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Sleep Characteristics in Adolescents with Major Depressive Disorder: Associations with Non-Suicidal Self-Injury and Suicidality

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ABSTRACT

Background: Sleep disturbances impact outcomes in major depressive disorder (MDD). This cross-sectional study evaluated subjective sleep quality and habits in adolescents with MDD and their associations with suicide attempt (SA) and non-suicidal self-injury (NSSI).
Methods: Sixty-eight adolescents aged 13–18 years with MDD were classified as NSSI-only, SA-only, and combined. Clinical severity and depressive symptoms were measured with Clinical Global Impression-Severity scale and Children's Depression Rating Scale–Revised (CDRS-R). Self-harm and suicidality were evaluated using Inventory of Statements About Self-Injury and Columbia-Suicide Severity Rating Scale. Pittsburgh Sleep Quality Index (PSQI) and Children's Sleep Habits Questionnaire (CSHQ) assessed past-month sleep quality and past-week sleep habits.
Results: Participants had poor sleep quality (89.7%) and disturbed sleep habits (83.8%), without group differences. CSHQ scores correlated moderately with lifetime NSSI frequency ($r = .36$, $P = .003$), whereas PSQI scores correlated weakly ($r = .28$, $P = .023$). PSQI scores were modestly related to past-month suicidal ideation severity ($r = .28$, $P = .022$). In hierarchical regressions controlling for sedative medications and CDRS-R scores, CSHQ ($\beta = 0.299$, $P = .022$) and PSQI ($\beta = 0.248$, $P = .047$) accounted for incremental variance in lifetime NSSI frequency.
Conclusion: Sleep problems are common among adolescents with MDD, suggesting a clinical feature rather than a subgroup-specific trait. Sleep disturbance was associated with lifetime NSSI burden beyond depression severity, indicating a clinically relevant assessment domain. Prospective longitudinal studies should clarify temporal and bidirectional relationships with self-harm outcomes.
Keywords: adolescence; depression; self-injurious behavior; sleep; suicide

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INTRODUCTION

Major depressive disorder (MDD) is a mood disorder accompanied by psychophysiological changes, prominently including sleep disturbances. Since the 1960s, the relationship between depression and sleep has remained a subject of considerable interest, with more than 2000 publications.[1] As depression risk increases throughout adolescence, sleep alterations become consistent biological markers during this developmental period.[2] These observations suggest an association between depressive tendency and sleep disturbances,[3] which are closely linked to episode severity, self-injurious behavior, and suicide risk.[4]

Non-suicidal self-injury (NSSI) is direct, intentional, and repeated destruction of one's own body tissue, without suicidal intent, performed in a manner inconsistent with social expectations and norms.[5] The role of sleep in emotional regulation and cognitive function supports the hypothesis of a bidirectional relationship between sleep and NSSI.[6] Indeed, a higher risk of NSSI has been associated with severe insomnia symptoms, shorter sleep duration, prolonged sleep latency, frequent nocturnal awakenings, and poor overall sleep quality.[7]

Suicidal behavior is a broad term encompassing all death-oriented thoughts and behaviors. While suicidal ideation is defined by the American Psychiatric Association as thoughts of wanting to die, suicide attempt (SA) is defined as self-destructive behavior with direct or indirect evidence of the intent to die, which does not result in death.[8] In adolescents, insomnia is closely associated with suicidal ideation, attempts,[9] and completed suicides.[10] Furthermore, poor

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subjective sleep quality independently increases risk of suicidal ideation.[4,11]

Subjective sleep disturbance has been associated with the onset, recurrence, and severity of depressive episodes.[3] Given that both subjective and objective sleep measures are significant predictors of risk with comparable effect sizes, standardized self-report scales provide a valuable and practical alternative to continuous physiological monitoring.[12]

This study aimed to delineate the sleep characteristics of adolescents with MDD, evaluate the prevalence of sleep problems and comorbidities, and examine whether subjective sleep parameters differ between adolescents with NSSI and those with SA. Although sleep disturbances have been widely studied in adolescent depression, evidence directly comparing sleep characteristics across distinct self-harm phenotypes in clinically diagnosed adolescent MDD remains limited. By integrating both adolescent- and parent-reported sleep measures, we sought to explore whether sleep disturbances function as a transdiagnostic feature of depression or as a behavior-specific correlate within this high-risk population. The specific contribution of this study lies in examining NSSI-only, SA-only, and combined NSSI and SA histories within the same clinical sample, while also evaluating whether sleep parameters are associated with NSSI burden beyond depression severity and sedative medication use. The study aims to contribute descriptive and comparative data to clarify the clinical relevance of sleep disturbances in adolescents with MDD and self-harm histories.

MATERIALS AND METHODS

Participants and Procedures

This cross-sectional study included 68 adolescents aged 13–18 years recruited from the outpatient clinic of the Department of Child and Adolescent Psychiatry at Dokuz Eylül University Faculty of Medicine between March and July 2023. All participants met diagnostic criteria for MDD according to the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL)[13] and had a history of NSSI and/or SA within the past year. Some participants were receiving psychopharmacological treatment at assessment. Exclusion criteria included intellectual disability, autism spectrum disorder, bipolar disorder, psychotic spectrum disorders, or any severe developmental or physical condition precluding assessment.

Depressive symptoms and clinical severity were assessed using the Children's Depression Rating Scale–Revised (CDRS-R)[14] and Clinical Global Impression–Severity scale (CGI-S)[15]. Self-injurious behaviors were assessed using the Inventory of Statements About Self-Injury (ISAS), with NSSI frequency defined as the lifetime frequency of NSSI behaviors reported in the ISAS Behaviors section.[16] Suicidal ideation and behaviors were evaluated using the Columbia-Suicide Severity Rating Scale (C-SSRS).[17] Based on these assessments, the sample was categorized into three distinct subgroups according to the presence of NSSI and/or SA within the past year: NSSI-only (n = 25), defined as those with past-year NSSI and no past-year SA; SA-only (n = 18), defined as those with past-year SA and no past-year NSSI; and a combined NSSI-SA group (n = 25), defined as those with both past-year NSSI and SA. Thus, the term SA-only indicates that NSSI was absent during the past-year classification window. It does not exclude lifetime NSSI, as participants in this group may have had a remote history of NSSI.

Sleep quality and habits were evaluated for all participants using the Pittsburgh Sleep Quality Index (PSQI)[18], completed by the adolescents to assess sleep quality over the past month, and the Children's Sleep Habits Questionnaire (CSHQ)[19], completed by parents or caregivers to assess sleep habits over the previous week. All participants underwent clinical interviews conducted by a child and adolescent psychiatrist. The study protocol was approved by the Dokuz Eylül University Non-Interventional Research Ethics Committee

(Approval No: 2023/04-34; Date: 15/02/2023).

Measures

Validity and reliability have been established for the Turkish versions of the K-SADS-PL,[20] CDRS-R,[21] ISAS,[22] C-SSRS,[23] PSQI,[24] and CSHQ.[25] The CGI-S is scored from 1 to 7.[15] The C-SSRS ideation-intensity subscale is scored from 0 to 25.[17] PSQI global scores > 5 indicate poor sleep quality,[24] whereas CSHQ total scores ≥ 41 are clinically significant.[25]

The Turkish CDRS-R validation study reported a Cronbach α of 0.876,[21] whereas α was 0.70 in this sample. Cronbach α for the ISAS Behaviors section was 0.79 in the Turkish validation study[22] and 0.81 among participants with NSSI in this sample. In the Turkish C-SSRS validation study, Cronbach α for current ideation intensity was 0.93;[23] the value

MAIN POINTS

- Sleep disturbances are highly prevalent in adolescents with major depressive disorder, regardless of the specific self-harm phenotype.
- Both disturbed sleep habits and poorer subjective sleep quality accounted for modest incremental variance in lifetime non-suicidal self-injury frequency, after controlling for depression symptom severity and sedative medication use.
- Sleep impairment may represent a clinically relevant assessment domain that warrants routine evaluation in comprehensive safety planning.

was 0.74 in this sample. Internal consistency of the PSQI was 0.80 in the Turkish validation study[24] and 0.69 in this sample. The CSHQ total score yielded the same Cronbach α in the Turkish validation study and this sample (0.78).[25]

Statistical Analysis

No directly comparable prior study reported summary statistics or effect sizes for sleep parameters across NSSI-only, SA-only, and combined NSSI-SA subgroups in adolescents with MDD. Therefore, the a priori sample size calculation was based on Cohen's conventional large effect size for a fixed-effects omnibus one-way ANOVA comparing three independent groups (Cohen's f = 0.40), as a convention-based assumption rather than a study-derived estimate. With an alpha level of .05, 80% statistical power, and equal group allocation, G*Power version 3.1.9.2 indicated a minimum sample of 66 participants (22 per group). To account for possible outliers, incomplete data, or exclusions, the target sample size was increased to 25 participants per group.

Due to the relative rarity of suicide attempts in the outpatient clinical setting during the study period, the final sample size of the SA-only group (n = 18) fell below this target. A sensitivity analysis based on the final sample size indicated that, at α = .05 and 80% power, the study could detect effects of approximately Cohen's f = 0.39 or larger. Therefore, the achieved sample size was adequate primarily for detecting large effects, whereas medium or smaller effects may have remained undetected. Accordingly, analyses involving suicide attempts—particularly logistic regression models—were considered exploratory rather than confirmatory and intended to inform future studies with larger, more balanced samples.

Statistical analyses were performed using IBM SPSS Statistics for Macintosh, version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive data were presented as frequencies (n) and percentages (%) for categorical variables, and as mean ± standard deviation or median (minimum-maximum) for continuous variables, depending on the distribution. Normality was assessed using the Shapiro–Wilk test.

For group comparisons, the one-way analysis of variance (ANOVA) was used for normally distributed data, and the Kruskal–Wallis H test was used for non-normally distributed data. Levene's test was employed to assess the homogeneity of variances. For post hoc comparisons following a significant Kruskal–Wallis test, Dunn–Bonferroni post hoc tests were performed, and adjusted P values were reported. Categorical variables were compared using Pearson's chi-squared test or the Fisher–Freeman–Halton test, with Bonferroni correction where appropriate.

Eta-squared (η^2) was reported for one-way ANOVA, epsilon-squared (ε^2) for Kruskal–Wallis tests, and Cramér's V for categorical group comparisons analyzed using Pearson's chi-squared or Fisher–Freeman–Halton tests. These values were reported as overall effect sizes for group comparisons. For pairwise comparisons following significant omnibus tests, pairwise effect sizes were also calculated where applicable. For non-parametric pairwise comparisons, Dunn–Bonferroni adjusted P values were reported, and pairwise effect sizes were calculated as r values based on the standardized z statistics of the Dunn post hoc tests.

Confidence intervals for pairwise r values were estimated using Fisher's z transformation where applicable. Cramér's V was used for categorical pairwise comparisons. Confidence intervals were reported in the Results section for statistically significant or clinically emphasized pairwise effect sizes where applicable. For regression analyses, R^2 and change in R^2 (ΔR^2) values were considered indicators of model-level and incremental effect size, respectively. Nagelkerke R^2 was reported for logistic regression models.

Two separate hierarchical multiple linear regression analyses were conducted to examine factors associated with NSSI frequency. Prior to analysis, NSSI frequency was log-transformed [log(1+NSSI)] to better approximate normality and stabilize variance. Independent variables were entered in two blocks: sedative medication use (antipsychotics and/or benzodiazepines) and depression severity (CDRS-R T-score) were entered in the first step, followed by the respective sleep parameter (PSQI or CSHQ total score) in the second step to observe its incremental contribution to the model.

Finally, binary logistic regression analyses were conducted to examine whether subjective sleep quality (PSQI total score) and parent-reported sleep habits (CSHQ total score) were associated with the presence of suicide attempts (yes/no). Separate models were estimated for each sleep scale. Sedative medication use and depressive symptom severity were included as covariates. Model fit was evaluated using the Hosmer–Lemeshow goodness-of-fit test.

There were no missing data for the primary study variables; all 68 participants completed the PSQI, CSHQ, CDRS-R, CGI-S, ISAS, and C-SSRS assessments, and all cases were therefore included in every analysis. The statistical significance level was set at $P < .05$ for all analyses (two-tailed).

RESULTS

Sociodemographic Characteristics and Clinical History

Participants had a mean age of 15.9 ± 1.4 years and were predominantly female (91.2%). The groups were comparable in most sociodemographic and clinical characteristics. Education level differed overall (P = .042), but no post hoc comparison was significant (NSSI vs SA: P = 1.000; NSSI vs NSSI-SA: P = .150; SA vs NSSI-SA: P = 1.000). Cigarette use differed across groups (P = .024), and Bonferroni-adjusted post hoc comparisons showed higher use in the combined NSSI-SA group than in the SA-only group (adjusted P = .027,

Table 1. Comparison of sociodemographic variables among case groups.

	NSSI (n=25)	SA (n=18)	NSSI and SA (n=25)	*P* value	Effect size	Pairwise comparisons, adj. *P* values and effect size
Age, mean ± SD	16.1 ± 1.5	16.0 ± 1.3	15.7 ± 1.3	.547[1]	.018[a]	
Gender, Female, n (%)	23 (92.0%)	16 (88.9%)	23 (92.0%)	1.0[2]	.048[b]	
Education level n, (%)				**.042**[2]	.310[b]	NSSI vs SA: *P* = 1.000, *V* = .206; NSSI vs NSSI-SA: *P* = .150, *V* = .333; SA vs NSSI-SA: *P* = 1.000, *V* = .182
Middle school	5 (20.0%)	1 (5.6%)	0 (0.0%)			
High school	20 (80.0%)	17 (94.4%)	25 (100.0%)			
Academic achievement, n, (%)				.261[3]	.197[b]	
Good	7 (28.0%)	10 (55.5%)	8 (32.0%)			
Moderate	6 (24.0%)	4 (22.2%)	4 (16.0%)			
Bad	12 (48.0%)	4 (22.2%)	13 (52.0%)			
Peer relationships, n, (%)				.443[3]	.166[b]	
Good	10 (40.0%)	9 (50.0%)	6 (24.0%)			
Moderate	9 (36.0%)	4 (22.2%)	11 (44.0%)			
Bad	6 (24.0%)	5 (27.8%)	8 (32.0%)			
Family structure, n, (%)				.207[2]	.181[b]	
Married	16 (64.0%)	16 (88.9%)	17 (68.0%)			
Divorced	9 (36.0%)	2 (11.1%)	7 (28.0%)			
Deceased Parent	0 (0.0%)	0 (0.0%)	1 (4.0%)			
Maternal education level, n, (%)				.859[2]	.141[b]	
Primary school or less	7 (28.0%)	6 (33.3%)	9 (36.0%)			
Middle school	4 (16.0%)	1 (5.6%)	5 (20.0%)			
High school	9 (36.0%)	8 (44.4%)	7 (28.0%)			
University or higher	5 (20.0%)	3 (16.7%)	4 (16.0%)			
Paternal education level, n, (%)				.997[2]	.069[b]	
Primary school or less	8 (32.0%)	4 (22.2%)	8 (32.0%)			
Middle school	4 (16.0%)	3 (16.7%)	4 (16.0%)			
High school	7 (28.0%)	6 (33.3%)	7 (28.0%)			
University or higher	6 (24.0%)	5 (27.8%)	6 (24.0%)			

Smoking, n, (%)	9 (36.0%)	5 (27.8%)	17 (68.0%)	**.024**[3]	.349[b]	NSSI vs SA: *P* = 1.000, *V* = .087; NSSI vs NSSI-SA: *P* = .072, *V* = .320; SA vs NSSI-SA: ***P* = .027, *V* = .397**
Alcohol use, n, (%)	9 (36.0%)	3 (16.7%)	13 (52.0%)	.060[3]	.288[b]	
Substance use, n, (%)	2 (8.0%)	1 (5.6%)	7 (28.0%)	.087[2]	.287[b]	
Psychotropic medication use, n, (%)	14 (56.0%)	14 (77.8%)	20 (80.0%)	.130[3]	.245[b]	
Antidepressants	13 (52.0%)	9 (50.0%)	13 (52.0%)	.989[3]	.018[b]	
Antipsychotics	10 (40.0%)	13 (72.2%)	14 (56.0%)	.110[3]	.255[b]	
Benzodiazepines	0 (0.0%)	0 (0.0%)	1 (4.0%)	1.0[2]	.160[b]	
Mood Stabilizers	0 (0.0%)	1 (5.6%)	2 (8.0%)	.476[2]	.170[b]	
Psychostimulants	1 (4.0%)	0 (0.0%)	2 (8.0%)	.776[2]	.154[b]	
Medical history, n, (%)	13 (52.0%)	10 (55.6%)	17 (68.0%)	.489[3]	.145[b]	
Family history of psychiatric disorders, n, (%)	11 (44.0%)	5 (27.8%)	18 (72.0%)	**.013**[2]	.359[b]	NSSI vs SA: *P* = .834, *V* = .166; NSSI vs NSSI-SA: *P* = .135, *V* = .166; SA vs NSSI-SA: ***P* = .012, *V* = .437**

Note: Statistically significant P values (P < .05) are presented in bold. Pairwise comparisons were Bonferroni-corrected where appropriate. Effect sizes refer to overall group comparisons unless reported within the pairwise comparisons column. Pairwise effect sizes for categorical comparisons are presented as Cramér's V. Abbreviations: NSSI, non-suicidal self-injury; SA, suicide attempt; SD, standard deviation. [1] *one-way ANOVA,* [2] *Fisher-Freeman-Halton test,* [3] *Pearson's chi-squared test.* *Eta-squared (η²),* *Cramér's V.*

Cramér's *V* = .397, 95% CI [0.109, 0.669]). Other comparisons were nonsignificant (NSSI vs SA: *P* = 1.000; NSSI vs NSSI-SA: *P* = .072). Family history of psychiatric disorders also differed (*P* = .013), with a higher frequency in the combined NSSI-SA group than in the SA-only group (adjusted *P* = .012, Cramér's *V* = .437, 95% CI [0.151, 0.706]). Other comparisons were nonsignificant (NSSI vs SA: *P* = .834; NSSI vs NSSI-SA: *P* = .135) (Table 1).

Psychiatric Comorbidities and Clinical Severity

All participants met the diagnostic criteria for MDD, and 70.6% presented with at least one comorbid psychiatric diagnosis. The most prevalent comorbidities were anxiety disorders (52.9%) and attention-deficit/hyperactivity disorder (14.7%), followed by eating disorders (11.8%). Other less frequent comorbidities included disruptive, impulse-control, and conduct disorders, trauma and stressor-related disorders, obsessive-compulsive disorder, dissociative disorders, and somatic symptom disorders. Group comparisons revealed no significant differences in the presence or type of comorbid diagnoses (all *P* > .05).

CDRS-R T-scores were similar across groups (*P* = .384), whereas CGI-S scores differed significantly (*P* = .005). Post hoc comparisons showed that the NSSI-only group had lower CGI-S scores than the SA-only group (adjusted *P* = .024, *r* = −.405, 95% CI [−0.629, −0.119]) and the combined NSSI-SA group (adjusted *P* = .010, *r* = −.415, 95% CI [−0.622, −0.154]) (Table 2).

Comparison of Sleep Characteristics Among Groups

Mean sleep duration was 7.4 ± 2.7 hours on the PSQI and 7.9 ± 2.3 hours on the CSHQ, without between-group differences. Poor sleep quality (89.7%) and disturbed sleep habits (83.8%) were common; however, total PSQI and CSHQ scores and rates of insomnia (33.8%) and hypersomnia

Table 2. Comparison of depression severity and general illness severity among case groups.

	NSSI (n=25)	SA (n=18)	NSSI and SA (n=25)	*P* value	Effect size (ε^2)	Pairwise comparisons, adj. *P* values and effect size
CDRS-R T-score	74 (66–85)	74.5 (59–85)	80 (61–85)	.384	.029	
CGI-S score	4.0 (3–6)	5.0 (3–7)	5.0 (3–7)	.005	.161	NSSI vs SA: *P* = .024, *r* = −.405; NSSI vs NSSI-SA: *P* = .010, *r* = −.415; SA vs NSSI-SA: *P* = 1.000, *r* = −.005

Note: Kruskal-Wallis test was used. The median and min-max values are presented. Statistically significant P values (P < .05) are presented in bold. Dunn-Bonferroni adjusted P values are reported for pairwise comparisons. Effect sizes (ε^2) refer to overall group comparisons; pairwise effect sizes are presented as Dunn-test-based r values. Abbreviations: CDRS-R, Children's Depression Rating Scale-Revised; CGI-S, Clinical Global Impression-Severity scale; NSSI, non-suicidal self-injury; SA, suicide attempt

(13.2%) did not differ across groups (all $P > .05$). In an exploratory subscale analysis, the CSHQ sleep onset delay subscale differed significantly among groups ($P = .006$). Post hoc tests showed that the NSSI-only group had higher scores than the SA-only group (adjusted $P = .005$, $r = .479$, 95% CI [0.209, 0.681]) and that the SA-only group had lower scores than the combined NSSI-SA group (adjusted $P = .049$, $r = -.366$, 95% CI [−0.600, −0.074]) (Table 3).

Correlation Analysis

Total PSQI scores showed a weak positive correlation with the severity of past-month suicidal ideation ($r = .28$, $P = .02$). Lifetime NSSI frequency demonstrated a moderate positive correlation with CSHQ total scores ($r = .36$, $P = .003$) and a weak-to-moderate correlation with PSQI total scores ($r = .28$, $P = .023$). Lifetime NSSI frequency also showed a weak positive correlation with past-month suicidal ideation severity ($r = .29$, $P = .02$) (Table 4).

Regression Analyses of Sleep Parameters on Self-Harm Behaviors

Association with Subjective Sleep Quality (PSQI): Sedative medication use and depression severity were entered in Step 1. The model was nonsignificant (model $P = .082$) and explained 7.4% of the variance ($R^2 = .074$). At this stage, depression severity was the only significant predictor ($\beta = 0.268$, $P = .029$). Adding PSQI in Step 2 significantly improved the model ($\Delta R^2 = .056$, $P = .047$). The final model was statistically significant overall (model $P = .030$) and explained 12.9% of the variance ($R^2 = .129$). In the final model, poorer subjective sleep quality was significantly associated with higher log-transformed NSSI frequency ($\beta = 0.248$, $P = .047$), while both covariates were nonsignificant (Table 5).

Association with Sleep Habits (CSHQ): Step 1 used the same covariates. Adding CSHQ in Step 2 significantly improved the model ($\Delta R^2 = .073$, $P = .022$). The final model was statistically significant overall and explained 14.7% of the variance ($R^2 = .147$, model $P = .017$). Disturbed sleep habits were significantly associated with higher log-transformed NSSI frequency ($\beta = 0.299$, $P = .022$), whereas both covariates were nonsignificant (Table 6).

Logistic regression analyses did not demonstrate significant associations between sleep measures and SA in this sample. In the PSQI model, subjective sleep quality was not associated with SA (OR = 0.99, 95% CI = 0.85–1.15, $P = .88$), and the overall model was nonsignificant (model $P = .55$, Nagelkerke $R^2 = .05$). In the CSHQ model, parent-reported sleep habits were not associated with SA (OR = 0.93, 95% CI = 0.86–1.01, $P = .094$). The model explained a limited proportion of variance (Nagelkerke $R^2 = .106$), and the overall model was nonsignificant (model $P = .168$). Moreover, the Hosmer–Lemeshow goodness-of-fit test indicated suboptimal model fit ($P = .031$). Sedative medication use (OR = 1.68, 95% CI = 0.52–5.38, $P = .383$) and depressive symptom severity (OR = 1.07, 95% CI = 0.97–1.17, $P = .177$) were also nonsignificant (Table 7).

DISCUSSION

Evaluation of Sociodemographic Risk Factors

The mean age of approximately 16 years in our study was consistent with the literature examining depression and sleep in adolescents.[26] The female predominance reflected the established higher prevalence of depression,[27] SA[28] and NSSI[29] among girls during the transition to adolescence. Cigarette use was higher in the combined NSSI-SA group than in the SA-only group. Although some studies report

Table 3. Comparison of sleep patterns among case groups.

	NSSI (n=25)	SA (n=18)	NSSI and SA (n=25)	*P* value	Effect size	Pairwise comparisons, adj. *P* values and effect size
PSQI score, mean ± SD	10.8 ± 3.2	8.7 ± 3.9	10.8 ± 4.3	.133[1]	.060[a]	
PSQI components, median, (min-max)						
Subjective sleep quality	2 (1–3)	1.5 (1–3)	2 (1–3)	.115[2]	.065[b]	
Sleep latency	2 (0–3)	2 (0–3)	2 (0–3)	.316[2]	.034[b]	
Sleep duration	0 (0–3)	0 (0–2)	1 (0–3)	.252[2]	.041[b]	
Habitual sleep efficiency	0 (0–3)	0 (0–3)	0 (0–3)	.298[2]	.036[b]	
Sleep disturbances	2 (1–3)	2 (1-3)	2 (1–3)	.665[2]	.012[b]	
Use of sleeping medications	0 (0–3)	0 (0-3)	0 (0–3)	.423[2]	.026[b]	
Daytime dysfunction	3 (1–3)	3 (0–3)	2 (1–3)	.445[2]	.024[b]	
CSHQ score, mean ± SD	52.8 ± 8.5	47.2 ± 5.7	50.0 ± 8.1	.076[1]	.076[a]	
CSHQ components						
Bedtime resistance, median, (min-max)	9 (6–18)	8 (6–11)	7 (6–13)	.059[2]	.084[b]	
Sleep onset delay, median, (min-max)	3 (1–3)	1.5 (1–3)	3 (1–3)	.006[2]	.155[b]	NSSI vs SA: *P* = .005, *r* = .479; NSSI vs NSSI-SA: *P* = 1.000, *r* = .115; SA vs NSSI-SA: *P* = .049, *r* = −.366
Sleep anxiety, median, (min-max)	4 (4–10)	4 (4–6)	4 (4–8)	.106[2]	.067[b]	
Night wakings, median, (min-max)	4 (3–8)	4 (3–6)	5 (3–7)	.213[2]	.046[b]	
Parasomnias, mean ± SD	9.5 ± 2.0	9.0 ± 1.6	9.1 ± 1.5	.578[1]	.017[a]	
Sleep disordered breathing, median, (min-max)	3 (3–8)	3.5 (3–6)	4 (3–7)	.654[2]	.013[b]	
Daytime sleepiness, median, (min-max)	15 (10–21)	13 (8–20)	14 (8–21)	.832[2]	.006[b]	
Sleep Disorders, n (%)						
Insomnia	10 (40.0%)	6 (33.3%)	7 (28.0%)	.668[3]	.109[c]	
Hypersomnia	3 (12.0%)	0 (0.0%)	6 (24.0%)	.061[4]	.279[c]	

Note: Statistically significant P values (P < .05) are presented in bold. Dunn-Bonferroni adjusted P values are reported for pairwise comparisons following significant omnibus tests. No additional correction was applied across the multiple sleep components and subscales examined. Effect sizes refer to overall group comparisons unless reported within the pairwise comparisons column. Pairwise effect sizes are presented as Dunn-test-based r values. Abbreviations: CSHQ, Children's Sleep Habits Questionnaire; NSSI, non-suicidal self-injury; PSQI, Pittsburgh Sleep Quality Index; SA, suicide attempt; SD, standard deviation. 1 one-way ANOVA, 2 Kruskal-Wallis test, 3 Pearson's chi-squared test, 4 Fisher-Freeman-Halton test. [a] Eta-squared (η^2), [b] Epsilon-squared (ε^2), [c] Cramér's V.

Table 4. Spearman correlations between sleep measures, NSSI frequency, and suicidal ideation.

	1	2	3	4
1. PSQI	–			
2. CSHQ	.367*	–		
3. NSSI frequency, lifetime	.275*	.360**	–	
4. Severity of suicidal ideation, past month	.277*	.098	.294*	–

*Note: Statistically significant correlation coefficients (P < .05) are presented in bold * P < .05, ** P < .01. Abbreviations: CSHQ, Children's Sleep Habits Questionnaire; NSSI, non-suicidal self-injury; PSQI, Pittsburgh Sleep Quality Index*

Table 5. Hierarchical regression analysis of associations between subjective sleep quality (PSQI) and lifetime NSSI frequency.

Variable	ΔR^2	Overall model *P*	B	SE	*β*	95% CI for B	*P*
Step 1	.074	.082					
Constant			−1.068	1.368	–	[−3.800, 1.663]	.438
Sedative medication use			−0.174	0.241	−0.087	[−0.655, 0.307]	.473
CDRS-R T-score			0.040	0.018	0.268	[0.004, 0.076]	.029
Step 2	.056	.030					
Constant			−0.908	1.339	–	[−3.582, 1.766]	.500
Sedative medication use			−0.114	0.237	−0.057	[−0.588, 0.360]	.632
CDRS-R T-score			0.029	0.018	0.193	[−0.008, 0.066]	.121
PSQI Total			0.064	0.032	0.248	[0.001, 0.127]	.047

Note: n = 68. Sedative medication use was dummy coded (0 = No, 1 = Yes). Dependent variable: log(1+NSSI). Final cumulative R^2 = .129. For Step 1, ΔR^2 is equivalent to R^2. The Overall model P column indicates the overall significance of each regression model, whereas the P column indicates predictor-level P values. Statistically significant P values (P < .05) are presented in bold. Abbreviations: B, unstandardized coefficient; SE, standard error; B, standardized coefficient; CI, confidence interval; CDRS-R, Children's Depression Rating Scale–Revised; PSQI, Pittsburgh Sleep Quality Index; NSSI, non-suicidal self-injury.

elevated smoking rates in SA cases,[30] others find no significant differences,[31] highlighting the complexity of this association. Furthermore, higher familial psychopathology in the combined group aligns with reported parental problems in self-harming adolescents[32] and may reflect familial vulnerability to more severe self-harm phenotypes. The possible small-to-moderate effect was imprecisely estimated, as reflected by wide confidence intervals, and should therefore be interpreted cautiously.

Clinical Characteristics and Severity Across Clinical Subgroups

Approximately 70% of participants had at least one comorbidity. Anxiety disorders were the most prevalent, consistent with findings in adolescent depression[33] and studies comparing NSSI and suicidal behaviors.[34]

Aligning with the Treatment of Resistant Depression in Adolescents (TORDIA) study, depression severity was similar across groups, suggesting that neither NSSI nor suicidal behavior necessarily corresponds to MDD severity.[35] Nevertheless, CGI-S scores were significantly lower in the NSSI-only group compared with the other groups. The CGI-S effect size estimates were consistent with possible group differences, although these should be interpreted cautiously given the modest and unbalanced subgroup sizes. This distinction likely reflects the clinician's perception of "imminent risk"; SA and combined NSSI-SA present a more urgent clinical picture compared with NSSI alone. Alternatively, this may suggest a progression in severity whereby adolescents initially engaging in NSSI may escalate to suicidal behaviors if their distress remains unresolved. While sleep disturbances may exacerbate suicidal ideation, progression to an attempt may involve rumination and impulsivity beyond depression severity.[36,37]

Table 6. Hierarchical regression analysis of associations between sleep habits (CSHQ) and lifetime NSSI frequency.

Variable	ΔR^2	Overall model *P*	B	SE	*β*	95% CI for B	*P*
Step 1	.074	.082					
Constant			−1.068	1.368	–	[−3.800, 1.663]	.438
Sedative medication use			−0.174	0.241	−0.087	[−0.655, 0.307]	.473
CDRS-R T-score			0.040	0.018	0.268	[0.004, 0.076]	.029
Step 2	.073	.017					
Constant			−1.542	1.338	–	[−4.215, 1.132]	.254
Sedative medication use			−0.092	0.236	−0.046	[−0.563, 0.378]	.696
CDRS-R T-score			0.021	0.019	0.142	[−0.017, 0.060]	.273
CSHQ Total			0.037	0.016	0.299	[0.005, 0.069]	.022

Note: n = 68. Sedative medication use was dummy coded (0 = No, 1 = Yes). Dependent variable: log(1+NSSI). Final cumulative R^2 = .147. For Step 1, ΔR^2 is equivalent to R^2. The Overall model P column indicates the overall significance of each regression model, whereas the P column indicates predictor-level P values. Statistically significant P values (P < .05) are presented in bold. Abbreviations: B, unstandardized coefficient; SE, standard error; β, standardized coefficient; CI, confidence interval; CDRS-R, Children's Depression Rating Scale–Revised; CSHQ, Children's Sleep Habits Questionnaire; NSSI, non-suicidal self-injury.

Table 7. Logistic regression analyses of associations between sleep measures and suicide attempt status.

Variable	Nagelkerke R^2	Overall model *P*	B	SE	Adjusted OR	95% CI for OR	*P*
PSQI Model	.045	.554					
Sedative medication use			0.666	0.577	1.946	[0.629, 6.025]	.248
CDRS-R T-score			0.032	0.045	1.032	[0.945, 1.126]	.483
PSQI Total			−0.012	0.076	0.988	[0.851, 1.148]	.878
CSHQ Model	.106	.168					
Sedative medication use			0.518	0.594	1.679	[0.524, 5.376]	.383
CDRS-R T-score			0.066	0.049	1.068	[0.971, 1.175]	.177
CSHQ Total			−0.068	0.041	0.934	[0.863, 1.012]	.094

Note: n = 68. The dependent variable was SA status (0 = No, 1 = Yes). Sedative medication use was dummy coded (0 = No, 1 = Yes). All ORs are adjusted for the other variables included in the same model. The Overall model P column indicates model-level significance, whereas the final P column indicates predictor-level significance. Abbreviations: B, unstandardized logit coefficient; CDRS-R, Children's Depression Rating Scale-Revised; CI, confidence interval; CSHQ, Children's Sleep Habits Questionnaire; OR, odds ratio; PSQI, Pittsburgh Sleep Quality Index; SA, suicide attempt; SE, standard error.

Sleep Patterns Across Clinical Subgroups

Poor sleep quality and disturbed sleep habits were observed in over 80% of our participants, with no group differences. These findings suggest that sleep disruption is a common feature of adolescent MDD across self-harm phenotypes.[38]

An unexpected exploratory finding was that the CSHQ sleep onset delay score was significantly lower, indicating fewer problems, in the SA-only group compared to the other groups. The effect size estimates for sleep onset delay suggested a possible small-to-moderate group difference; however, the relatively wide confidence intervals indicated limited precision. Therefore, this exploratory subscale finding should be interpreted cautiously rather than as evidence of a stable subgroup-specific sleep phenotype. While the literature generally associates prolonged sleep latency with suicide risk,[39-41] this discrepancy may reflect clinical heterogeneity. Adolescents with NSSI may suffer more from pre-sleep depressive rumination and difficulty quieting intrusive thoughts,[6,7] whereas SA may be characterized by high impulsivity and rapid escalation of distress rather than prolonged wakefulness.[30,32] Additionally,

higher sedative antipsychotic use in the SA group (72.2%) may have attenuated onset difficulties. Parent reports may also underestimate internal sleep-onset difficulties relative to visible restlessness associated with NSSI.

According to Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria, insomnia and hypersomnia were diagnosed in 33.8% and 13.2% of participants, respectively. Although insomnia is the most prevalent sleep disorder among depressed youth,[42] the absence of group differences suggests that these sleep phenotypes may reflect underlying depression rather than self-harm type.

Associations of Sleep Parameters with NSSI and Suicidal Behaviors

A notable explanatory finding was that poorer sleep quality (PSQI) and more disturbed sleep habits (CSHQ) each explained incremental variance in lifetime NSSI frequency, beyond depression severity and sedative medication use. However, the amount of variance explained by the final models was modest, indicating that sleep parameters should be interpreted as clinically relevant but limited correlates of NSSI burden rather than as major determinants of self-injury. This limited contribution also underscores the multifactorial nature of NSSI and suggests that the unexplained variance may be attributable to other biological, psychological, and contextual factors.[43] Although sleep scores accounted for incremental variance after controlling for CDRS-R scores, they may still partly capture symptom variance shared with depression severity. Similar findings across adolescent (PSQI) and parent (CSHQ) reports suggest consistency across informants. These findings may be interpreted in the context of emotion regulation models, which propose links between sleep disturbance, prefrontal control, emotion regulation, and vulnerability to NSSI.[7] Related literature, including longitudinal data[44] and polysomnographic evidence[45], has highlighted associations between sleep disturbance and NSSI. Nevertheless, given the cross-sectional design and differing assessment time frames, the present findings should be interpreted as associations between recent sleep problems and cumulative NSSI history rather than as evidence of temporal ordering, causality, or independent mechanistic significance. Taken together with recent prediction-focused studies, including that of He et al., these findings suggest that sleep disturbance may represent a clinically relevant dimension that complements established psychosocial risk frameworks, rather than replacing them.[46]

Regarding suicidality, poorer past-month sleep quality was weakly correlated with the severity of suicidal ideation during the same period. This supports the view that the relationship between sleep and suicidality may be stronger during acute periods.[12] Theoretically, poor sleep may undermine resilience and increase vulnerability to suicidal ideation through emotional dysregulation and heightened arousal.[47,48]

Contrary to our expectations, sleep parameters were not significantly associated with SA in this sample. Although the CSHQ logistic regression model yielded a predictor-level *P* value close to conventional significance, this result should not be interpreted as trend-level or suggestive evidence, because the model showed suboptimal fit on the Hosmer-Lemeshow test. Accordingly, although the point estimate for parent-reported sleep habits was below unity (OR = 0.93), its confidence interval included 1, thus precluding firm conclusions about the direction of the association. The suboptimal fit may be related to the small and unbalanced subgroup sizes, particularly the limited size of the SA-only group. Possible nonlinearity of continuous predictors and unmeasured clinical confounding may also have contributed. Thus, the nonsignificant findings may reflect limited statistical power and model-fit constraints rather than a true absence of association. Alternatively, social support, clinical monitoring, or ongoing treatment may buffer progression from suicidal ideation to action despite sleep disturbances.[12,49] Therefore, the potential role of sleep-related factors in the transition from suicidal ideation to attempt should be examined in larger and more homogeneous samples.

Limitations

The results of this study should be interpreted in light of several limitations. The cross-sectional design precludes causal inference between sleep disturbances and self-harm behaviors. The predominance of female participants may limit the generalizability of the findings, particularly to male adolescents. Given the modest and unbalanced sample, especially the small SA-only group, analyses involving this subgroup and SA-related outcomes may have had limited power and were therefore exploratory and hypothesis-generating rather than confirmatory. Logistic regression estimates and nonsignificant findings regarding sleep parameters and SA require caution because model-fit constraints limit inference and Type II error may obscure a true association.

Another limitation is that subgroup classification was based on the presence of NSSI and/or SA within the past year. Therefore, adolescents classified as SA-only may have had a remote history of NSSI outside this one-year window, potentially introducing heterogeneity within this group. Relatedly, there was incomplete alignment between the assessment time frames of the primary variables. While NSSI frequency was operationalized as lifetime frequency using the ISAS Behaviors section, sleep quality and sleep habits were assessed over the past month and past week, respectively. Therefore, the observed associations should be interpreted as relationships between current or recent sleep disturbances and cumulative NSSI burden, rather than temporally proximal or causal effects. Sleep assessment also relied on questionnaire-based adolescent and parent reports. Although two informants may provide complementary perspectives, these measures remain

subjective and may be affected by recall bias, reporting bias, and individual or parental perceptions of sleep problems. The absence of objective sleep measures, such as actigraphy or polysomnography, limited our ability to characterize sleep duration, sleep efficiency, sleep architecture, and night-to-night variability more precisely. In addition, the modest internal consistency of the PSQI and the lower-bound acceptable reliability coefficient of the CDRS-R should be considered when interpreting associations involving subjective sleep quality and depressive symptom severity. Future studies incorporating objective and subjective sleep assessments may provide a more comprehensive evaluation of sleep disturbances in adolescents with MDD and self-harm histories.

Lastly, most participants were receiving pharmacological treatment. Although antipsychotics and benzodiazepines were combined into a single sedative medication variable and included as a covariate, this approach may not fully account for the heterogeneous effects of different psychotropic agents on sleep architecture, sleep continuity, daytime sleepiness, and other sleep-related symptoms; given the very low frequency of benzodiazepine use in this sample, this combined variable largely reflected antipsychotic exposure. Because medication dose, duration, timing of administration, and specific pharmacological profiles could not be modeled in detail, residual medication-related confounding cannot be excluded.

CONCLUSION

In summary, sleep disturbances were common but did not differ across self-harm subgroups in adolescents with MDD. Poorer adolescent-reported sleep quality and more disturbed parent-reported sleep habits were associated with greater lifetime NSSI burden beyond depression severity and sedative medication use. Given the cross-sectional design, differing assessment time frames, and sample limitations, these findings should be viewed as preliminary associations between recent sleep problems and cumulative NSSI history rather than evidence of temporal or causal relationships. Future studies should ideally use prospective longitudinal designs with repeated sleep assessments to clarify the temporal and potentially bidirectional relationships between sleep disturbances and self-harm outcomes and examine whether sleep improvements are associated with subsequent changes in NSSI or suicidal behaviors.

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Informed Consent: Written informed consent was obtained from all participants and their parents or legal guardians prior to participation.

Declaration of Helsinki: The study was conducted in accordance with the principles of the Declaration of Helsinki.

Reporting Guideline: This study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies. The completed STROBE checklist is provided as Supplementary Material 1.

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