1 (4%)		
Years of education	14.07 ± 1.85	15.04 ± 1.72	−1.99	0.052
DERS score[a]	96.72 ± 19.43	64.12 ± 15.69	6.71	<0.001
ERS score	32.52 ± 15.85	17.36 ± 12.82	3.82	<0.001
BDI-II score	22.28 ± 10.66	6.16 ± 5.59	7.09	<0.001
NSSI versatility	5.21 ± 2.57			
NSSI onset age	13.21 ± 3.68			
Comorbid psychiatric diagnosis	20 (69%)			

NSSI, non-suicidal self-injury; DERS, Difficulties in Emotion Regulation Scale; ERS, Emotion Reactivity Scale; BDI-II, Beck Depression Inventory-II.
a. Owing to a negative factor loading in the Korean version of DERS, item 17 was excluded from scoring.

Interscanner ratings

Across both groups, participants reported the highest mean valence during the positive emotion reactivity conditions (mean 3.90; s.d. = 0.69), followed by the positive emotion regulation (mean 3.25; s.d. = 0.41), baseline (attend-neutral) (mean 3.15; s.d. = 0.34), negative emotion regulation (mean 2.44; s.d. = 0.54) and negative emotion reactivity (mean 1.67; s.d. = 0.48) conditions. These results show that emotional images prompted the intended emotional responses and that participants properly understood the instructions. No significant differences were observed between groups across any of the conditions.

fMRI task-related activations

Emotion reactivity condition: attend-emotion > attend-neutral

In the whole-brain analysis, individuals with NSSI showed greater activation in the right superior temporal gyrus (STG), right parahippocampal gyrus (PHG) and supramarginal gyrus (SMG)

Table 2 Regional activity differences for emotion reactivity and regulation between NSSI and control groups

Contrast	Area	MNI coordinates x	y	z	K_E	Peak Z
Negative emotion reactivity						
NSSI > CONT	Right superior temporal gyrus	60	−30	10	93	3.95
		50	−32	4		3.78
		60	−38	4		3.54
	Right parahippocampal gyrus	34	−38	−6	41	4.07
	Right supramarginal gyrus	40	−34	26	24	3.78
NSSI < CONT	None					
Positive emotion reactivity						
NSSI > CONT	Right middle temporal gyrus	60	−40	4	201	4.30
		58	−26	8		4.21
		64	−32	10		4.07
	Left superior temporal gyrus	−38	−34	16	61	4.13
		−42	−40	20		3.42
		−50	−18	6	29	3.93
NSSI < CONT	None					
Negative emotion regulation						
NSSI > CONT	None					
NSSI < CONT	Left supplementary motor area	−4	20	44	107	4.06
		6	22	48		3.42
	Right superior temporal gyrus	54	−36	6	71	3.60
		50	−40	−2		3.45
	Right putamen	26	24	−2	30	3.63
	Left inferior frontal gyrus	−44	30	12	24	3.92
	Right thalamus	20	−10	0	21	4.10
Positive emotion regulation						
NSSI > CONT	None					
NSSI < CONT	Left ventral striatum	−8	16	−2	32	3.70
		−14	20	−6		3.64

NSSI, non-suicidal self-injury; CONT, control; MNI, Montreal Neurological Institute.

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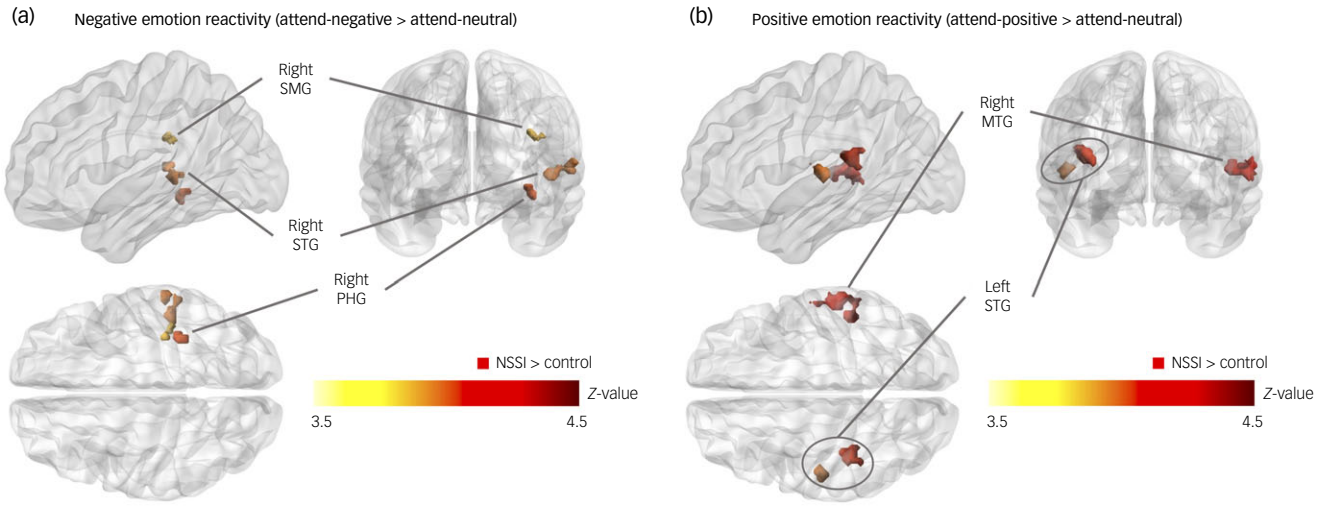

Fig. 1 Brain regions with significant hyperactivation in the non-suicidal self-injury (NSSI) group compared with controls during attention to emotions elicited by stimuli. (a) Individuals with NSSI showed increased neural responses in the right superior temporal gyrus (STG), parahippocampal gyrus (PHG) and supramarginal gyrus (SMG) during negative emotion reactivity (attend-negative > attend-neutral). (b) In the NSSI group, activation patterns in the right middle temporal gyrus (MTG) and the left STG were heightened during positive emotion reactivity (attend-positive > attend-neutral). All results are displayed at a voxel-level threshold of $P < 0.001$ with a cluster extent of $k = 20$.

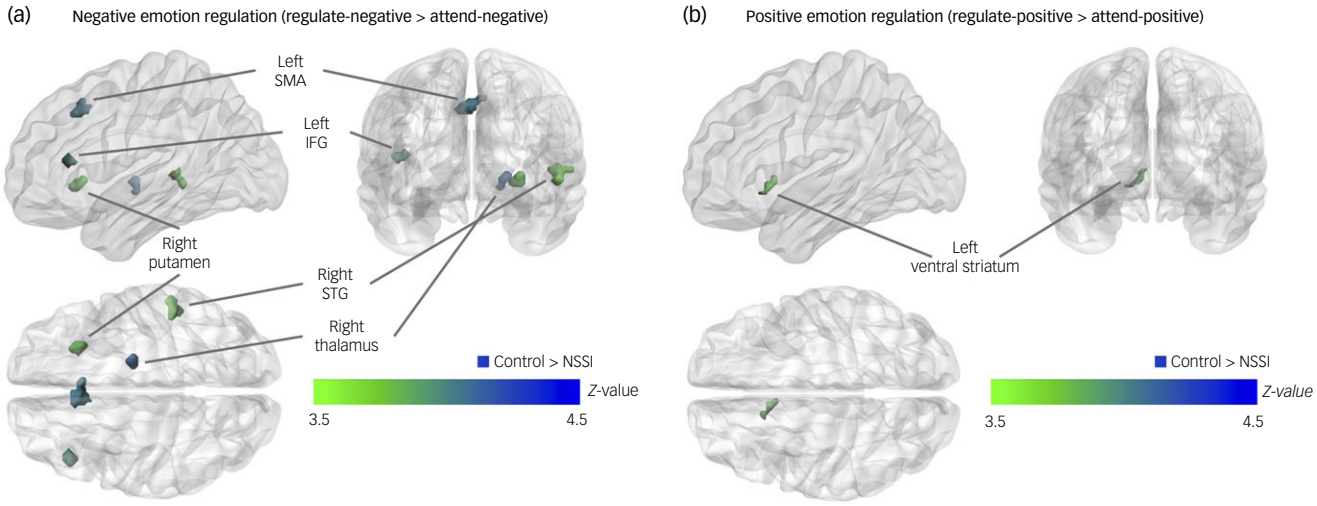

Fig. 2 Brain regions with significant hypoactivation in the non-suicidal self-injury (NSSI) group compared with controls during emotion regulation elicited by stimuli. (a) Individuals with NSSI exhibited decreased activation in the left supplementary motor area (SMA) and inferior frontal gyrus (IFG) and in the right superior temporal gyrus (STG), putamen and thalamus during negative emotion regulation (regulate-negative > attend-negative). (b) In the NSSI group, neural responses during positive emotion regulation (regulate-positive > attend-positive) in the left ventral striatum were decreased compared with controls. All results are displayed at a voxel-level threshold of $P < 0.001$ with a cluster extent of $k = 20$.

during negative emotion reactivity (attend-negative > attend-neutral) than controls. The NSSI group also exhibited increased activation of the right middle temporal gyrus (MTG) and left STG compared with the control group during positive emotion reactivity (attend-positive > attend-neutral). No significant differences were observed between individuals with NSSI and controls during the attend-neutral condition. Results are presented in Table 2 and Fig. 1.

Emotion regulation condition: regulate-emotion > attend-emotion

During negative emotion regulation (regulate-negative > attend-negative), control individuals showed enhanced activation of the left SMA, left inferior frontal gyrus (IFG), right STG, right putamen and right thalamus compared with individuals with NSSI. The control group also showed greater activation of the left ventral striatum than the NSSI group during positive emotion regulation (regulate-positive > attend-positive). Results are shown in Table 2 and Fig. 2.

Relationships between clinical characteristics and neural activations

In individuals with NSSI, increased STG activation during negative emotion reactivity was significantly correlated with higher DERS scores ($r = 0.51$, $P = 0.005$; Fig. 3). During negative emotion regulation, the decreased left SMA activation in the NSSI group showed a trend-level correlation with higher DERS scores

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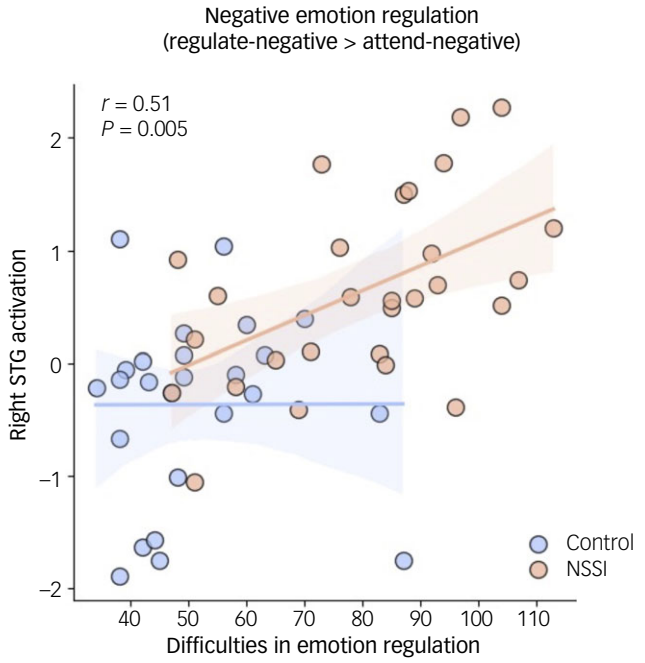

Fig. 3 Scatterplot depicting correlations between difficulties in emotion regulation and right superior temporal gyrus (STG) activation in the non-suicidal self-injury (NSSI) and control groups. A significant positive correlation was found in the NSSI group.

Table 3 Results for post-scan ratings: ratings for emotional valence, arousal and intensity of discrete emotion types

	NSSI ($n = 29$)	Control ($n = 25$)	t	P
Ratings of negative pictures				
Arousal	6.05 ± 1.40	6.25 ± 1.20	−0.56	0.580
Valence	2.58 ± 0.92	2.32 ± 0.60	1.21	0.232
Sad	5.86 ± 1.57	6.04 ± 1.76	−0.39	0.699
Happy	1.26 ± .028	1.12 ± 0.25	1.98	0.053
Fear	6.40 ± 1.57	6.07 ± 2.17	0.64	0.526
Disgust	4.25 ± 1.58	4.34 ± 1.86	−0.19	0.853
Anger	4.34 ± 1.61	4.57 ± 2.00	−0.47	0.639
Ratings of positive pictures				
Arousal	4.77 ± 1.36	5.34 ± 1.50	−1.46	0.152
Valence	6.11 ± 0.97	6.71 ± 0.77	−2.55	**0.014**
Sad	1.62 ± 0.71	1.31 ± 0.42	2.00	0.051
Happy	5.91 ± 1.82	6.47 ± 1.56	−1.21	0.232
Fear	2.99 ± 1.08	2.79 ± 1.05	0.68	0.500
Disgust	1.51 ± 0.67	1.16 ± 0.26	2.60	**0.013**
Anger	1.29 ± 0.58	1.09 ± 0.22	1.67	0.104
Ratings of neutral pictures				
Arousal	2.35 ± 1.02	2.05 ± 1.00	1.10	0.276
Valence	4.92 ± 0.63	5.08 ± 0.34	−1.19	0.241
Sad	2.05 ± 0.82	1.72 ± 0.93	1.35	0.182
Happy	2.06 ± 1.00	2.16 ± 1.25	−0.35	0.729
Fear	1.85 ± 0.80	1.38 ± 0.67	2.33	**0.024**
Disgust	1.52 ± 0.67	1.34 ± 0.62	1.04	0.304
Anger	1.39 ± 0.58	1.37 ± 0.64	0.024	0.877

NSSI, non-suicidal self-injury.
Bold values indicate statistical significance at $P < 0.05$.

($r = -0.44$, $P = 0.018$), which did not survive the Bonferroni-corrected threshold of $P < 0.017$. No significant associations were observed between BDI-II scores and neural activity in any brain region across both groups.

Post-scan ratings

Post-scan ratings revealed that individuals with NSSI perceived lower valence for positive images than the control group ($t(51.67) = -2.55$, $P = 0.014$). Specifically, the NSSI group reported significantly higher levels of disgust for positive images ($t(37.31) = 2.60$, $P = 0.013$) and significantly higher levels of fear for neutral images ($t(52) = 2.33$, $P = 0.024$). There was no significant difference in appraisals of negative images between groups (Table 3).

Discussion

The results of this fMRI study indicate that unmedicated individuals with NSSI exhibit distinct neural mechanisms underlying emotion reactivity and regulation, as well as differential processing of negative and positive emotions, compared with control individuals. Regarding emotion reactivity, individuals with NSSI showed hyperactivation in the STG, PHG and SMG in response to negative emotions, whereas the MTG and STG showed hyperactivation in response to positive emotions. By contrast, during emotion regulation, individuals with NSSI showed predominant hypoactivation in a wide range of cortical regions when regulating negative emotions, whereas hypoactivation in the ventral striatum was observed during positive emotion regulation. Furthermore, these neural patterns were correlated with the severity of emotion dysregulation. These findings highlight distinctive neural patterns of heightened emotion reactivity and insufficient emotion regulation in individuals with NSSI compared to controls. Thus, this study demonstrates dysfunction in reactivity to and regulation of negative emotions in individuals with NSSI and also provides novel insights into the altered processing of positive emotions using neuroimaging evidence.

Negative emotion reactivity

Individuals with NSSI exhibited hyperactivation of the right PHG in response to negative emotions. The PHG also showed hyperactivation during viewing of emotional images across a broad spectrum of psychiatric disorders.[39] Given that the PHG has a crucial role in facilitating contextual association and maintenance and retrieval of emotional memory,[40] this finding suggests a neural tendency of individuals with NSSI to amplify internal emotional states by integrating augmented contextual cues associated with negative emotions and elaborating on these experiences. Similarly, increased activation of the right SMG aligns with a previous finding linking this region to heightened negative emotion reactivity in individuals with NSSI.[16] The right SMG is involved in distinguishing external emotional stimuli from internal affective states, with greater activation observed when self–other distinction is particularly required in the context of shared negative emotion.[41] This suggests that individuals with NSSI may struggle to regulate the boundaries between negative external stimuli and their emotional experiences.

In addition, hyperactivation of the STG was observed during negative emotion reactivity in individuals with NSSI. The role of the STG in emotion recognition and attention to salient emotional information[42] is crucial to generating one's emotional state. Therefore, this STG hyperactivation in the NSSI group indicates not only increased attention to negative stimuli but also overexpression of negative emotions in individuals with NSSI. Notably, the increased STG activation observed during attention to negative emotions was correlated with higher levels of emotion dysregulation. This correlation implies that increased neural reactivity to negative emotions may impede an individual's ability to regulate emotions. Taken together, these findings suggest that heightened emotion reactivity to negative emotions in individuals with NSSI is

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due to dysfunctions in both automatic attention to negative emotional information and its subsequent semantic elaboration, exacerbating emotion dysregulation in individuals with NSSI.

Positive emotion reactivity

Compared with controls, individuals with NSSI showed increased activation in the MTG and STG while viewing positive pictures. Both of these regions have been implicated in explicit attention to emotional information,[43] implying increased sensitivity to emotional stimuli, consistent with recent meta-analytic findings in individuals with suicidality.[44] In particular, the MTG is involved in cognitive processing of emotional information,[45] and the right MTG is associated with response inhibition.[46] This may reflect a tendency among individuals with NSSI to inhibit responses to positive stimuli, even when participants were asked to merely attend to the emotional stimuli. In fact, the post-scan ratings showed that individuals with NSSI perceived less positive valence for positive images and reported significantly more disgust, indicating a disturbance in the automatic representation of positive emotions. This is consistent with previous research demonstrating that individuals with NSSI experience shorter durations of positive emotions than controls,[47] suggesting disengagement from positive emotional experiences. A longitudinal study of young people with NSSI also found that reduced positive emotional experiences were associated with greater engagement in self-harm.[48] These findings highlight a potential need for interventions for individuals with NSSI that promote acceptance of and engagement with positive emotions, as well as management of negative emotions. Future research is warranted to elucidate the propensity to withdraw from positive emotional experiences in individuals with NSSI.

Negative emotion regulation

Individuals with NSSI showed hypoactivation in the SMA and STG during negative emotion regulation compared with controls. This is consistent with previous findings in clinical populations including individuals with anxiety disorders[49] and depression.[50] The SMA and STG have key roles in cognitive emotion regulation;[23,40] therefore, attenuated activation in these regions may reflect reduced ability to downregulate unpleasant emotions in individuals with NSSI.

In addition, individuals with NSSI showed decreased recruitment in several regions, including the IFG, putamen and thalamus, during negative emotion regulation compared with controls. These regions have been implicated in cognitive emotion regulation,[23] and their hypoactivation has been consistently identified in meta-analyses of patients with bipolar disorder[51] and attention-deficit hyperactivity disorder,[52] both of which are characterised by difficulties in managing responses to emotional stimuli. In addition, decreased putamen activation during cognitive reappraisal has been reported in a meta-analysis of mood and anxiety disorders.[53] Therefore, the hypoactivation in emotion-regulatory regions in the NSSI group during negative emotion regulation may represent a transdiagnostic feature across different clinical populations with emotion regulation difficulties.

Notably, individuals with NSSI exhibited STG hyperactivation during both negative and positive emotion reactivity, alongside hypoactivation during negative emotion regulation. Previous studies have identified the STG as a key component of brain networks involved in regulating both negative and positive emotions.[23,54] Reduced right STG activation in individuals with NSSI suggests diminished engagement in cognitive processes essential for modulating emotional experiences. This distinct pattern of STG – hyperactivation during emotion reactivity and hypoactivation during emotion regulation – may indicate disruptions in emotional processing in individuals with NSSI, characterised by heightened emotion reactivity and subsequent difficulties in regulating intensified emotional responses.

Positive emotion regulation

Individuals with NSSI showed hypoactivation in the left ventral striatum during positive emotion regulation compared with controls. The ventral striatum is critical for emotion- and value-based decision-making.[55,56] Positive emotion regulation is sometimes used strategically when it is beneficial to disengage oneself from an elated mood, and this involves complex learning to modify the initial perceived value of emotional cues. Indeed, high levels of unregulated positive emotion have been associated with maladaptive coping strategies, such as NSSI.[57] Therefore, hypoactivation in these regions may indicate a neurological vulnerability in individuals with NSSI with respect to their ability to reappraise emotional values. Future work is needed to investigate whether the deficits in positive emotion processing in individuals with NSSI are related to difficulties in coping with intense positive emotions.

Our findings suggest that neural alterations in individuals with NSSI are linked to emotion dysregulation; however, this relationship was not consistent across all identified brain regions. Although certain areas, including the right STG and left SMA, showed associations with DERS scores, many altered activation patterns were not correlated with clinical measures, indicating that additional factors may contribute to neural differences in individuals with NSSI. Several explanations may account for this pattern. First, widely used self-report measures, such as the DERS and ERS, primarily assess difficulties in processing negative emotions, which may explain the lack of associations between these measures and neural responses during the processing of positive emotions. Second, emotion regulation is a complex process that involves distributed neural networks and individual variability, which may not be fully captured by region-specific activation analyses alone. Use of multimodal approaches that integrate behavioural, physiological and neuroimaging data may offer deeper insights into the relationship between neural activation and emotion dysregulation in individuals with NSSI.

Limitations

Several limitations should be noted. First, the cross-sectional nature of this study limited our ability to establish a causal relationship between engagement in NSSI and emotion dysregulation. Although our findings suggest that neural alterations are present in individuals with NSSI, it remains unclear whether these differences pre-date the onset of NSSI or develop as a consequence of prolonged engagement in self-injury. Even though no significant correlations were observed between age at onset of NSSI and neural activation patterns in our additional analysis, we could not rule out the possibility that longer NSSI duration contributes to neural alterations. To address this, future research should employ longitudinal designs to track the neurodevelopmental trajectory of NSSI and disentangle the long-term effects of persistent engagement in self-injury from pre-existing neurobiological vulnerabilities. Second, although NSSI characteristics and associated neural correlates may vary by gender, the small number of male participants in our sample limited the feasibility of gender-specific analyses. Incorporating data on sexual and gender identity, beyond gender assigned at birth, is also warranted. Third, this study categorised individuals with NSSI on the basis of diagnostic criteria to ensure clinically meaningful group distinctions. However, the lack of standardised quantitative measures for NSSI severity remains a limitation. Further research is necessary to create improved severity indices that more accurately reflect the

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complexity of NSSI and its neural correlates. Last, it is important to interpret these findings with caution because of the modest sample size. As Kaess et al[19] noted, the variability in characteristics of individuals with NSSI and associated psychiatric conditions presents a significant challenge in neurobiological research on NSSI. Furthermore, the potential long-term effects of prior pharmacological or psychotherapeutic interventions cannot be fully ruled out. Although participants were excluded if they had received treatment within the past month, certain neurobiological and psychological changes from previous interventions may have persisted beyond this exclusion window, potentially influencing the results. To improve the generalisability of the results of future studies, researchers should aim to recruit more extensive and diverse samples, including appropriate clinical control groups.

Despite these limitations, this study had several strengths. The careful recruitment of unmedicated participants minimised potential confounding effects of medication on neural differences between groups. In addition, the integration of neurobehavioral and self-report measures enhanced the study's validity, enabling us to offer novel insights into emotional processing in individuals with NSSI.

Clinical implications

This study has provided clear evidence of the neurobiological nature of alterations in emotion reactivity and regulation in individuals who engage in NSSI, including neural hyperactivation during emotion reactivity and hypoactivation during emotion regulation, changes that extended beyond negative emotion processing to include difficulties in managing positive emotions. Therefore, the results underscore the need for more comprehensive therapeutic approaches that promote adaptive processing of both negative and positive emotions in individuals with NSSI. Future research should aim to deepen our understanding of the neurophysiological basis of emotional processing in individuals with NSSI by examining the long-term effects of these interventions and the neural changes associated with successful treatment outcomes.

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First received 18 Sep 2024, final revision 13 May 2025, accepted 3 Jun 2025

Data availability

The data that support the findings of this study are not publicly available owing to ethical considerations: participants did not provide written consent for their data to be publicly shared. Instead, de-identified participant data will be made available upon reasonable request by the corresponding author.

Acknowledgement

We thank all individuals who participated in this study.

Author contributions

G.K. and J.-W.H. conceptualised the study and contributed to formal analysis and methodology; G.K., H.S. and J.-W.H. conducted investigations; G.K. performed visualisations; and J.-W.H. supervised the study and acquired funding. G.K. and J.-W.H. wrote the original draft of the manuscript, and G.K., H.S. and J.-W.H. contributed to writing, reviewing and editing.

Funding

This research was supported by a grant of the Mental Health related Social Problem Solving Project, funded by the Ministry of Health & Welfare, Republic of Korea (grant number: RS-2024-00406503), and a National Research Foundation of Korea grant funded by the Ministry of Science, ICT & Future Planning, Republic of Korea (NRF-2022R1A2C2011467).

Declaration of interest

None.

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