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The Association Between Sleep Circadian Rhythm and Adolescent Depression

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Abstract. Adolescent depression is a major global public health concern, and circadian rhythm research provides a new perspective for understanding adolescent depression vulnerability. The adolescent circadian system undergoes natural developmental remodeling characterized by physiological phase delay, which conflicts with rigid social temporal constraints, leading to widespread social jet lag among adolescents. This paper systematically elucidates the bidirectional coupling mechanism between circadian disruption and adolescent depression, summarizes the applicability of chronotherapeutic interventions, and explores the structural social drivers of rhythm misalignment, and finds that longitudinal studies confirm the bidirectional coupling between circadian disruption and adolescent depression, validated through long-term dynamic observations, and the degree of circadian disruption progressively increases with the recurrence of depressive episodes. Neuroimaging evidence further reveals the neural basis of this interaction. Integrated chronotherapy with cognitive behavioral therapy can effectively restore circadian homeostasis and alleviate depressive symptoms. This paper systematically examines the interaction between adolescent circadian development and social structural constraints, providing new theoretical perspectives and practical pathways for constructing adolescent mental health promotion systems.

Keywords: circadian rhythm, adolescent depression, circadian clock genes, social jet lag, emotional regulation

1. Introduction

Adolescent depression has become a significant issue in the field of global public health. A global epidemiological systematic review conducted in 2025 (encompassing 37 high-quality studies with a total sample size of 1,590,000) revealed that the overall prevalence of mental disorders among adolescents aged 10-19 worldwide was 14.53%, of which major depressive disorder (MDD) accounted for over 40%. Over the past decade, there has been a dual trend of increasing incidence rates and decreasing age of onset [1].

Existing research predominantly explores the influencing factors of adolescent depression from perspectives such as genetics, family dynamics, and academic stress, but three critical gaps remain. Most studies focus on static correlations between risk factors and depressive symptoms, neglecting the dynamic regulatory mechanisms underlying disease progression. Compounding this limitation,

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intervention research tends to prioritize individual-level clinical strategies, without adequately addressing the social structural constraints that shape adolescent health trajectories. Further, scholarship is largely confined to single-discipline frameworks, lacking interdisciplinary integration of physiological developmental characteristics and social structural contexts—particularly lacking a systematic examination of the association between physiological traits across different developmental periods and psychological pathology.

As a core endogenous physiological trait, circadian rhythm provides a novel analytical framework to address the research gap identified above. The misalignment between adolescent developmental phase delay and rigid social time schedules impairs emotion regulation via neuroendocrine and cognitive pathways, constituting a key mechanistic basis for depression vulnerability. This paper systematically elucidates the bidirectional coupling mechanism between circadian disruption and adolescent depression, summarizes the applicability of chronotherapeutic interventions, and explores the structural social drivers of rhythm misalignment, to provide empirical insights for adolescent mental health promotion.

2. Developmental characteristics of the circadian rhythm during adolescence

2.1. The intrinsic mechanisms and core regulatory system of the circadian rhythm

The circadian rhythm is an endogenous ~24-hour physiological and behavioral rhythm evolved to adapt to Earth's rotation, with its regulatory core located in the hypothalamic suprachiasmatic nucleus (SCN). This region acts as the central biological clock, integrating external time cues (e.g., light exposure) and coordinating rhythmic physiological processes including the sleep-wake cycle, hormone secretion, and energy metabolism [2]. At the molecular level, circadian rhythm is driven by a conserved transcription-translation negative feedback loop of clock genes: CLOCK and BMAL1 proteins form a heterodimer that activates the transcription of downstream PER and CRY genes, the translated products enter the nucleus to inhibit their own transcription, generating ~24-hour molecular oscillations [3].

Melatonin and cortisol are key endocrine markers reflecting circadian rhythm status: melatonin is synthesized by the pineal gland in dark environments and serves as a crucial signaling molecule for sleep initiation, cortisol levels rise rapidly before morning awakening to form the "morning peak," playing a role in the arousal process and stress adaptation, the rhythmic stability of these two hormones is directly correlated with adolescent emotional regulation abilities, while abnormal phase variation or reduced signal amplitude of these endocrine markers often accompany depressive symptom onset [4].

2.2. Developmental transition characteristics of the circadian rhythm during adolescence

Adolescence is a critical period of circadian system remodeling, characterized by a significant delay in dim light melatonin onset (DLMO). Longitudinal studies show DLMO shifts from ~20:00 in childhood to ~23:00 in late adolescence, accompanied by slower sleep homeostatic pressure accumulation, forming the typical adolescent sleep phase delay phenotype [5]. The developmental temporal sequence and key maturation nodes of this transition are visualized in Figure 1.

This endogenous phase delay misaligns with fixed early school schedules, creating "social time discrepancy" (social jet lag) whose prevalence increases with age [6]. Cumulative social jet lag directly causes chronic sleep deprivation, and disrupts emotional regulation via three pathways:

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autonomic nervous system imbalance, prefrontal executive function impairment, and hypothalamic-pituitary-adrenal axis hyperresponsiveness.

Academic stress and nighttime electronic screen exposure further amplify this susceptibility: blue light from light-emitting devices inhibits already delayed melatonin secretion, worsening sleep onset difficulty [7]. Adolescent sleep distress is thus not merely a behavioral self-discipline issue, but a product of interaction between developmental biological traits and social temporal constraints, forming the contextual basis for understanding circadian dysregulation and adolescent depression comorbidity.

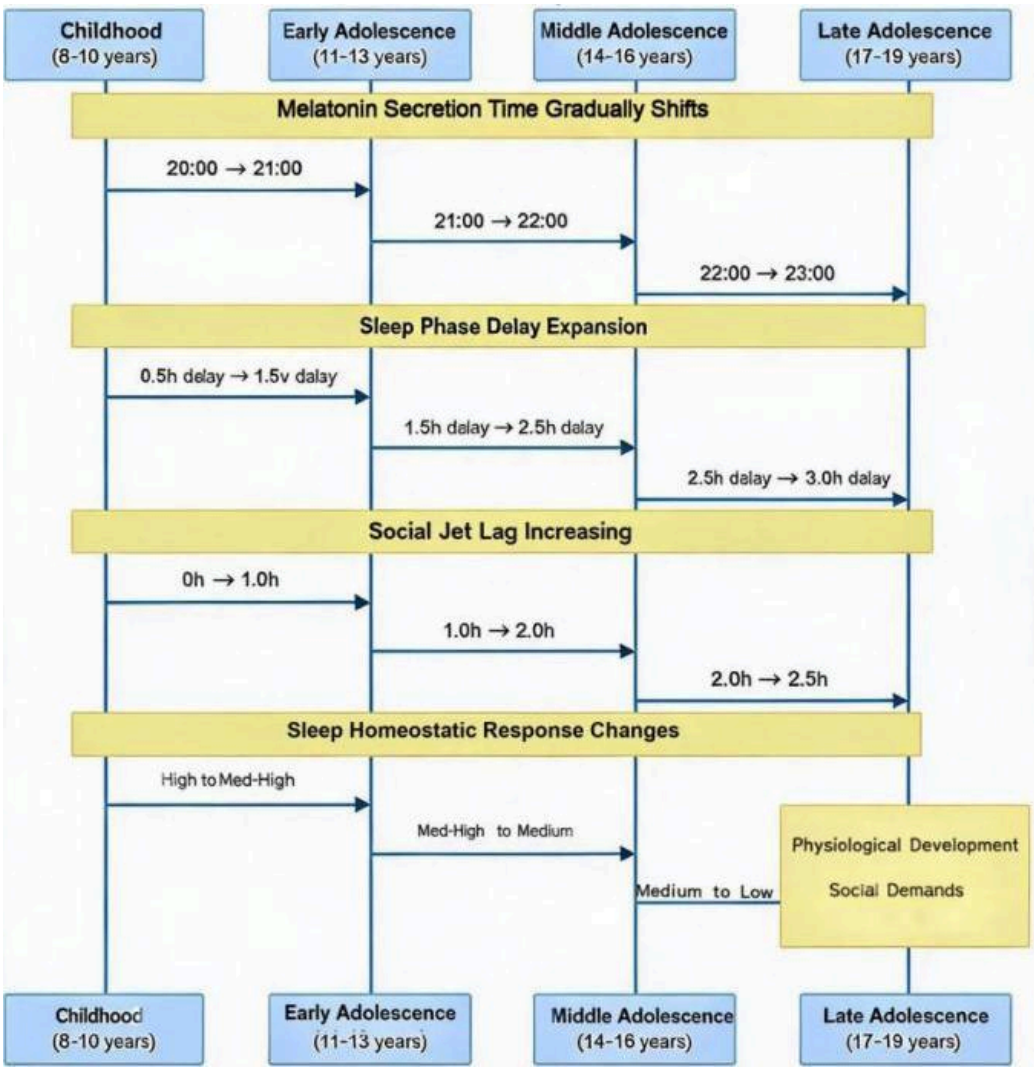

Figure 1. Temporal sequence of circadian rhythm transition during adolescence

3. The bidirectional coupling mechanism between circadian rhythm disruption and adolescent depression

3.1. The bidirectional relationship between circadian rhythm types and depressive symptoms

Circadian rhythm regulates core physiological functions, including sleep-wake cycles, hormonal balance, and emotional processing. The conflict between adolescent late-type circadian preference and social temporal constraints renders this population highly susceptible to depression [8].

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Epidemiological evidence confirms that late-type rhythm is not only associated with reduced sleep duration, poor sleep quality, and increased social jet lag, but also significantly elevates depressive symptom risk. Prolonged social jet lag, a key marker of circadian misalignment, impairs emotional regulation homeostasis by reducing heart rate variability during REM sleep [8].

Circadian disruption and depression follow a dynamic bidirectional coupling pattern rather than a unidirectional causal relationship. Cross-lagged longitudinal studies show that baseline late-type rhythm predicts 6-month worsening of depressive symptoms, while initial depression severity also predicts subsequent circadian disruption, forming a mutually reinforcing vicious cycle [9]. Emotional regulation strategies play a key mediating role: circadian dysrhythmia reduces cognitive reappraisal capacity, triggering increased rumination and depressive symptoms, conversely, rumination further disrupts sleep maintenance, exacerbating rhythm desynchronization [9].

Neuroimaging studies have elucidated the biological basis of this relationship: late-type rhythm in adolescents with mood disorders predicts white matter integrity impairment in emotional regulation pathways (e.g., anterior corona radiata, corpus callosum genu), forming the neural structural foundation for comorbid circadian disruption and depression [10].

Circadian disruption is both a depression biomarker and a dynamic regulator across the disorder course. Compared to first-onset adolescent depression patients, those with recurrent depression show more pronounced multidimensional rhythm decoupling, as visualized in Figure 2. A Korean clinical study found no significant difference in total depression scale scores between the two groups, but the recurrent group exhibited systematically higher circadian disruption scores across sleep (3.75 vs. 3.02), activity (4.03 vs. 3.89), social interaction (4.86 vs. 3.93), and diet (3.75 vs. 3.00) dimensions, indicating more extensive daily behavioral rhythm stability impairment [11].

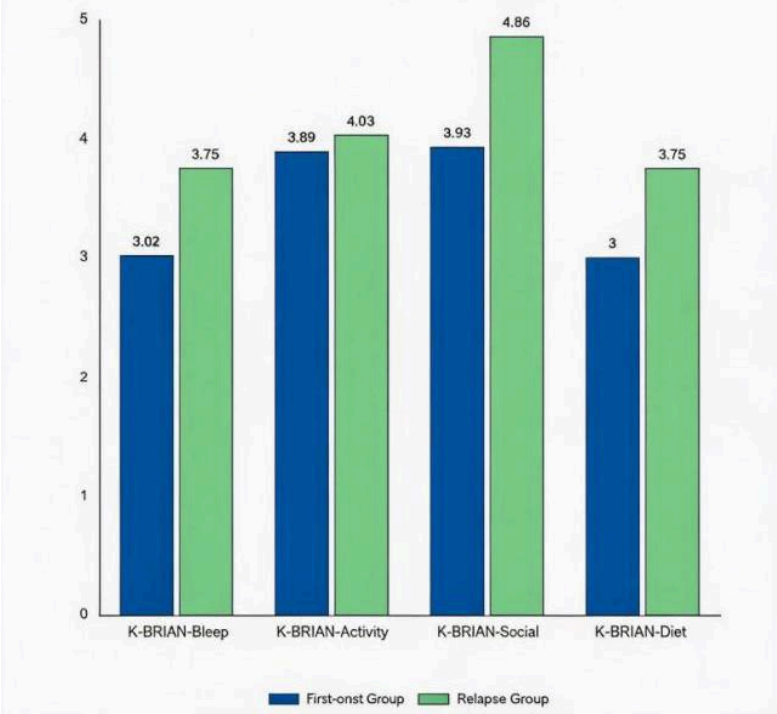

Figure 2. Comparison of circadian rhythm disruption between the initial and recurrent groups

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3.2. The dynamic role of circadian rhythm disruption in the progression of major depressive disorder (MDD)

Longitudinal follow-up studies confirm that circadian disruption severity progressively increases with depressive episode recurrence, validating its dynamic regulatory role in disease progression [12].

This exacerbation arises from the interaction between neural plasticity remodeling under prolonged depressive states and social functioning deterioration. Specifically, chronic circadian disruption reduces oligodendrocyte proliferation and differentiation, impairing prefrontal-limbic pathway myelin integrity, which lowers emotional regulation circuit information transmission efficiency and worsens depressive symptoms [10].

Consistently, objective indicators, including social jet lag and sleep midpoint shift, are positively correlated with depressive episode frequency, indicating that circadian misalignment degree directly reflects cumulative disease burden [12].

Incorporating circadian parameters into adolescent depression clinical assessment systems thus facilitates high-risk individual identification, provides personalized chronotherapy targets, and establishes a theoretical foundation for non-pharmacological intervention development.

Recent wearable sensing technology advances enable continuous monitoring of core circadian parameters: rest-activity amplitude, sleep-wake regularity, light exposure patterns, and heart rate variability diurnal fluctuations. A 2024 cohort study showed an XGBoost model integrating these parameters achieved 0.82 AUC for predicting 12-month adolescent depression onset, outperforming traditional models [13]. This framework supports early at-risk adolescent identification and dynamic intervention strategy adjustment, representing a promising precision mental health practice direction.

4. Clinical intervention pathways and their social implications

4.1. Clinical practice of time-based therapy and rhythm reshaping

Building upon the bidirectional coupling mechanism between circadian disruption and adolescent depression validated through epidemiological, neuroimaging and longitudinal evidence in Section 3, chronotherapeutic interventions targeting the "rhythm–sleep–emotion" network have emerged as core non-pharmacological strategies [14]. The integrated framework includes three evidence-based techniques: Cognitive Behavioral Therapy for Insomnia (CBT-I) to correct maladaptive sleep behaviors, Bright Light Therapy (BLT) to adjust circadian phase, and Social Rhythm Therapy to stabilize daily activity patterns. Clinical evidence supports differentiated personalized matching: single CBT-I is recommended for primary insomnia with moderate efficacy, single BLT is preferred for delayed sleep phase syndrome with significant phase-shifting effects, CBT-I+ BLT combination shows synergistic effects across all outcomes as the first-line regimen for comorbid cases. Time Therapy can be added as an adjunct for long-term rhythm reshaping, particularly with school schedule adjustments [15]. Notwithstanding the clinical utility of these individual-level interventions, their efficacy is inherently constrained by the structural social determinants of adolescent circadian health, necessitating an expanded analytical lens beyond clinical practice alone.

4.2. Social structural constraints on adolescent circadian health

The circadian rhythm is not merely a physiological regulatory system, it also represents an embodied expression of the relationship between individuals and the social temporal order.

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Adolescents' endogenous phase-delayed biological clocks face two core structural constraints in modern society: first, mandatory early school start times force alignment with rigid social time norms, generating widespread social jet lag, second, pervasive nighttime digital media exposure suppresses melatonin secretion and further delays sleep onset [16]. The combined effect of these institutional and technological pressures constitutes a systematic deprivation of adolescents' circadian autonomy.

Empirical evidence confirms a linear dose–response relationship between circadian misalignment and adolescent depression risk: comparing absolute morning-type to absolute night-type adolescents, depression symptom scores increase from 9.4±1.6 to 24.3±3.1, average sleep duration decreases from 8.3±0.9 hours to 6.2±0.5 hours, and melatonin peak phase shifts from 00:15 to 03:00, demonstrating a clear cumulative stress effect [17]. This gradient association indicates that individual clinical interventions alone cannot fully address the systemic roots of adolescent circadian vulnerability, highlighting the urgent need for multi-level policy adjustments to support adolescent circadian health.

5. Conclusion

Geographic homogeneity of study samples, predominantly recruited from East Asian and Western European contexts, limits the generalizability of findings to low- and middle-income regions with distinct socio-temporal structures. Quantitative mediating pathways linking macro-level time policies to individual circadian disruption and mental health outcomes remain insufficiently characterized, restricting translational potential for evidence-based policy formulation. Future research should adopt multi-modal longitudinal study designs integrating wearable circadian monitoring, neuroimaging assessments and socio-temporal surveys to unpack dynamic causal pathways underlying the circadian rhythm-depression association across adolescent developmental stages. Cross-cultural comparative research exploring variations in social time arrangements is also recommended to address current sampling bias and facilitate the development of context-adapted intervention strategies for global adolescent populations.

Addressing these identified limitations will not only strengthen the generalizability and translational potential of circadian rhythm research in adolescent mental health but also establish a more robust empirical foundation for bridging basic science insights with evidence-based policy and intervention frameworks.

Through an interdisciplinary framework integrating physiological developmental characteristics and social structural constraints, this paper systematically elucidates the bidirectional coupling mechanism between circadian rhythm disruption and adolescent depression, moving beyond the static correlation paradigm dominant in existing scholarship. The findings reveal that the mismatch between adolescents' endogenous circadian phase delay and rigid social temporal structures is a core driver of heightened depression vulnerability in this population, addressing the gap of insufficient attention to structural constraints in prior individual-focused intervention research. Accordingly, interventions to support adolescent circadian health should extend beyond individual behavioral strategies to the construction of an inclusive social temporal order that recognizes biological timing diversity, providing a novel theoretical foundation and actionable guidance for advancing adolescent mental health promotion systems.

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