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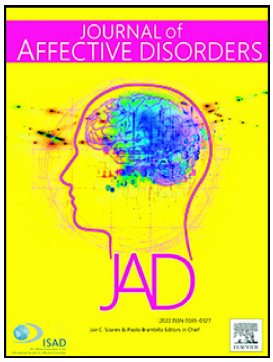

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Neurocognitive functioning in adolescents with non-suicidal self-injury

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Abstract

Background: Non-suicidal self-injury (NSSI) is a highly prevalent transdiagnostic psychiatric symptom in adolescents. Research in adults has begun to investigate neurocognitive processes associated with NSSI as potential underlying phenotypes. However, research on neurocognitive function in adolescent patients with NSSI is scarce.

Methods: In this study, we examined neurocognitive functioning in the domains of processing speed, attention, learning, working memory, and executive function in a relatively large sample of n=240 adolescent patients engaging in NSSI and n=49 healthy controls. Further, associations between neurocognitive performance and clinical characteristics in the patient group were examined.

Results: While conventional regression analyses showed somewhat weaker neurocognition in the NSSI group in several domains, propensity score matching for IQ showed little evidence that patients engaging in NSSI showed worse neurocognition when general intelligence was considered. Further, a random forest machine learning algorithm was not able to classify NSSI vs. control groups based on neurocognitive features. Within the patient group, linear regression and latent class analyses yielded little evidence that neurocognitive performance was related with clinical characteristics or phenotypes.

Limitations: As the study did not include a clinical control group, findings might not be specific to NSSI.

Conclusions: Our findings challenge the importance of specific neurocognitive measures related to the presence or severity of NSSI in adolescents. Future studies should consider general intelligence as an important confounding factor and should focus on domains of affective cognition. Finally, longitudinal studies are needed to determine whether low neurocognitive performance serves to inform prognosis of NSSI or psychopathology in general.

Keywords: Non-suicidal self-injury, adolescent, neurocognition, neuropsychology, intelligence

Introduction

Non-suicidal self-injury (NSSI) is defined as self-injurious behavior in the absence of suicidal intent. The rising clinical and scientific interest in the phenomenon of NSSI has been reflected by the introduction of the NSSI disorder to the 5th version of the Statistical and Diagnostic Manual of Mental Disorders (DSM-5) under section III, as a research diagnosis requiring further study (American Psychiatric Association, 2013). Criterion A of NSSI disorder is met when a person engages in self-injury without suicidal intent five or more days within the past year. Its prevalence has been found to be 4% in adolescent non-clinical samples (Plener et al., 2016) and around 50% among adolescent in-patient samples (Glenn & Klonsky, 2013; Groschwitz et al., 2015). Previous NSSI history was shown to predict future NSSI and suicide attempts (Koenig et al., 2017) and Borderline Personality Disorder (BPD) development (Ghinea et al., 2019). Recent research suggests that NSSI can be seen as a transdiagnostic risk marker of psychopathology in general (Ghinea et al., 2020). Specifically investigating the underlying phenotypes and mechanisms of NSSI may help to define risk groups, who in the future may receive individualized therapy adapted to their corresponding risk profile.

Neurocognition is one of the factors potentially contributing to an enhanced risk for self-harming behavior. As such, youth engaging in these behaviors show difficulties regulating their emotions and a lack of impulse control (Kaess et al., 2013). While individuals engaging in self-injury often reported greater impulsiveness in self-ratings, this association was rarely found in performance-based measures (Glenn & Klonsky, 2010; Janis & Nock, 2009; McCloskey et al., 2012). While a number of studies have investigated cognitive processes associated with NSSI, fewer have used behavioral neurocognitive measures apart from self-ratings (Cha et al., 2019). Existing studies targeting neurocognition and specifically "cool" executive functions in *adults* yielded little evidence for differences between individuals engaging in NSSI and healthy controls in inhibitory control/inhibition (Glenn & Klonsky, 2010; Hamza & Heffer, 2015; Liu et al., 2017; McCloskey et al., 2012), cognitive interference (Dahlgren et al., 2018), error monitoring (Vega et al., 2015) and decision making (Schatten et al., 2015). Interestingly, on tasks inducing forms of *negative affect* relevant to NSSI, deficits in inhibitory control have been found more consistently (Allen, Fox, et al., 2019; Allen & Hooley, 2015). This

might imply that the emotional component in e.g., executive function tasks is responsible for deficits rather than cognitive performance per se. In fact, there is first evidence for the importance of 'affective cognition' in the context of NSSI (Allen & Hooley, 2019; Burke et al., 2021).

Considerable development of neurocognition and in particular executive functioning alongside the remarkable neural changes during adolescence (Blakemore, 2008) increase vulnerability for mental health difficulties in young people (Heim & Binder, 2012; Leichsenring et al., 2011; Spatz Widom et al., 2007). Therefore, studying neurocognitive concomitants potentially intertwined with the emergence of NSSI at an early age is of particular importance. Regarding neurocognition in *adolescent* NSSI, research is scarce. A recent meta-analysis on inhibition and impulsive decision making in young patients (< 30 years old) with self-harming and suicidal behavior reported overall deficits in patients (McHugh et al., 2019). Out of eight included studies investigating task-based neurocognitive differences between young patients engaging in NSSI and controls, however, only one study showed evidence of higher impulsivity in adolescents with low-severity NSSI than healthy controls (Fikke et al., 2011). Due to the limited literature in adolescents and mixed findings regarding neurocognitive deficits in individuals engaging in NSSI, the present investigation aimed to examine whether neurocognition was related to NSSI in a fairly large sample of adolescent patients and controls.

The first aim of the current study was to explore alterations in neurocognitive performance including processing speed, attention, memory, and executive functions in a large sample of adolescents engaging in NSSI, compared to age-matched healthy controls. The second aim was to investigate whether neurocognitive performance is associated dimensionally with the severity of psychopathology in adolescents with NSSI. It is of vital importance to determine potential risk factors related to clinical outcome in adolescents engaging in NSSI. Therefore, investigating neurocognitive functioning associated with NSSI in adolescence may help to clarify potential underlying neurocognitive phenotypes associated with the risk for self-harming behavior.

Methods and Materials

Participants and Procedure

Partients with NSSI disorder according to DSM-5 were consecutively recruited from the outpatient clinic for risk-taking and self-harming behavior (AtR!Sk; *Ambulanz für Risikoverhaltensweisen und Selbstschädigung* (Kaess et al., 2017) at the University of Heidelberg. The study was approved by the ethical committee of the University of Heidelberg (study-ID S-449/2013; study-ID S-514/2015). 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. Adolescents and their caregivers provided written informed consent before inclusion in the study. General inclusion criteria were presentation in our outpatient clinic, written informed consent of adolescents and their caregivers, age between 12 and 17 years and fluent German language. General exclusion criteria for study participation were acute psychosis, pregnancy, and neurological, endocrinological, or cardiovascular primary diseases. Healthy controls were recruited via advertisement and matched to the patient sample according to age. Exclusion criteria for controls were the same as for the patient group. Further exclusion criteria for the control group only were lifetime self-harming behavior, lifetime psychological or psychiatric treatment, or any current psychiatric disorder. All participants received an allowance of 40€ for study participation.

Clinical Assessment

All patients underwent comprehensive clinical assessment. The presence of NSSI, suicidal thoughts and behavior were assessed using the German version of the Self-Injurious Thoughts and Behaviors interview (SITBI-G) (Fischer et al., 2014; Nock et al., 2007). Healthy controls were first interviewed via telephone using screening questions from the SITBI-G to ensure that they had no history of NSSI or suicidal behavior. They then underwent a shortened clinical interview to ascertain they did not meet the criteria for any current mental disorder and were not under psychological or pharmacological treatment. Those participants meeting the criteria for any psychiatric disorder in an additional diagnostic interview were compensated for their participation in the diagnostic assessment and excluded

from further study appointments. Other clinical measures obtained but not used in the statistical analyses are described in the Supplementary material.

Neurocognitive Assessment

The intelligence quotient (IQ) was assessed using the Hamburg Wechsler Intelligence Scale for Children-IV (HAWIK-IV). The Cognitive Basic Assessment (COGBAT), a validated computerized neurocognitive test battery, was used to measure the cognitive domains of processing speed, attention, memory, and executive functions in adolescents and adults. It has been validated in n = 269 young adolescents aged 12 – 15 years (Mürner-Lavanchy et al., under review).

Processing Speed

Processing speed was assessed using the Trail Making Test Langensteinbacher Version TMT-L Part A. Pseudorandomized numbers (1 to 25) were to be clicked on the screen as fast as possible in ascending order (main variable: processing time in seconds, lower values indicative of faster processing speed). As a further measure, the WISC processing speed index, consisting of the subtests *coding* and *symbol search*, was evaluated.

Attention

Simple attention was measured with the Perceptual and Attentional Functions - Alertness (WAF-A) test, which involved responding as quickly as possible to a simple visual stimulus (main variable: mean reaction time). Divided attention was assessed using the Perceptual and Attentional Functions (WAF-G) test, in which visual and auditory stimuli were presented simultaneously and changed in brightness (visual) or volume (auditory). Participants were asked to respond as soon as the stimuli changed in the same way twice in a row (main variable: mean reaction time and omissions).

Memory

Visual learning was assessed using the Figural Memory Test FGT. Participants were presented with figures they were asked to remember and reproduce directly. The first variable of interest was the sum of all correctly reproduced figures (learning sum). After five minutes, the figures were to be recalled and reproduced freely without being presented again (main variable: number of correctly recalled

figures: immediate recall). After 30 minutes, free figure reproduction was repeated (main variable: delayed recall). This was followed by a phase of recognition via a forced-choice task (main variable: false-positive recognition errors). Verbal working memory was assessed using the N-Back (2-back) Verbal Test NBV (main variable: number of correct answers). As a further measure of verbal working memory, the WISC working memory index, consisting of the subtests *digit span*, *letter-number-sequencing*, and *arithmetic* was evaluated.

Executive Functions

Cognitive flexibility was assessed using the Trail Making Test-Langensteinbacher Version TMT-L Part B. The subject was asked to alternately connect circles with numbers from 1 to 13 and letters from A to L in an ascending order. The variable of interest was the processing time from start to finish. Response inhibition was measured via the Go/Nogo paradigm INHIB. Participants were asked to respond to one kind of stimuli (triangle) but not to respond to another kind of stimuli (circle) (main variable: number of commission errors). Planning ability was assessed with the Tower of London-Freiburg version TOL-F. The difficulty level was ascending (from three- to six-item tasks) and ended when the maximum processing time (60s) was exceeded for three tasks in a row (main variable: number of tasks solved in the given moves).

Statistical Analyses

Statistical analyses were performed using Stata (Version 16; StataCorp LP, College Station, TX, USA). Overall, interpretation of the findings was based on overall patterns and magnitude of differences rather than individual *p*-values. Patients' and participants' demographic characteristics were analyzed using descriptive statistics. Sex, smoking, and drug use were not distributed evenly across groups and were regarded as covariates in further analyses. For the first aim, neurocognitive performance was compared between the patient group and healthy control group using regression models. Model M1 tested for a group difference without covariates and Model M2 for a group difference adjusted for sex. To determine the best model (M1 or M2), Bayes Factors (BF) of the individual models were compared, and the model with the highest BF which was significantly different from M1 as determined by the Likelihood Ration Test ($p < .05$) was defined to be the best fit. Additional sensitivity

analyses included smoking, drug use and IQ as control variables and compared whether these models resulted in a better model fit than the unadjusted models.

In order to further examine group differences in neurocognitive domains independent from the confounding factor of IQ, i.e. to avoid collinearity by controlling for IQ within the models, we applied a matching procedure (Stuart, 2010). This statistical approach has been recommended for observational studies to reduce bias due to covariates (Rubin, 2007) and has been applied in other studies investigating group differences in the context of psychopathology (Hoffmann et al., 2016). Using 1:1 linear propensity score matching, for each control participant (n = 49) the closest neighbor from the NSSI group in terms of IQ, age, and sex, was identified (n = 49). Further abovementioned analyses of group differences were repeated with these two groups matching in IQ, age and sex.

Additionally, a machine-learning approach was used to analyze whether group differences in neurocognition were detectable on a purely data-driven basis. All neurocognitive variables were entered as features to a random forest algorithm. To test whether a classification into two groups (NSSI and controls) could be reached based on the neurocognitive data, the model was calculated based on a training set, consisting of 80% of the data (80% of data from the NSSI group and 80% of data from the control group). Multiple models were calculated by growing 1000 bootstrapped trees using the Gini impurity measure and different numbers out of 24 neurocognitive variables. The simplest model with perfect classification was applied to the remaining 20% of data for validation.

For the second aim, in patients only, associations between the main variables of neurocognitive performance and clinical characteristics, namely NSSI, suicidal thoughts, suicide attempts, number of BPD diagnostic criteria, depressive symptoms, patient's global functioning (GAF) and severity of psychiatric symptoms (CGI), were examined using regression. Again, a model comparison approach was chosen to compare between M1, an unadjusted model and M2 testing for associations adjusted for age. In these analyses, outliers at more than 3 standard deviations (SD) above the group mean were excluded.

To further examine potential associations between neurocognition and clinical characteristics, a latent class analysis (LCA) was computed. First, we applied LCA to find a potential underlying data structure, i.e., to test whether variables of neurocognition could be grouped to build meaningful classes.

Second, post-hoc analyses were conducted with resulting classes as independent variables to determine whether class-membership was associated with specific clinical outcomes. LCA models were estimated with an increasing number of classes, starting with one class. Models were evaluated based on the Bayesian Information Criterion (BIC), Akaike's Information Criterion (AIC) and entropy. A difference of more than 10 in BIC values between two models indicated support for the model with the lower value (Raftery, 1995). In addition to the fit indices discussed, entropy was evaluated; a measure of the degree to which the latent classes are distinguishable and the precision with which individuals can be placed into classes. It ranges from 0 to 1, with higher values indicating clearer class separation. A value of ≥.80 is recommended, when participants should be classified based on the "most likely class membership" for further analysis (Celeux & Soromenho, 1996). Finally, post-hoc analyses were conducted to determine the validity of the classes identified by the best fitting model. Therefore, participants were grouped according to their most likely latent class membership and compared regarding clinical variables.

Results

Participants

N = 240 adolescents engaging in NSSI and n = 49 healthy controls were recruited. Demographic and clinical characteristics for both groups are detailed in **Table 1** (an overview of the comorbid diagnoses can be found in Supplementary Table 1). There was a greater proportion of females in the control than in the NSSI group (p = 0.015). Regarding school-type, n = 83 adolescents engaging in NSSI and n = 30 controls attended the *Gymnasium* (graduation qualifies for university entrance); n = 103 adolescents from the NSSI group and n = 15 controls attended or completed the *Realschule* (secondary school level certificate); and n = 34 from the NSSI group, and n = 3 controls attended or completed the *Hauptschule* (9 years of elementary school). Groups did not differ significantly regarding school-type (χ^2 = 15.08, p = .179).

In the NSSI group, the mean frequency of NSSI was 58.16 (SD = 72.15) during the last year and 5.57 (SD = 7.61) during the past month. The mean reported number of suicide attempts was 3.93 (SD = 33.04) across the lifetime and 1.25 (SD = 6.38) during the past year.

Neurocognition in NSSI and healthy controls

Details of group differences in neurocognition are depicted in **Table 2**. For the majority of the neurocognitive variables, M1 modelling the group difference without any covariates, fitted best. The NSSI group showed lower IQ than the control group and worse performance in processing speed, learning, working memory, inhibition (response variability), and planning ability. Processing speed (TMT A errors and WISC processing speed index) showed an additional effect of sex, with female participants and patients showing better performance. Effects were generally small (β ranged between 0.12 and 0.20). Neurocognitive performance of the control group was comparable with German normative scores established in a similar age group (Mürner-Lavanchy et al., under review). Neurocognitive variables in the domains of processing speed, alertness, inhibition and planning were correlated with age, with better performance associated with increasing age (see **Table 3**). There were no group-by-age interactions for any of the variables. Sensitivity analyses including smoking and drug use as covariates resulted in worse model fit compared to the unadjusted models. Sensitivity analyses

including IQ as a covariate (see **Table 2** for IQ-adjusted model estimates) resulted in better model fit compared to the unadjusted models. Accounting for IQ considerably decreased differences in neurocognitive functioning between NSSI and control groups, with weak evidence of worse performance in the NSSI group in WISC processing speed index (β = -0.13) and inhibition response variability (β = 0.11).

Group differences in neurocognition independent from general intelligence

After matching the NSSI and control groups based on IQ, age and sex, the NSSI group (n = 49) showed worse performance than the control group (n = 49) in delayed recall (more errors, M_{NSSI} = 1.73, SD = 1.87; $M_{control}$ = 0.69, SD = 1.14, p = .001, β = 0.32) and inhibition response variability, reflecting inattentiveness (M_{NSSI} = 0.12, SD = 0.08; $M_{control}$ = 0.09, SD = 0.05, p = .015, β = 0.24) (see **Table 4**). The effects were small and given the number of statistical comparisons, there was no strong evidence of group differences in neurocognition independent from IQ.

Data-driven machine learning approach of classification into patient and participant groups

With n = 5 out of n = 24 neurocognitive features, a perfect classification was reached by the random forest algorithm in the training set (100% sensitivity and 100% specificity, AUC = 1). However, applied to the test data, this classification yielded poor sensitivity (0%; specificity: 100%; AUC = 0.5). This means the model preforms well when applied to data that the model is based on, but fails to predict unseen data.

Associations between clinical patient characteristics and neurocognition

Associations between the neurocognitive variables of interest and psychopathology are detailed in **Table 5**. Overall, there was weak evidence that neurocognitive performance was correlated with the severity of psychopathology in adolescents engaging in NSSI. An unclear pattern of findings emerged: Frequency of NSSI during the lifetime was associated with better planning ability (β = 0.13). Further, the higher the frequency of NSSI during the past year, the better was divided attention (β = -0.14) verbal learning (β = 0.15) and planning (β = 0.17). Frequency of NSSI in the past month was associated with better verbal learning (β = 0.19) and flexibility (β = -0.14). The higher the num-

ber of suicide attempts during the lifetime, the worse was verbal learning (β = -0.16). Finally, higher depression severity correlated with better planning ability (β = 0.18). However, effects were relatively small and given the number of statistical comparisons, these results must be interpreted with caution.

Latent component analysis approach

Table 6 illustrates the fit indices for the latent class models. According to BIC values, the two-class solution showed better model fit compared to the one-class model. N = 176 (73.3%) patients were more likely to belong to class 1 and n = 64 (26.67%) patients were more likely to belong to class 2. Class 2 was characterized by worse neurocognitive performance on almost all tests (23 out of 24 variables). The two classes did not differ with regards to most of the clinical variables (clinical global impression, global functioning, NSSI behavior, BPD diagnosis and number of BPD criteria), except for frequency of NSSI during the past month, with Class 1 showing more phases of NSSI than Class 2 (M_{class1} (SD) = 6.39 (8.28), M_{class2} (SD) = 3.33 (4.31), p = .006).

Discussion

The aim of the present study was to explore alterations in neurocognitive performance in a large sample of adolescents engaging in NSSI compared to age-matched healthy controls with a variety of statistical approaches. We further examined whether neurocognitive performance was associated dimensionally with the severity of psychopathology in adolescents with NSSI.

The assessment of general intelligence in addition to specific neurocognitive domains allowed us to find pronounced group differences in IQ between adolescents engaging in NSSI and healthy controls. Given that lower intelligence confers vulnerability to psychopathology (Gale et al., 2010), this finding is not surprising. On the one hand, lower intelligence may lead to a decreased ability to solve life problems, lead to lower educational attainment, poor employment opportunities and financial difficulties, which in turn are risk factors for psychopathology (Chang et al., 2014). On the other hand, risk behavior and associated mental illness may lead to lower intelligence. Alternatively, there might be common causes of poor cognitive ability and psychopathology, such as severe childhood adversities (Gale et al., 2010). Finally, it is also thinkable, that mental illness temporarily hinders patients from

performing to their full potential. Up to now, there is very limited research on the association between general intelligence and non-suicidal self-injury in adolescents. It has recently been suggested that there might be opposite associations between intelligence and suicidal behavior vs. suicidal ideation: higher intelligence might be associated with suicidal ideation, but not behavior (i.e. NSSI) and vice-versa (Allen, Fox, et al., 2019). However, previous findings are mixed (Chang et al., 2014; Saffer & Klonsky, 2018).

The group difference in general intelligence represents a major confound when aiming to examine group differences in neurocognitive functioning. Therefore, we applied several statistical methods allowing us to test whether neurocognitive differences could be found independent from general intelligence. These additional analyses, however, provided little evidence of meaningful group differences in several domains of neurocognition. Executive functions, such as impulse control have been the most prominent neurocognitive measures investigated in the context of NSSI so far. While previous meta-analytic research showed higher impulsivity in adolescents engaging in self-harm (McHugh et al., 2019), the confounding factor of intelligence has rarely been considered and thus, deficits in neurocognition might have been driven by general IQ-differences between groups. Only one study investigated associations between impulsivity and NSSI independent from general intelligence (Fikke et al., 2011) and found evidence of higher impulsivity in adolescents with low-severity NSSI compared to high-severity NSSI and healthy controls. The authors interpreted that while adolescents with low-severity NSSI engaged in the behavior impulsively, the high-severity group may have engaged in a more deliberate way. Similar to previous literature in depression (Fieker et al., 2016; Moritz et al., 2017), the results of the present study do not support deficits in task-based inhibition in a sample of high-severity NSSI patients compared to controls.

Using dimensional analyses, our findings provided little evidence for a systematic relationship between neurocognitive functioning and NSSI severity in patients. Using LCA, we further examined whether 'neurocognitive subtypes' exist within the clinical sample. We found two classes of patients: those with higher performance in almost all neurocognitive measures and those with worse performance on the measures assessed. However, this difference in neurocognition was not meaningfully

related to clinical characteristics. Consequently, neurocognitive performance as measured in our study, does not provide useful insight into the mechanisms of NSSI in adolescents.

While we did not find evidence that our measures of "non-affective" neurocognition were related to NSSI, it is likely that measures of affective cognition are associated with emotion regulation in the context of NSSI (Allen, Bozzay, et al., 2019; Cha et al., 2019). In adult samples, individuals who self-injured exhibited poorer inhibitory control over negative images, but did not differ in response to neutral stimuli (Allen & Hooley, 2015). In the context of risky decision-making in response to critical feedback (eliciting negative affect), individuals with NSSI history were more likely to make impulsive choices during negative mood, but not necessarily in its absence (Allen, Fox, et al., 2019). Further, adults with NSSI history had worse so-called negative emotional action termination (NEAT) than healthy controls in an emotional Go/Nogo task (Allen & Hooley, 2019). Interestingly, NEAT also explained variance in the association between negative urgency (i.e. the self-reported tendency to act impulsively when distressed) and NSSI, suggesting that impulsive behavior in NSSI may involve specifically impaired inhibitory control over initiated negative emotional impulses. In an ecological momentary assessment study with young university students with a history of NSSI (ages 18-26), emotional response inhibition to self-harm images interacted with momentary negative affect to predict the strength of real-time NSSI urges, after adjusting for emotional response inhibition to neutral images (Burke et al., 2021). Consequently, so-called 'hot' executive functions (Kerr & Zelazo, 2004) are associated with NSSI rather than the purely cognitive aspects of 'cool' executive functions such as problem solving and planning (Allen, Bozzay, et al., 2019). From a neurobiological perspective, 'cool' executive functions are mainly associated with the dorsolateral prefrontal cortex (Zelazo & Müller, 2002), whereas 'hot' executive functions have primarily been associated with the orbitofrontal cortex (OFC), involved in the reappraisal of affective or motivational significance of stimuli (Rolls, 2004), and affective decision-making (Chavez-Arana et al., 2018). Interestingly, alterations in OFC activity and connectivity as well as network efficiency have been found in adolescents engaging in NSSI (Koenig et al., 2021; Poon et al., 2019), highlighting the role of the brain's reward circuitry in the context of NSSI. Up to now, few studies have investigated associations between measures of social or affective cognition and NSSI, and even less in adolescent samples.

One important limitation of our study is that we did not recruit a clinical control group. Consequently, our findings might not be specific to NSSI, but rather relevant to general psychopathology. We included patients with NSSI disorder according to the DSM-5 for the sake of comparability between studies. However, the DSM-5 cut-off defined in criterion A is arbitrary and is under dispute for its validity and clinical utility (Ammerman et al., 2019; Hooley et al., 2020; Muehlenkamp et al., 2017; Zetterqvist et al., 2020). Finally, due to the use of a standardized cognitive test battery with predefined tasks, we did not include affective neurocognitive measures, such as emotional inhibition or reward-based tasks and value-based decision making.

Conclusion

In conclusion, the results of the present study yielded no evidence of differences in neurocognition between adolescents engaging in NSSI and healthy controls. Measures of affective cognition might be more useful in this regard and should be the focus of future investigations in adolescent samples. Further, general intelligence should be considered a confounding factor in future studies.

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Conflict of interest

None.

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Data statement

Our research data is unavailable for access due to confidentiality.

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Table 1. Participant characteristics in adolescents engaging in NSSI and healthy controls

	NSSI n (%) / mean (SD)	**Control** n (%) / mean (SD)
N	240	49
Age	14.95 (1.46)	14.80 (1.31)
Female sex	197 (82.08)	47 (95.92)
Psychoactive medication	20 (8.33)	0 (0)
Smoking [a]	93 (38.75)	1 (2.04)
Alcohol consumption [a]	52 (21.67)	11 (22.45)
Drug consumption [b]	35 (14.58)	0 (0)
Number of BPD diagnostic criteria	3.05 (2.13)	0.6 (0.32)
BPD diagnosis	63 (26.58)	0 (0)
Acts of NSSI lifetime	132.67 (250.09)	-
Acts of NSSI past year	52.10 (70.56)	-
Acts of NSSI past month	4.99 (7.40)	-
Suicide attempts lifetime	3.93 (33.05)	
Suicide attempts past year	1.25 (6.98)	-
Suicide attempts past month	0.11 (0.45)	-
Depressive symptoms	27.76 (9.21)	6.77 (5.28)
Childhood abuse [c]	134 (54.11)	4 (8.16)
Clinical Global Impression Scale	4.82 (0.88)	-
Global Assessment of Functioning	51.60 (9.84)	-

Note. NSSI = non-suicidal self-injury; BPD = Borderline Personality Disorder; SD = standard deviation. [a] On three or more days in the last month. [b] On one or more days in the last month. [c] Childhood Experience of Care and Abuse (CECA) questionnaire.

Table 2. Group differences in neurocognition.

	NSSI M (SD)	**Control** M (SD)	*Unadjusted model* *β* (95% CI)	*p*	*IQ-adjusted model* *β* (95% CI)	*p*
General intelligence						
WISC IQ	100.70 (13.78)	106.24 (11.70)	-0.15 (-0.71, -0.10)	.009		
Processing speed						
TMT A time	19.00 (4.20)	18.75 (3.81)	-0.02 (-0.37, 0.24)	.687	-0.08 (-0.50, 0.09)	.136
TMT A errors [a]	0.08 (0.29)	0.04 (0.20)	0.02 (-0.26, 0.35)	.766	0.03 (-0.22, 0.40)	.579
WISC processing speed [b]	100.88 (14.16)	109.98 (13.68)	-0.20 (-0.83, -0.24)	< .001	-0.13 (-0.57, -0.13)	.002
Alertness						
WAFA Mean RT	236.66 (40.92)	233.57 (40.84)	0.03 (-0.22, 0.39)	.630	0.03 (-0.22, -0.39)	.593
WAFA Omissions	0.25 (0.82)	0.31 (0.82)	-0.02 (-0.38, 0.24)	.663	-0.02 (-0.37, 0.24)	.674
Divided attention						
WAFG Mean RT	557.06 (147.49)	531.96 (147.49)	0.06 (-0.15, 0.47)	.307	0.03 (-0.23, 0.38)	.631
WAFG Omissions	6.54 (4.34)	6.59 (5.18)	-0.00 (-0.32, 0.30)	.941	-0.07 (-0.47, 0.08)	.171
Learning						
FGT Learning Sum	30.72 (8.44)	34.38 (7.85)	-0.15 (-0.72, -0.11)	.008	-0.07 (-0.46, 0.07)	.141
FGT Immediate recall	7.24 (2.03)	8.06 (1.59)	-0.16 (-0.72, -0.11)	.008	-0.08 (-0.47, 0.07)	.138
FGT Immediate recall errors	1.30 (1.98)	0.69 (1.14)	0.12 (0.02, 0.63)	.038	0.09 (-0.05, 0.56)	.104
FGT Delayed recall	7.19 (2.04)	7.94 (1.76)	-0.14 (-0.67, -0.06)	.020	-0.06 (-0.43, 0.11)	.241
FGT Delayed recall errors	1.09 (1.69)	0.63 (1.25)	0.10 (-0.03, 0.58)	.076	0.06 (-0.13, 0.47)	.256
FGT Recognition	8.41 (1.00)	8.79 (0.46)	-0.15 (-0.70, -0.09)	.011	-0.10 (-0.57, 0.02)	.066
FGT Recognition errors	1.15 (1.52)	0.71 (1.15)	0.11 (-0.01, 0.60)	.057	0.03 (-0.18, 0.35)	.561
Working memory						
N-back	10.31 (3.11)	11.45 (3.00)	-0.14 (-0.67, -0.06)	.020	-0.07 (-0.47, 0.08)	.173
N-back errors	9.61 (11.95)	7.39 (12.83)	0.07 (-0.12, 0.49)	.242	0.01 (-0.26, 0.32)	.831
WISC working memory	99.61 (14.01)	105.33 (12.01)	-0.16 (-0.72, -0.11)	.008	-0.04 (-0.29, 0.10)	.324
Executive Functions						
Inhibition errors	9.85 (4.08)	9.22 (3.58)	0.06 (-0.15, 0.47)	.316	0.03 (-0.23, 0.38)	.639
Inhibition reaction time	0.28 (0.05)	0.27 (0.04)	0.09 (-0.07, 0.54)	.134	0.04 (-0.17, 0.43)	.401
Inhibition variability	0.12 (0.07)	0.09 (0.05)	0.16 (0.11, 0.72)	.008	0.11 (0.00, 0.59)	.049
TMT B Flexibility	29.05 (9.95)	26.21 (7.88)	0.11 (-0.01, 0.60)	.061	0.02 (-0.19, 0.31)	.643

TMT B Flexibility errors	0.99 (1.21)	0.67 (1.18)	0.10 (-0.04, 0.57)	.092	0.05 (-0.16, 0.44)	.349
TOL Planning ability	13.31 (3.24)	14.33 (2.84)	-0.12 (-0.62, -0.01)	.042	-0.06 (-0.44, 0.13)	.285

Note. Beta-coefficients for the main effect of group are reported for the best fitting model. RT = Reaction Time. [a] Best fitting model included main effect of sex β (95% CI) = 0.20 (0.23, 0.86), p <.001. [b] Best fitting model included main effect of sex beta β (95% CI) = -0.24 (-0.96, -0.35), p <.001

Table 3. Correlations between neurocognitive variables and age

	n	*r*	(95% CI)	*p*
WISC IQ	289	-0.11	(-0.22, 0.003)	.056
TMT A time	291	-0.26	(-0.36, -0.14)	< .001
TMT A errors	291	0.08	(-0.04, 0.19)	.194
WISC processing speed	290	-0.20	(-0.31, -0.09)	.001
WAFA Mean RT	291	-0.26	(-0.37, -0.15)	< .001
WAFA Omissions	291	-0.12	(-0.23, -0.001)	.047
WAFG Mean RT	290	-0.02	(-0.14, 0.09)	.697
WAFG Omissions	290	-0.11	(-0.23, 0.002)	.055
FGT Learning Sum	291	0.11	(-0.001, 0.23)	.053
FGT Immediate recall	291	0.10	(-0.02, 0.21)	.102
FGT Immediate recall errors	291	-0.04	(-0.16, 0.07)	.456
FGT Delayed recall	290	0.12	(0.01, 0.23)	.039
FGT Delayed recall errors	290	-0.004	(-0.12, 0.11)	.944
FGT Recognition	290	0.04	(-0.08, 0.15)	.52[illegible]
FGT Recognition errors	290	-0.12	(-0.23, 0.00)	.05[illegible]
N-back	291	0.09	(-0.03, 0.20)	.1[illegible]
N-back errors	291	-0.13	(-0.24, -0.01)	.030
WISC working memory	290	-0.10	(-0.21, 0.02)	.093
Inhibition errors	291	-0.28	(-0.38, -0.17)	< .001
Inhibition reaction time	291	-0.20	(-0.31, -0.09)	.001
Inhibition variability	291	-0.18	(-0.29, -0.07)	.002
TMT B Flexibility	291	-0.12	(-0.23, -0.01)	.039
TMT B Flexibility errors	291	-0.09	(-0.20, 0.02)	.123
TOL Planning ability	290	0.16	(0.04, 0.027)	.007

Table 4. Group differences in neurocognition in IQ-matched samples

	NSSI M (SD) n = 49	**Control** M (SD) n = 49	*β* (95% CI)	*p*
General intelligence				
WISC IQ [a]	106.29 (11.85)	106.24 (11.70)	0.002 (-0.35, 0.36)	.948
Processing speed				
TMT A time	19.00 (4.20)	18.65 (3.53)	-0.04 (-0.49, 0.31)	.664
TMT A errors [b]	0.08 (0.28)	0.04 (0.20)	0.09 (-0.20, 0.54)	.370
WISC processing speed [c]	107.20 (13.43)	109.98 (13.68)	-0.10 (-0.57, 0.16)	.268
Alertness				
WAFA Mean RT	234.94 (37.72)	233.57 (40.84)	0.02 (-0.37, 0.44)	.864
WAFA Omissions	0.22 (0.59)	0.31 (0.82)	-0.06 (-0.52, 0.29)	.573
Divided attention				
WAFG Mean RT	544.18 (143.52)	531.96 (147.49)	0.04 (-0.32, 0.49)	.678
WAFG Omissions	5.55 (3.91)	6.39 (5.18)	-0.11 (-0.63, 0.17)	.264
Learning				
FGT Learning Sum	31.67 (7.85)	34.18 (7.85)	-0.16 (-0.72, 0.08)	.117
FGT Immediate recall [d]	7.59 (1.51)	8.06 (1.59)	-0.11 (-0.60, 0.17)	.276
FGT Immediate recall errors	1.73 (1.87)	0.69 (1.14)	0.32 (0.25, 1.02)	.001
FGT Delayed recall	7.67 (1.71)	7.94 (1.76)	-0.11 (-0.61, 0.19)	.300
FGT Delayed recall errors [e]	1.02 (1.40)	0.63 (1.25)	0.17 (-0.03, 0.71)	.071
FGT Recognition [f]	8.53 (0.89)	8.79 (0.46)	-0.13 (-0.64, 0.13)	.191
FGT Recognition errors	1.02 (1.27)	0.71 (1.15)	0.13 (-0.14, 0.65)	.207
Working memory				
N-back	10.78 (3.27)	11.45 (3.00)	-0.11 (-0.62, 0.19)	.291
N-back errors	10.65 (14.49)	7.39 (12.83)	0.12 (-0.16, 0.64)	.240
WISC working memory [g]	103.71 (12.41)	105.33 (12.01)	-0.07 (-0.52, 0.25)	.498
Executive Functions				
Inhibition errors	10.08 (4.15)	9.22 (3.58)	0.11 (-0.18, 0.62)	.276
Inhibition reaction time	0.29 (0.06)	0.27 (0.04)	0.13 (-0.15, 0.65)	.212
Inhibition variability	0.12 (0.08)	0.09 (0.05)	0.24 (0.09, 0.87)	.015
TMT B Flexibility [h]	26.89 (9.41)	26.21 (7.88)	-0.03 (-0.43, 0.33)	.782

TMT B Flexibility errors	0.63 (0.83)	0.67 (1.18)	-0.02 (-0.44, 0.36) .844
TOL Planning ability	13.23 (3.37)	14.33 (2.84)	-0.17 (-0.74, 0.05) .086

Note. Beta-coefficients for the main effect of group are reported for the best fitting model. RT = Reaction Time. Several best fitting models included a main effect of sex or an interaction between groups and sex: [a] main effect sex β (95% CI) = -0.50 (-3.38, -1.60), p <.001. [b] main effect sex β (95% CI) = 0.378 (0.95, 2.84), p <.001. [c] main effect sex β (95% CI) = -0.42 (-3.04, -1.2), p <.001. [d] interaction between group and sex β (95% CI) = -0.35 (-4.34, -0.54), p = .012. [e] main effect sex β (95% CI) = 0.52 (1.32, 3.93), p <.001. [f] interaction between group and sex β (95% CI) = -0.36 (-4.43, -0.61), p = .010. [g] main effect sex β (95% CI) = -0.29 (-2.46, -0.50), p <.003. [h] interaction between group and sex β (95% CI) = -0.43 (1.19, 4.93), p = .002.

Table 5. Associations between neurocognitive performance and psychopathology in NSSI patients

	Divided attention		Learning		Working memory		Executive functions					
	Mean RT		Learning sum		N-back		Inhibition Errors [a]		Flexibility		Planning	
	β (95% CI)	*p*	*β* (95% CI)	*p*	*β* (95% CI)	*p*	*β* (95% CI)	*p*	*β* (95% CI)	*p*	*β* (95% CI)	*p*
NSSI												
lifetime	-0.06 (-0.19, 0.07)	.389	0.005 (-0.12, 0.14)	.932	0.04 (-0.09, 0.17)	.502	0.00 (-0.13, 0.13)	.960	-0.05 (-0.18, 0.08)	.422	0.13 (0.00, 0.26)	.045
past year	-0.14 (-0.27, -0.00)	.039	0.15 (0.03, 0.28)	.018	0.08 (-0.05, 0.20)	.249	0.00 (-0.12, 0.13)	.985	-0.11 (-0.24, 0.08)	.088	0.17 (0.04, 0.29)	.010
past month	-0.03 (-0.16, 0.09)	.600	0.19 (0.07, 0.32)	.003	0.02 (-0.11, 0.15)	.786	-0.09 (-0.21, 0.04)	.179	-0.14 (-0.27, -0.02)	.027	0.17 (0.05, 0.30)	.007
Suicide attempts												
lifetime	0.04 (-0.09, 0.17)	.528	-0.16 (-0.29, -0.04)	.012	-0.11 (-0.24, 0.02)	.087	0.04 (-0.09, 0.16)	.571	0.05 (-0.08, 0.18)	.449	-0.07 (-0.19, 0.06)	.301
past year	-0.05 (-0.17, 0.08)	.482	-0.04 (-0.17, 0.08)	.497	-0.01 (-0.14, 0.12)	.867	0.03 (-0.10, 0.15)	.646	-0.02 (-0.15, 0.10)	.704	0.02 (-0.11, 0.15)	.755
Suicidal thoughts												
lifetime	-0.09 (-0.22, 0.04)	.176	-0.00 (-0.13, 0.13)	.991	0.05 (-0.08, 0.18)	.407	0.03 (-0.10, 0.16)	.631	-0.07 (-.20, 0.06)	.315	0.07 (-0.05, 0.20)	.258
past year	-0.12 (-0.24, 0.01)	.075	0.01 (-0.12, 0.14)	.850	0.04 (-0.09, 0.17)	.531	0.09 (-0.04, 0.21)	.159	-0.10 (-0.24, 0.02)	.110	0.08 (-0.04, 0.21)	.193
No. BPD criteria	-0.10 (-0.23, 0.02)	.113	0.04 (-0.08, 0.18)	.434	-0.05 (-0.18, 0.07)	.418	0.03 (-0.10, 0.16)	.628	-0.05 (-0.18, 0.08)	.461	0.02 (-0.11, 0.15)	.789
Depression severity	-0.03 (-0.17, 0.10)	.628	0.07 (-0.07, 0.20)	.340	0.03 (-0.11, 0.17)	.660	0.10 (-0.04, 0.23)	.163	-0.13 (-0.26, 0.01)	.067	0.18 (0.03, 0.30)	.015
Psychopathology severity (CGI)	-.08 (-0.21, 0.05)	.212	-0.01 (-0.14, 0.12)	.855	0.01 (-0.12, 0.14)	.867	0.06 (-0.07, 0.19)	.392	-0.03 (-0.16, 0.10)	.620	0.01 (-0.12, 0.14)	.901
Level of functioning (GAF)	0.06 (-0.07, 0.19)	.341	-0.03 (-0.16, 0.09)	.598	0.04 (-0.09, 0.16)	.592	-0.02 (-0.14, 0.11)	.813	-0.07 (-0.20, 0.06)	.287	-0.06 (-0.19, 0.07)	.335

Note. NSSI = Non-suicidal self-injury, BPD = Borderline Personality Disorder, CGI = Clinical Global Impression Scale, GAF = Global Assessment of Functioning, RT = Reaction time, CI = Confidence Interval. Results from best fitting model are reported. [a] Model including age as a covariate was best, and age was associated with outcome.

Table 6. Model fit comparison for latent class analysis

Model	BIC	AIC	Entropy
One-class	29180.79	29013.72	.
Two-class	28221.94	27967.85	0.95

Note. BIC = Bayesian Information Criterion, AIC = Akaike's Information Criterion

Author Statement

Contributors

Ines Mürner-Lavanchy: conceptualization, methodology, formal analysis, writing of the original draft. **Julian Koenig:** writing - review and editing, investigation. **Stefan Lerch:** methodology, formal analysis, writing - review and editing. **Patrice van der Venne**: writing - review and editing, investigation. **Saskia Höper:** writing - review and editing, investigation. **Franz Resch:** writing - review and editing, funding acquisition. **Michael Kaess:** conceptualization, methodology, writing - review and editing, funding acquisition, supervision.

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Conflict of interest

The authors have no conflict of interest to declare.

Highlights

- Prior findings on neurocognition as underlying mechanism for NSSI are inconsistent
- We applied advanced statistics to neurocognition in a large adolescent NSSI sample
- There was little evidence of neurocognitive group differences apart from IQ
- Findings question the clinical relevance of neurocognition for NSSI in adolescents