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Original Article

A Trial Testing Whether Targeted Sleep-Wake Rhythm Stabilization Outperforms Cognitive Therapy for Adolescent Anxiety Comorbid with Depression

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ABSTRACT

Background: Anxiety and depression frequently co-occur during adolescence and are often accompanied by irregular sleep–wake patterns that may worsen emotional dysregulation and functional impairment. Targeting sleep timing and circadian regularity may provide a biologically informed therapeutic approach for adolescents whose internalizing symptoms are linked with unstable daily rhythms. Objective: To compare the effectiveness of targeted sleep–wake rhythm stabilization with cognitive therapy in reducing anxiety and depressive symptoms among adolescents with co-occurring anxiety, depression, and persistent sleep–wake irregularity. Methods: A parallel-group randomized controlled trial was conducted over eight weeks in the Islamabad–Rawalpindi metropolitan region. Adolescents aged 14–18 years with clinically significant anxiety and depressive symptoms and sleep–wake irregularity for at least three months were randomized to targeted sleep–wake rhythm stabilization or cognitive therapy. Anxiety and depressive symptoms were assessed using the Generalized Anxiety Disorder-7 and Patient Health Questionnaire-9, while sleep regularity was monitored using daily sleep diaries and wrist-worn actigraphy. Results: Among 57 completers, the sleep–wake group showed greater reductions in GAD-7 scores than the cognitive therapy group (6.2 ± 2.1 vs 3.8 ± 1.9) and greater reductions in PHQ-9 scores (5.9 ± 2.3 vs 3.5 ± 2.0). Sleep irregularity improved more in the sleep–wake group (1.8 ± 0.6 vs 0.6 ± 0.5 hours), and clinically significant improvement occurred in 57.1% versus 27.6% of participants. Conclusion: Targeted sleep–wake rhythm stabilization produced greater short-term improvement in anxiety, depressive symptoms, and sleep regularity than cognitive therapy, supporting its clinical relevance as a structured intervention for adolescents with emotional symptoms and irregular sleep–wake patterns. Keywords: Adolescent; Anxiety; Depression; Circadian Rhythm; Sleep–Wake Rhythm; Cognitive Therapy.

INTRODUCTION

Adolescent anxiety and depression frequently co-occur and represent a clinically important public health concern because their combined presentation is associated with greater symptom persistence, impaired academic and social functioning, poorer treatment response, and increased risk of long-term psychological morbidity. During adolescence, rapid neurobiological maturation, changing sleep biology, academic pressure, peer-related stress, and increasing autonomy in daily routines can intensify emotional vulnerability. When anxiety and depressive symptoms occur together, they may reinforce one

another through avoidance, rumination, reduced motivation, disrupted social participation, and declining school performance, making early and targeted intervention particularly important (1). Although psychological therapies remain central to adolescent mental health care, a considerable proportion of young people experience incomplete symptom remission, suggesting that additional modifiable mechanisms contributing to emotional dysregulation require closer clinical attention (2).

Sleep–wake disruption is one such mechanism that is highly relevant during adolescence. Delayed bedtimes, inconsistent wake times, irregular weekday–weekend schedules, shortened sleep duration, evening screen exposure, and circadian misalignment are common in this age group and may interact with emotional symptoms through physiological, cognitive, and behavioral pathways. Disturbed sleep timing can increase daytime fatigue, impair attentional control, heighten emotional reactivity, and reduce stress tolerance, all of which may worsen anxiety and depressive symptoms. Emerging evidence indicates that the sleep–circadian system is closely linked with neuroaffective development and mental disorder vulnerability, making rhythm instability more than a secondary symptom in adolescents with internalizing problems (3). This relationship is especially important in adolescents with comorbid anxiety and depression, in whom sleep irregularity may maintain distress by weakening emotion regulation and disrupting daily functioning (4).

Conventional cognitive-based therapies target maladaptive thoughts, avoidance patterns, behavioral withdrawal, and coping deficits, and they are widely used for adolescent anxiety and depressive symptoms. However, these interventions often require sustained attention, verbal engagement, reflective capacity, motivation, and consistent homework completion, which may be difficult for adolescents experiencing fatigue, low mood, excessive worry, or poor sleep. Moreover, cognitive therapy may not directly address biological rhythm disruption when irregular sleep timing is a central contributor to symptom persistence. If circadian misalignment remains untreated, improvements in cognitive and emotional functioning may be limited, even when psychological strategies are delivered appropriately (5). This limitation supports the need to evaluate interventions that directly target sleep timing and daily rhythm stability as potential therapeutic mechanisms in adolescent mental health care (6).

Targeted sleep–wake rhythm stabilization is a structured, biologically informed approach that prioritizes consistency in wake time, bedtime routines, light exposure, evening screen regulation, and daytime activity scheduling. Unlike general sleep advice, rhythm stabilization focuses on reducing variability in sleep–wake timing and strengthening environmental cues that support circadian alignment. By improving regularity rather than only increasing sleep duration, this intervention may reduce physiological arousal, improve daytime alertness, support emotional regulation, and enhance the adolescent's capacity to engage in school, family, and social activities. Such strategies may be practical and scalable because they are behavioral, low-cost, and potentially acceptable to adolescents who struggle with traditional talk-based approaches (7). Prior work has suggested that sleep-focused and circadian-informed interventions may improve internalizing symptoms, but these approaches are often examined as adjunctive components rather than as primary interventions compared directly with established psychological treatment models (8).

Despite growing recognition of the sleep–mental health relationship, direct comparative evidence remains limited regarding whether targeted sleep–wake rhythm stabilization can produce greater reductions in anxiety and depressive symptoms than cognitive therapy among adolescents with both emotional distress and irregular sleep patterns. This gap is clinically important because treatment selection for adolescents may improve if interventions are matched to maintaining mechanisms such as circadian instability, fatigue, and daily rhythm disruption. A direct comparison between a sleep–wake rhythm intervention and cognitive therapy can therefore clarify whether addressing sleep timing itself provides meaningful symptomatic benefit, whether improvements in sleep regularity are associated with reductions in anxiety and depression, and whether rhythm stabilization may serve as a feasible primary or complementary therapeutic strategy in this population (9).

Using a PICO framework, the present randomized controlled trial enrolled adolescents aged 14–18 years with clinically significant anxiety and depressive symptoms and persistent sleep–wake irregularity. The intervention was targeted sleep–wake rhythm stabilization, the comparator was cognitive therapy, and the primary clinical outcomes were changes in anxiety and depressive symptom severity measured using validated symptom scales. The study was designed to test the hypothesis that targeted stabilization of sleep–wake rhythms would result in greater reductions in co-occurring anxiety and depressive symptoms than cognitive therapy over an eight-week intervention period, and that improvement in sleep regularity would be associated with greater symptom reduction (10).

MATERIALS AND METHODS

This study was conducted as a parallel-group randomized controlled trial designed to compare the effectiveness of targeted sleep–wake rhythm stabilization with cognitive therapy in adolescents presenting with co-occurring anxiety and depressive symptoms and persistent sleep–wake irregularity. The trial was carried out over an eight-week intervention period in the Islamabad–Rawalpindi metropolitan region, with recruitment from secondary schools, colleges, and adolescent psychiatry outpatient clinics. The setting was selected because adolescents in this region commonly experience academic pressure, variable school schedules, high evening technology exposure, and irregular daily routines, all of which are relevant to sleep–wake disruption and emotional symptoms. Participants were recruited through school counselors, clinical referrals, and informational sessions conducted for eligible institutions and outpatient services.

Adolescents aged 14–18 years were eligible for inclusion if they had clinically significant symptoms of both anxiety and depression, reported irregular sleep–wake timing for at least three months, were currently enrolled in school or college, and were able to complete daily sleep monitoring during the study period. Clinically significant anxiety and depressive symptoms were assessed using standardized screening instruments before enrollment. Sleep–wake irregularity was defined as persistent variability in bedtime, wake time, or weekday–weekend sleep timing sufficient to interfere with regular daily functioning. Adolescents were excluded if they had a current psychotic disorder, bipolar disorder, neurodevelopmental disorder, substance use disorder, current use of sleep-altering medication, or participation in structured psychotherapy during the preceding three months. These exclusion criteria were applied to reduce clinical heterogeneity and minimize the influence of conditions or treatments that could independently affect sleep patterns or emotional symptoms.

After screening for eligibility, written informed consent was obtained from parents or legal guardians, and assent was obtained from participating adolescents. Eligible participants were randomly assigned in a 1:1 ratio to either the targeted sleep–wake rhythm stabilization group or the cognitive therapy group. Randomization was performed using a computer-generated allocation sequence prepared by an independent researcher who was not involved in intervention delivery or outcome assessment. Sixty participants were randomized, with 30 allocated to each intervention arm. During the intervention period, two participants from the sleep–wake rhythm stabilization group and one participant from the cognitive therapy group withdrew because of scheduling conflicts, resulting in 57 participants completing post-intervention assessment.

The targeted sleep–wake rhythm stabilization intervention was delivered weekly for eight weeks by trained mental health professionals. The intervention focused on establishing a fixed wake time, reducing bedtime variability, regulating evening screen exposure, encouraging consistent morning light exposure, and structuring daytime activity patterns to strengthen circadian stability. Participants were guided to maintain a consistent sleep–wake schedule across weekdays and weekends, reduce behaviors that delayed sleep onset, and develop regular daily routines supporting sleep timing consistency. Daily sleep diaries were used to monitor adherence to sleep timing targets, and wrist-worn actigraphy was used to provide objective estimates of sleep–wake regularity. Intervention sessions reviewed diary and

actigraphy patterns, identified barriers to rhythm consistency, and reinforced individualized behavioral strategies for maintaining stable sleep and wake timing.

Participants assigned to the cognitive therapy group received weekly individual cognitive-based sessions over the same eight-week period. Sessions focused on identifying anxiety- and mood-related maladaptive thoughts, cognitive restructuring, behavioral activation, coping skills, and strategies for reducing avoidance and improving emotional regulation. The cognitive therapy arm served as an active psychological comparator and was delivered by trained mental health professionals using a structured therapeutic approach. Both groups received equal intervention duration and weekly contact frequency to reduce differences in therapist exposure and participant attention between trial arms.

The primary outcomes were changes in anxiety and depressive symptom severity from baseline to the end of the eight-week intervention. Anxiety symptoms were measured using the Generalized Anxiety Disorder-7 scale, and depressive symptoms were measured using the Patient Health Questionnaire-9. Both instruments were administered at baseline and post-intervention using standardized scoring procedures. Sleep timing regularity was assessed using daily sleep diaries supported by wrist-worn actigraphy, with sleep irregularity operationalized as variability in sleep–wake timing across the monitoring period. Daytime functioning was assessed using the Pediatric Daytime Sleepiness Scale as a secondary measure. Clinically significant improvement was defined as at least a 50% reduction in both anxiety and depressive symptom scores from baseline to post-intervention.

Data collection was performed at baseline before randomization and repeated after completion of the eight-week intervention. Baseline data included age, sex, school type, anxiety severity, depressive symptom severity, and sleep irregularity. Participants maintained daily sleep diaries throughout the intervention period, recording bedtime, wake time, perceived sleep quality, screen exposure patterns, and adherence to prescribed sleep–wake schedules. Actigraphy data were used to supplement self-reported sleep information and improve the reliability of sleep timing estimates. Outcome data were checked for completeness and consistency before analysis, and participant records were coded to maintain confidentiality.

Bias and confounding were addressed through randomized group allocation, use of standardized symptom measures, equivalent intervention duration across groups, objective sleep monitoring with actigraphy, and predefined eligibility criteria. Restricting enrollment to adolescents with both emotional symptoms and persistent sleep–wake irregularity helped ensure that the enrolled population was clinically aligned with the intervention target. Exclusion of participants using sleep-altering medication or currently receiving structured psychotherapy reduced the likelihood that observed changes were attributable to concurrent treatments. Baseline demographic and clinical characteristics were compared between groups to evaluate comparability after randomization.

The planned sample size was 60 participants, selected to provide adequate power to detect moderate between-group differences in symptom reduction while remaining feasible within the recruitment period and intervention duration. Allowance was made for limited attrition during follow-up, and the final analysis included 57 participants who completed post-intervention assessment. Data were analyzed using statistical software after assessment of distributional assumptions. Continuous variables were summarized as means and standard deviations, while categorical variables were summarized as frequencies and percentages. Normality of continuous outcomes was evaluated using the Shapiro–Wilk test. Within-group changes from baseline to post-intervention were analyzed using paired-sample t-tests, and between-group differences were assessed using independent-sample t-tests. Repeated-measures analysis of variance was used to examine group-by-time differences in symptom trajectories. Pearson correlation analysis was used to evaluate associations between improvement in sleep regularity and reductions in anxiety and depressive symptoms. Categorical response rates were compared between groups using appropriate tests for proportions. Statistical significance was set at a two-tailed p-value of less than 0.05.

Missing data were minimized through weekly participant contact, review of diary completion, and follow-up reminders during the intervention period. Participants who withdrew before post-intervention assessment were not included in the final completer analysis. Data integrity was maintained through standardized data collection forms, consistent scoring procedures for psychometric instruments, cross-checking of diary and actigraphy records, and coded participant identifiers. All participant information was handled confidentially, and consent and assent procedures were completed before enrollment.

RESULTS

A total of 78 adolescents were screened for eligibility, of whom 60 met the inclusion criteria and were randomized equally into the targeted sleep–wake rhythm stabilization group and the cognitive therapy group. During the eight-week intervention period, two participants in the sleep–wake group and one participant in the cognitive therapy group withdrew because of scheduling conflicts. The final completer sample included 57 adolescents, with 28 participants in the sleep–wake rhythm stabilization group and 29 participants in the cognitive therapy group, giving an overall retention rate of 95.0%. The mean age of participants was 16.2 ± 1.3 years. Females represented 56.1% of the sample, and 61.4% of participants were enrolled in public schools. Baseline clinical severity was comparable between groups, with mean GAD-7 scores of 14.0 ± 3.2 in the sleep–wake group and 13.6 ± 3.0 in the cognitive therapy group, and mean PHQ-9 scores of 14.6 ± 2.9 and 14.4 ± 2.7, respectively.

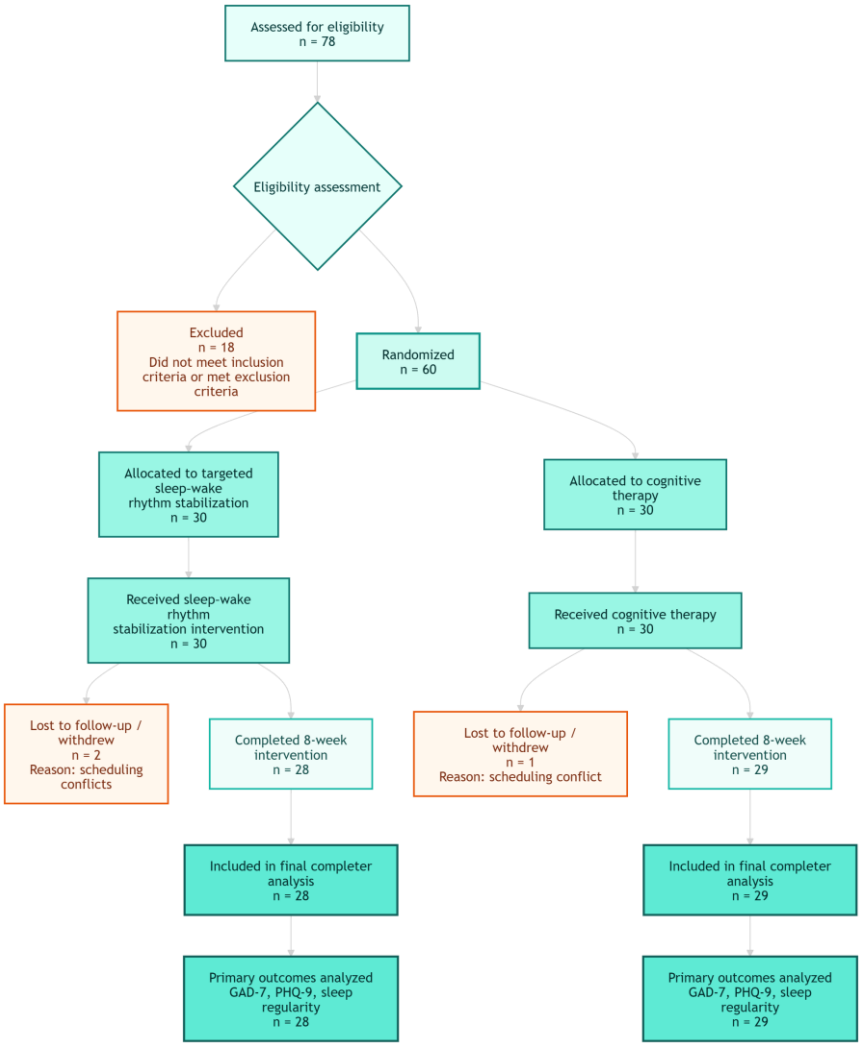

Figure 1. CONSORT Flow Diagram of Participant Screening, Randomization, Follow-Up, and Final Analysis

Figure caption: CONSORT flow diagram showing participant screening, randomization, allocation, follow-up, and final completer analysis. Of 78 adolescents screened, 60 were randomized equally to

targeted sleep–wake rhythm stabilization or cognitive therapy. After three withdrawals due to scheduling conflicts, 57 participants completed the eight-week intervention and were included in the final analysis.

Table 1. Baseline Demographic and Clinical Characteristics of Participants

Variable	Total Sample (N=57)	Sleep–Wake Rhythm Stabilization (n=28)	Cognitive Therapy (n=29)	Between-Group Difference / Effect Estimate	p-value
Age, years, mean ± SD	16.2 ± 1.3	16.1 ± 1.2	16.3 ± 1.4	Mean difference: -0.20 years; 95% CI: -0.89 to 0.49	0.56
Female sex, n (%)	32 (56.1)	16 (57.1)	16 (55.2)	OR: 1.08	0.88
Male sex, n (%)	25 (43.9)	12 (42.9)	13 (44.8)		
Public school, n (%)	35 (61.4)	17 (60.7)	18 (62.1)	OR: 0.94	0.92
Private school, n (%)	22 (38.6)	11 (39.3)	11 (37.9)		
Baseline GAD-7 score, mean ± SD	13.8 ± 3.1	14.0 ± 3.2	13.6 ± 3.0	Mean difference: 0.40; 95% CI: -1.25 to 2.05	0.63
Baseline PHQ-9 score, mean ± SD	14.5 ± 2.8	14.6 ± 2.9	14.4 ± 2.7	Mean difference: 0.20; 95% CI: -1.29 to 1.69	0.79
Baseline sleep irregularity, hours, mean ± SD	2.6 ± 0.9	2.5 ± 0.8	2.7 ± 1.0	Mean difference: -0.20 hours; 95% CI: -0.68 to 0.28	0.41

At baseline, the two intervention groups were similar across demographic, educational, clinical, and sleep-related characteristics. The mean age differed by only 0.20 years between groups, and the 95% confidence interval crossed zero. Baseline anxiety and depressive symptom scores were also closely aligned, with between-group differences of 0.40 points for GAD-7 and 0.20 points for PHQ-9. Baseline sleep irregularity was slightly lower in the sleep–wake rhythm stabilization group than in the cognitive therapy group, but the difference was small and not statistically significant.

After eight weeks, both groups showed reductions in anxiety and depressive symptoms, but improvements were larger in the sleep–wake rhythm stabilization group. The mean GAD-7 score declined from 14.0 ± 3.2 to 7.8 ± 2.4 in the sleep–wake group, corresponding to a mean reduction of 6.2 ± 2.1 points. In the cognitive therapy group, the GAD-7 score declined from 13.6 ± 3.0 to 9.8 ± 2.5, corresponding to a mean reduction of 3.8 ± 1.9 points. The between-group difference in anxiety symptom reduction was 2.40 points, with a 95% CI of 1.34 to 3.46 and a large standardized effect size.

Table 2. Changes in Anxiety and Depressive Symptoms From Baseline to Post-Intervention

Outcome	Sleep–Wake Rhythm Stabilization (n=28), Mean ± SD	Cognitive Therapy (n=29), Mean ± SD	Between-Group Difference	95% CI	Effect Size, Cohen's d	p-value
GAD-7 baseline score	14.0 ± 3.2	13.6 ± 3.0	0.40	-1.25 to 2.05	0.13	0.63
GAD-7 post-intervention score	7.8 ± 2.4	9.8 ± 2.5	-2.00	-3.30 to -0.70	-0.82	0.003
GAD-7 reduction	6.2 ± 2.1	3.8 ± 1.9	2.40	1.34 to 3.46	1.20	<0.001
PHQ-9 baseline score	14.6 ± 2.9	14.4 ± 2.7	0.20	-1.29 to 1.69	0.07	0.79
PHQ-9 post-intervention score	8.7 ± 2.5	10.9 ± 2.6	-2.20	-3.55 to -0.85	-0.86	0.002
PHQ-9 reduction	5.9 ± 2.3	3.5 ± 2.0	2.40	1.25 to 3.55	1.11	<0.001

Depressive symptoms followed a similar pattern. The sleep–wake rhythm stabilization group showed a mean PHQ-9 reduction of 5.9 ± 2.3 points, compared with 3.5 ± 2.0 points in the cognitive therapy group. The between-group difference in PHQ-9 reduction was 2.40 points, with a 95% CI of 1.25 to 3.55 and a large effect size. Post-intervention mean scores remained lower in the sleep–wake rhythm stabilization group for both GAD-7 and PHQ-9, indicating greater symptom improvement over the eight-week intervention period.

Sleep timing regularity improved more substantially in the sleep–wake rhythm stabilization group. Mean sleep irregularity decreased by 1.8 ± 0.6 hours in the sleep–wake group, compared with 0.6 ± 0.5 hours in the cognitive therapy group. The between-group difference in sleep-regularity improvement

was 1.20 hours, with a 95% CI of 0.91 to 1.49 and a large standardized effect size. Improvements in sleep regularity were also associated with reductions in both anxiety and depressive symptoms, with correlation coefficients of -0.62 for GAD-7 reduction and -0.59 for PHQ-9 reduction.

Table 3. Sleep-Regularity Improvement and Association With Symptom Reduction

Variable	Sleep–Wake Rhythm Stabilization (n=28), Mean ± SD	Cognitive Therapy (n=29), Mean ± SD	Between-Group Difference	95% CI	Effect Size, Cohen's d	p-value
Baseline sleep irregularity, hours	2.5 ± 0.8	2.7 ± 1.0	-0.20	-0.68 to 0.28	-0.22	0.41
Reduction in sleep irregularity, hours	1.8 ± 0.6	0.6 ± 0.5	1.20	0.91 to 1.49	2.18	<0.001
Correlation between sleep-regularity improvement and GAD-7 reduction	r = -0.62					<0.001
Correlation between sleep-regularity improvement and PHQ-9 reduction	r = -0.59					<0.001

Clinically significant improvement was achieved more frequently in the sleep–wake rhythm stabilization group. Sixteen of 28 participants in the sleep–wake group achieved at least a 50% reduction in both anxiety and depressive symptom scores, compared with 8 of 29 participants in the cognitive therapy group.

This corresponded to response rates of 57.1% and 27.6%, respectively. The absolute risk difference was 29.6 percentage points, and participants receiving sleep–wake rhythm stabilization were approximately twice as likely to achieve clinically significant improvement as those receiving cognitive therapy.

Table 4. Clinically Significant Improvement After Eight Weeks

Outcome	Sleep–Wake Rhythm Stabilization (n=28)	Cognitive Therapy (n=29)	Effect Estimate	95% CI	p-value
≥50% reduction in both GAD-7 and PHQ-9, n (%)	16 (57.1)	8 (27.6)	Risk difference: 29.6 percentage points	5.0 to 54.1	0.024
Relative likelihood of clinically significant improvement			Relative risk: 2.07	1.06 to 4.05	0.033
Odds of clinically significant improvement			Odds ratio: 3.50	1.16 to 10.58	0.033

Overall, the results showed greater improvement in anxiety symptoms, depressive symptoms, and sleep timing regularity among adolescents receiving targeted sleep–wake rhythm stabilization compared with cognitive therapy.

The largest between-group effect was observed for sleep irregularity reduction, followed by anxiety and depressive symptom reductions. The pattern of findings indicates that improvement in sleep–wake regularity was closely linked with clinical symptom improvement over the eight-week intervention period.

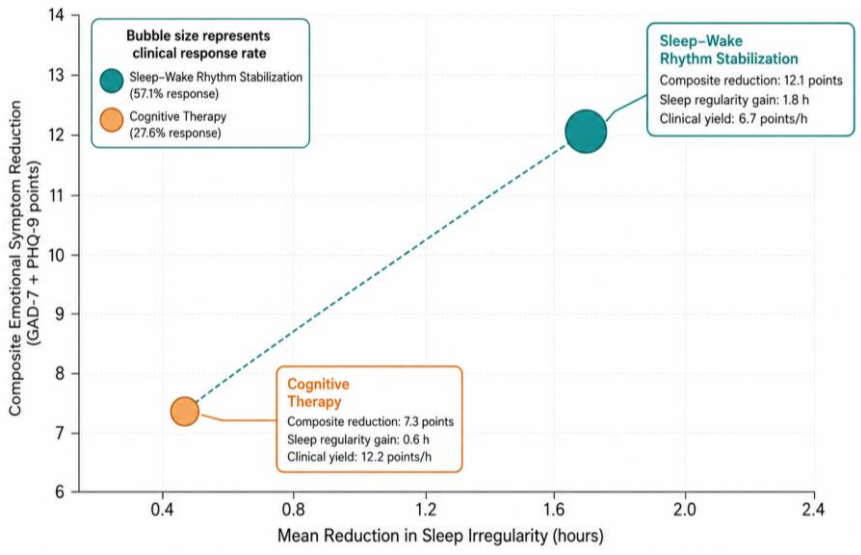

Figure 2. Clinical Symptom Gain Across Sleep–Wake Regularity Improvement

The sleep–wake rhythm stabilization group demonstrated a larger integrated clinical gain, with a 1.8-hour mean reduction in sleep irregularity accompanied by a 12.1-point composite reduction across GAD-7 and PHQ-9 scores and a 57.1% dual-scale clinical response rate. In comparison, cognitive therapy produced a 0.6-hour improvement in sleep regularity, a 7.3-point composite symptom reduction, and a 27.6% response rate. Although the cognitive therapy group showed a higher symptom-reduction yield per hour of sleep-regularity improvement, the sleep–wake rhythm stabilization group achieved a substantially greater absolute improvement across sleep regularity, emotional symptom burden, and clinically significant response, supporting the clinical relevance of rhythm stabilization as a targeted intervention pathway.

DISCUSSION

The present trial demonstrated that targeted stabilization of sleep–wake rhythms produced greater improvement in co-occurring anxiety and depressive symptoms among adolescents than cognitive therapy over an eight-week intervention period. Adolescents receiving the sleep–wake rhythm stabilization intervention showed larger reductions in both GAD-7 and PHQ-9 scores, indicating that regulation of daily sleep timing may have meaningful clinical relevance for emotional symptom reduction in this population. The magnitude of improvement was particularly notable because participants presented with both anxiety and depressive symptoms alongside persistent sleep–wake irregularity, a clinical profile in which emotional distress and circadian disruption may reinforce each other through fatigue, hyperarousal, impaired concentration, reduced daytime functioning, and inconsistent behavioral routines (11). These findings support the view that sleep–wake regularity is not merely a secondary lifestyle factor but may represent an important therapeutic target in adolescents with internalizing symptoms.

The greater reduction in anxiety symptoms observed in the sleep–wake rhythm stabilization group suggests that consistent wake times, reduced bedtime variability, structured daytime activity, and improved regulation of evening routines may reduce physiological and cognitive arousal. Anxiety in adolescents is often sustained by anticipatory worry, poor sleep onset regulation, irregular daily structure, and heightened sensitivity to stress. Stabilizing sleep–wake timing may improve predictability in daily routines and strengthen circadian cues, thereby reducing the emotional volatility that can accompany rhythm instability. The observed reduction in GAD-7 scores is consistent with evidence that sleep and circadian disturbances are closely linked with anxiety severity, stress reactivity, and impaired emotional control during adolescence (12). By targeting the biological and behavioral conditions that

influence arousal regulation, sleep–wake rhythm stabilization may enhance the adolescent's capacity to manage anxiety symptoms more effectively.

Depressive symptoms also improved more substantially in the sleep–wake rhythm stabilization group than in the cognitive therapy group. This finding is clinically important because depressive symptoms in adolescents are frequently associated with delayed sleep timing, irregular routines, low daytime activation, and reduced exposure to morning light. The intervention directly addressed these mechanisms by encouraging consistent sleep and wake schedules, daytime activity structuring, and regulation of behaviors that disrupt circadian alignment. Improvement in PHQ-9 scores may therefore reflect both direct biological effects of rhythm stabilization and indirect behavioral benefits, including greater daytime alertness, improved routine consistency, and increased engagement in daily activities. These results align with the broader concept that sleep–circadian regulation plays a central role in mood stability and that interventions targeting rhythm regularity may reduce depressive symptom burden in youth (13).

A key finding of the study was the strong association between improvement in sleep regularity and reduction in emotional symptoms. Participants who achieved greater reductions in sleep irregularity tended to show larger decreases in anxiety and depressive symptom scores, suggesting that stabilization of sleep timing may be closely connected with clinical improvement. This relationship strengthens the mechanistic plausibility of the intervention, as it indicates that symptom reduction was not isolated from the intended treatment target. However, the association should be interpreted within the context of the trial design, as improvement in sleep regularity may operate alongside other behavioral changes, including better daytime structure, reduced screen exposure, improved family routine support, and increased adherence to health-promoting behaviors. Even so, the correlation between rhythm stabilization and symptom improvement supports the clinical value of assessing sleep regularity when treating adolescents with anxiety and depression (14,15).

The higher proportion of clinically significant improvement in the sleep–wake rhythm stabilization group further supports the practical relevance of the intervention. More than half of participants in this group achieved at least a 50% reduction in both anxiety and depressive symptom scores, compared with just over one-quarter of participants receiving cognitive therapy. This difference suggests that a sleep-focused intervention may be particularly beneficial for adolescents whose emotional symptoms are accompanied by persistent sleep–wake irregularity. While cognitive therapy remains an established and valuable approach for adolescent internalizing symptoms, its effectiveness may be reduced when fatigue, poor sleep timing, low motivation, and biological rhythm disruption interfere with attention, emotional processing, and therapeutic engagement. A rhythm-focused approach may therefore be useful either as a primary intervention for carefully selected adolescents or as an early adjunct that improves readiness for psychological therapy (16,17).

The findings also have implications for treatment personalization. Adolescents with anxiety and depression are a heterogeneous group, and not all patients may benefit equally from the same intervention model. For those with prominent cognitive distortions, avoidance patterns, or interpersonal stressors, cognitive therapy may remain central. For adolescents whose symptoms are accompanied by irregular sleep timing, delayed routines, and daytime sleepiness, targeting sleep–wake rhythm stability may address a maintaining factor that standard cognitive approaches do not fully correct. This supports a mechanism-informed model of care in which intervention selection is guided by the dominant clinical drivers of distress. Incorporating sleep regularity assessment into routine adolescent mental health screening may help clinicians identify individuals likely to benefit from circadian-focused behavioral strategies (18).

The results extend prior work by evaluating sleep–wake rhythm stabilization as an active intervention rather than only as an adjunctive lifestyle recommendation. Many sleep-focused approaches in adolescent mental health care are framed as supportive or secondary to psychological therapy. In

contrast, the present findings suggest that structured rhythm stabilization can produce clinically meaningful reductions in emotional symptoms over a relatively brief treatment period. This does not diminish the importance of cognitive therapy; rather, it highlights that biological and behavioral rhythm regulation may be an important parallel pathway for improving adolescent mental health outcomes. A combined treatment model may be particularly valuable, with rhythm stabilization used to reduce fatigue and improve daily functioning while cognitive therapy targets maladaptive thought patterns, avoidance, and coping skills (19,20).

Several strengths enhance the clinical interpretability of the findings. The randomized parallel-group design allowed direct comparison between two active interventions. The use of validated symptom scales provided standardized measurement of anxiety and depressive severity, while the combination of daily sleep diaries and actigraphy strengthened assessment of sleep–wake regularity. The eight-week intervention period was clinically practical and demonstrated that measurable improvement can occur within a relatively short timeframe. The retention rate was high, suggesting that both interventions were acceptable to participants and feasible within adolescent school and clinical contexts. Recruitment from schools, colleges, and outpatient clinics also increased ecological relevance by including adolescents exposed to typical academic and social demands.

Despite these strengths, several limitations should be considered when interpreting the findings. The sample size was modest, which limits precision of effect estimates and reduces the ability to examine subgroup differences by sex, age, baseline severity, school type, or degree of sleep irregularity. The trial was conducted in a single urban region, and therefore the findings may not fully generalize to adolescents from rural settings, different socioeconomic backgrounds, or health systems with different mental health service structures. The intervention period was limited to eight weeks, and no long-term follow-up was included, leaving the durability of symptom improvement uncertain. In addition, although actigraphy and diaries improved sleep assessment, adherence to behavioral recommendations may still have varied across participants and could have influenced treatment response.

Another important consideration is that the study compared sleep–wake rhythm stabilization with cognitive therapy but did not include a combined intervention arm or a usual-care control group. Therefore, the findings clarify relative short-term benefit between two active approaches but do not determine whether combining rhythm stabilization with cognitive therapy would produce additive or synergistic effects. Because anxiety and depression in adolescence are shaped by biological, cognitive, interpersonal, family, and environmental influences, a multimodal intervention may ultimately provide the greatest benefit for many patients. Future trials should compare sleep-focused, cognitive, combined, and usual-care conditions to determine which adolescents respond best to each treatment pathway.

Future research should also evaluate longer follow-up periods to determine whether improvements in sleep regularity and emotional symptoms are maintained after active intervention ends. Larger multi-site randomized trials would allow more precise estimation of treatment effects and permit subgroup analyses based on baseline sleep irregularity, chronotype, symptom severity, school schedule, screen exposure, and family routine stability. Further work should examine whether changes in sleep regularity mediate symptom improvement and whether objective circadian markers, light exposure patterns, or daytime activity rhythms explain differential response. Digital sleep-monitoring tools and mobile behavioral supports may also improve adherence, scalability, and real-time reinforcement of rhythm-stabilizing behaviors in adolescent populations (21,22).

In summary, targeted sleep–wake rhythm stabilization produced greater short-term reductions in anxiety and depressive symptoms than cognitive therapy among adolescents with comorbid emotional symptoms and irregular sleep patterns. The strong association between improved sleep regularity and symptom reduction suggests that circadian and behavioral rhythm stability may be an important mechanism of clinical recovery. These findings support integrating sleep–wake assessment into

adolescent mental health care and considering rhythm stabilization as a structured therapeutic option for adolescents in whom irregular sleep timing contributes to emotional distress.

CONCLUSION

Targeted sleep–wake rhythm stabilization produced greater reductions in anxiety and depressive symptoms than cognitive therapy among adolescents with co-occurring emotional symptoms and persistent sleep–wake irregularity over eight weeks. Improvement in sleep regularity was closely associated with reductions in both GAD-7 and PHQ-9 scores, supporting the clinical importance of circadian and behavioral rhythm stability in adolescent emotional regulation. These findings suggest that structured interventions targeting consistent wake times, bedtime regularity, light exposure, screen-use regulation, and daytime activity scheduling may offer a practical and clinically meaningful approach for adolescents whose anxiety and depressive symptoms are accompanied by irregular sleep patterns. While cognitive therapy remains an important evidence-based treatment, sleep–wake rhythm stabilization may serve as a valuable targeted intervention or complementary therapeutic pathway within adolescent mental health care.

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