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Predicting Adolescent Depressive Symptom Severity from Rumination, Sleep Variability, and Heart-Rate Variability Using Multimodal Deep Learning

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A B S T R A C T

Objective: The objective of this study was to develop and evaluate a multimodal deep learning architecture capable of predicting adolescent depressive symptom severity by dynamically integrating static cognitive rumination scores with continuous, time-series sequences of sleep variability and heart-rate variability.

Methods and Materials: A two-week prospective observational study was conducted involving $N = 418$ adolescents (ages $13 - 18$) from urban and semi-urban districts of Lagos, Nigeria. Baseline cognitive vulnerability and depressive symptoms were assessed via self-report using the Ruminative Responses Scale (RRS) and the Patient Health Questionnaire for Adolescents (PHQ-A). Continuous physiological data, specifically nocturnal sleep variability (standard deviation of total sleep time) and heart-rate variability (HRV; specifically RMSSD), were collected continuously using wrist-worn actigraphy and photoplethysmography (PPG) devices. Data analysis was executed using a hybrid multimodal deep learning architecture featuring late fusion, which utilized Long Short-Term Memory (LSTM) networks to process the physiological time-series data and a Multilayer Perceptron (MLP) for the static cognitive data. The model was trained using the Adam optimizer to minimize Mean Squared Error and evaluated utilizing 5-fold cross-validation.

Findings: The final sample had a mean age of $M = 15.42$years ($SD = 1.35$), with baseline PHQ-A scores of $M = 9.85(SD = 4.62)$ and baseline RRS scores of $M = 46.30$. Bivariate analyses indicated that follow-up depression severity was significantly predicted by rumination ($r = .58, p < .001$), sleep variability ($r = .41, p < .001$), and nocturnal RMSSD ($r = -.36, p < .001$). The multimodal late-fusion network demonstrated exceptional predictive accuracy ($R^2 = .79$, $RMSE = 2.22$), substantially outperforming all isolated unimodal models (Static MLP $R^2 = .61$; Sleep LSTM $R^2 = .38$; HRV LSTM $R^2 = .32$). Ablation studies confirmed the necessity of all modalities and temporal dynamics; excluding the rumination feature caused a significant performance drop ($\Delta R^2 =$

−.14), and replacing the time-series physiological sequences with aggregated 14-day averages resulted in an identical loss of predictive power ($\Delta R^2 = -.14$).
Conclusion: Fusing objective, time-series physiological biomarkers with cognitive vulnerability profiles via multimodal deep learning provides a highly accurate and transformative computational framework for the proactive risk stratification and early clinical intervention of adolescent depression.
Keywords: *Adolescent Depression, Rumination, Sleep Variability, Heart-Rate Variability, Multimodal Deep Learning.*

1. Introduction

Adolescent depression constitutes a profound and escalating public health crisis worldwide, characterized by substantial psychosocial morbidity, disrupted cognitive development, and an elevated risk of mortality. The onset of major depressive disorder during the critical developmental window of adolescence frequently heralds a recurrent and chronic trajectory, precipitating severe impairments across academic, social, and familial domains. Notably, depressive disorders during this stage are associated with significant deficits in executive functions, often manifesting similarly to sluggish cognitive tempo, thereby compounding the academic and functional challenges faced by these youth (Abdolmohamadi & Ghadiri, 2023). Beyond the immediate psychological distress, early-onset major depressive disorder significantly predisposes youth to accelerated physiological weathering, notably an elevated risk for early atherosclerosis and subsequent cardiovascular disease (Goldstein et al., 2015). Given the multifaceted etiology of adolescent depression, traditional diagnostic and prognostic paradigms, which heavily rely on episodic and subjective self-report methodologies, frequently fail to capture the dynamic and complex interplay of cognitive, behavioral, and physiological vulnerabilities. Consequently, there is an urgent need to identify robust, multidimensional biomarkers that can accurately predict the severity of depressive symptoms, thereby facilitating targeted and timely interventions.

A central psychological vulnerability factor in the onset and maintenance of depressive symptomatology is rumination. Rumination is defined as a perseverative and passive cognitive style characterized by repetitive focus on the symptoms, causes, and consequences of one's distress. A well-established body of literature highlights the robust relationship between this ruminative cognitive style and the exacerbation of adolescent depression (Wang et al., 2024). The neurobiological underpinnings of this phenomenon involve aberrant brain mechanisms related to negative self-referential processing, which heavily compromise emotional regulation in depressed youth (Murray et al., 2024). Rumination does not occur in a vacuum; social interactions can profoundly influence its trajectory. For instance, co-rumination—the excessive discussion of personal problems within dyads—has been shown to significantly predict both depression and anxiety, even impacting long-term relationship satisfaction across varying age groups (Whitewolf, 2025). Meta-analytic evidence further confirms that co-rumination consistently correlates with heightened depressive symptoms across diverse adolescent populations (Dong et al., 2025).

The detrimental effects of rumination are pervasive and operate through various mediating and moderating pathways. In adolescents, severe rumination, coupled with poor emotional regulation, has been identified as a significant catalyst for non-suicidal self-injury behaviors (He et al., 2025). Furthermore, rumination acts as a crucial longitudinal mediator between depressive symptoms and behavioral addictions, such as problematic smartphone use, further isolating the adolescent (Li et al., 2024). The cognitive inflexibility inherent in rumination also manifests as self-criticism, which is strongly associated with emotional risk factors and an increased likelihood of suicidal ideation (Lo & Cheng, 2024). Even in broader clinical contexts, such as among infertile women (Luo et al., 2025) or individuals struggling with anger and unforgiveness (Rahmanian, 2025), rumination forms a toxic chain mediation that anchors the individual in a depressed mood. It has also been shown to interact with excessive mind-wandering and mindfulness deficits, mediating the relationship between attention-deficit hyperactivity disorder symptoms and adult depression (Kandeğer et al., 2024). Environmental stressors, including parental nurturing attitudes and peer victimization, frequently catalyze this depressive rumination, exacerbating anxiety and depression across the lifespan (Masuya et al., 2025). Given its clinical significance, therapies specifically targeting rumination, such as rumination-focused cognitive behavioral therapy, have demonstrated efficacy in reducing targeted cross-network brain connectivity and symptom severity in youth (Langenecker et al., 2024). Similarly, neuromodulatory interventions like repetitive transcranial

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magnetic stimulation have been effective in simultaneously addressing depression and ruminative responses in vulnerable populations (Saeidi-Nejad et al., 2025).

While rumination represents a critical cognitive diathesis, the behavioral and physiological dimensions of sleep architecture play an equally paramount role in adolescent depression. Sleep disturbances are universally recognized as both a core symptom and a bidirectional risk factor for affective disorders. Longitudinal cohort studies, such as a prominent 10-year analysis from England, unequivocally demonstrate that poor baseline sleep quality and deteriorating sleep trajectories are significantly associated with an elevated risk of developing clinical depression (Yang et al., 2025). The systemic impact of these sleep disorders is severe enough to accelerate biological aging pathways, with depressive symptoms acting as a primary mediating mechanism (Zhang et al., 2025). The modern adolescent environment, heavily saturated with digital stimuli, further complicates sleep hygiene; excessive smartphone usage frequently precipitates sleep degradation, a relationship mediated by heightened feelings of loneliness and depression (Lai et al., 2025). Conversely, the preservation of good sleep quality can serve as a potent protective factor, shielding individuals from depressive symptoms linked to social isolation (Jiang et al., 2025). Modifiable lifestyle factors, particularly physical exercise, have also been shown to moderate the debilitating pathway from poor sleep quality to severe outcomes like suicidal ideation (Liu et al., 2025). Given the centrality of sleep to mental health recovery, advanced clinical trials comparing dynamic interpersonal therapy, cognitive behavioral therapy, and pharmacotherapy frequently emphasize objective improvements in sleep and cognition as primary indicators of treatment success (Renani & Zare, 2025). While total sleep time is often evaluated, the day-to-day intra-individual variability of sleep—a metric of circadian instability—has emerged as a potentially more sensitive prognostic marker for affective dysregulation.

Parallel to cognitive and sleep-related vulnerabilities, dysregulation of the autonomic nervous system provides a critical, objective physiological window into depressive psychopathology. The bidirectional link between depression and cardiovascular health is well-documented; depression is an established independent risk factor for the development and progression of cardiovascular disease (Hare et al., 2014). In patients with compromised cardiac function, psychological distress, characterized by maladaptive coping strategies, directly correlates with elevated anxiety and depression levels (Bakhtiyarovich et al., 2023). Furthermore, feelings of loneliness and depression heavily predict declining physical and mental health-related quality of life in cardiac patients over time (Fan et al., 2023). Interventions addressing depression in these populations, such as cognitive behavior therapy for heart failure patients, aim to improve both self-care and autonomic outcomes (Freedland et al., 2015). In the context of adolescent depression, this autonomic dysregulation is most accurately quantified through continuous monitoring of Heart-Rate Variability (HRV). HRV, mathematically derived from the intervals between successive heartbeats, serves as a non-invasive proxy for vagal tone and parasympathetic nervous system flexibility. Depressed adolescents consistently exhibit blunted HRV, signifying a rigid autonomic state incapable of adaptively responding to environmental or psychological stressors.

Despite the established individual prognostic value of rumination, sleep variability, and HRV, the overwhelming majority of psychiatric literature evaluates these modalities in isolation. Depressive psychopathology, however, is inherently multidimensional, operating at the intersection of cognitive habits, behavioral routines, and physiological functioning. Traditional linear statistical models are frequently ill-equipped to handle the high-dimensional, time-series data generated by modern continuous physiological monitoring. To fully elucidate the predictive power of these intersecting domains, advanced computational architectures, specifically multimodal deep learning, are required. Deep learning frameworks, utilizing layers of artificial neurons mathematically structured to learn complex non-linear representations, offer a revolutionary approach to psychiatric prediction. By integrating static psychometric variables (such as cognitive rumination scores) with dynamic, sequential physiological data (such as 14-day vectors of sleep variability and HRV fluctuations), these models can map hidden interactive patterns that evade traditional analysis. For instance, an algorithm can identify how specific temporal fluctuations in autonomic arousal over $t$days interact with high baseline rumination $R$to produce acute exacerbations in depressive severity Y.

The integration of such disparate data streams into a cohesive predictive framework represents a critical frontier in precision psychiatry. To date, very few studies have attempted to computationally fuse subjective cognitive phenotypes with objective, continuous physiological biomarkers to predict mental health outcomes in vulnerable

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adolescent populations. Bridging this methodological gap is essential for the development of highly accurate, personalized risk stratification models. By leveraging the continuous data streams provided by wearable biosensors alongside validated psychological inventories, it becomes possible to capture the holistic state of the adolescent in their naturalistic environment. Therefore, the aim of this study is to develop and evaluate a multimodal deep learning architecture capable of predicting adolescent depressive symptom severity by integrating cognitive rumination scores with continuous, time-series data of sleep variability and heart-rate variability.

2. Methods and Materials

2.1. Study Design and Participants

This research utilized a prospective observational study design to capture multidimensional data over a continuous two-week period, aiming to predict the severity of depressive symptoms in adolescents. The primary population of interest consisted of high school students residing in urban and semi-urban districts of Lagos, Nigeria. A purposeful sampling strategy was initially employed to approach diverse educational institutions, followed by stratified random sampling to select individual participants across various socioeconomic backgrounds and gender identities. The final analytical sample comprised exactly four hundred and eighteen adolescents between the ages of thirteen and eighteen. Prior to enrollment, comprehensive written informed consent was obtained from the legal guardians or parents of all participants, alongside the documented assent of the adolescents themselves. To ensure the integrity of the multimodal data, stringent inclusion and exclusion criteria were applied. Participants were required to possess a basal level of physical health and be capable of operating the provided wearable technology. Individuals were excluded from the study if they had a prior clinical diagnosis of a severe psychiatric disorder outside of major depressive disorder, such as bipolar disorder or schizophrenia, or if they were currently taking medications known to significantly alter cardiovascular function or sleep architecture.

2.2. Measures

The comprehensive data collection framework integrated validated self-report psychological inventories with continuous physiological monitoring via consumer-grade wearable biosensors. To quantify cognitive rumination, participants completed the Ruminative Responses Scale, a widely utilized self-report questionnaire that assesses the tendency to repetitively focus on the causes, situational factors, and consequences of one's negative affective state. Depressive symptom severity, which served as the primary target variable for the predictive models, was evaluated using the Patient Health Questionnaire tailored for adolescents, administered both at the baseline and at the conclusion of the two-week monitoring phase. To objectively capture physiological data, each participant was equipped with a validated wrist-worn actigraphy and photoplethysmography device, which they were instructed to wear continuously for fourteen days. Sleep variability was operationalized by extracting daily metrics such as total sleep time, sleep onset latency, and wake after sleep onset, from which the intra-individual standard deviation across the monitoring period was computed to serve as the variability index. Heart-rate variability was derived from the continuous photoplethysmography signal, specifically focusing on the root mean square of successive differences between normal heartbeats and the standard deviation of normal-to-normal intervals. These physiological metrics were computed during a standardized nocturnal window to minimize the confounding effects of daytime physical exertion and acute environmental stressors, thereby providing a stable baseline of autonomic nervous system functioning.

2.3. Data Analysis

The analytical pipeline was centered on the development and evaluation of a multimodal deep learning architecture designed to integrate static psychometric scores with dynamic, time-series physiological data. Prior to model ingestion, all input features underwent rigorous preprocessing; missing physiological data points were imputed using a localized linear interpolation method, and all continuous variables were standardized using z-score normalization to ensure zero mean and unit variance. The deep learning model utilized a hybrid architecture featuring distinct processing branches for each data modality. Time-series data, specifically the daily sleep parameters and nocturnal heart-rate variability metrics, were fed into a Long Short-Term Memory network to capture temporal dependencies and sequential variability patterns. Concurrently, the static baseline data, including demographic information and the rumination scores, were

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processed through a multi-layer perceptron. The high-level feature representations generated by these parallel branches were subsequently concatenated in a late-fusion layer. This fused representation was then passed through a series of fully connected dense layers, culminating in a linear output layer that predicted the continuous depressive symptom severity score. The network was trained using the Adam optimizer with a learning rate dynamically adjusted via a cosine annealing schedule. The optimization objective was to minimize the mean squared error loss function, defined mathematically as $MSE = \frac{1}{n}\sum_{i=1}^{n}(y_i - \hat{y}_i)^2$, where $y_i$represents the actual depression score and $\hat{y}_i$represents the model's prediction. To prevent overfitting and ensure the generalizability of the model, a five-fold cross-validation strategy was implemented alongside dropout regularization techniques. Model performance was ultimately evaluated using a combination of the coefficient of determination R^2, the mean absolute error, and the root mean squared error, providing a comprehensive assessment of the model's predictive accuracy and clinical utility.

3. Findings and Results

The final analytical sample comprised $N = 418$adolescents. The mean age of the participants was $M = 15.42$ years ($SD = 1.35$), with a relatively balanced gender distribution consisting of $n = 218$ females (52.2%) and $n = 200$ males (47.8%). At the baseline assessment, the mean score for depressive symptom severity, measured by the Patient Health Questionnaire for Adolescents (PHQ-A), was $M = 9.85(SD = 4.62)$, indicating a sample average bordering on mild to moderate depressive symptomatology. Notably, an assessment of the PHQ-A categorical severity indicated that 28.5%of the sample met the criteria for moderate depression, while 14.2%met the criteria for moderately severe to severe depression prior to the monitoring period.

The physiological monitoring yielded consistent data across the two-week period. The average total sleep time (TST) was $M = 398.50$minutes per night ($SD = 45.20$). Sleep variability, operationalized as the intra-individual standard deviation of total sleep time across the fourteen days, averaged $M = 42.15$minutes ($SD = 18.30$). For nocturnal autonomic functioning, the root mean square of successive differences between normal heartbeats (RMSSD) yielded a sample mean of $M = 48.60$ms ($SD = 15.45$). A comprehensive summary of the demographic and baseline clinical characteristics is presented in Table 1.

Table 1

Demographic and Baseline Clinical Characteristics of the Study Sample (N=418)

Variable	Mean / Frequency	SD / Percentage
Age (years)	15.42	1.35
Gender (Female)	218	52.2%
Baseline PHQ-A Score	9.85	4.62
Ruminative Responses Scale (RRS)	46.30	11.85
Total Sleep Time (minutes/night)	398.50	45.20
Total Sleep Time Variability (minutes)	42.15	18.30
Nocturnal RMSSD (ms)	48.60	15.45
Nocturnal SDNN (ms)	62.35	18.90

Prior to the deep learning model ingestion, Pearson product-moment correlation coefficients were computed to examine the zero-order relationships between the primary predictors (rumination, sleep variability, and HRV) and the target variable, which was the post-monitoring PHQ-A depression score ($M = 10.12$, $SD = 4.85$). As detailed in Table 2, cognitive rumination demonstrated a strong, positive correlation with follow-up depressive severity ($r = .58$, $p < .001$).

Regarding the objective physiological markers, total sleep time variability showed a significant positive association with depression scores ($r = .41$, $p < .001$), suggesting that highly irregular sleep patterns over the two-week period were linked to greater symptom severity. Conversely, parasympathetic nervous system activity, as indexed by nocturnal RMSSD, was inversely correlated with depression severity ($r = -.36$, $p < .001$), indicating that higher resting vagal tone is associated with lower depressive symptomatology. While mean total sleep time did not significantly correlate with depression ($r = -.08$, $p = .103$), its variability was a much stronger prognostic

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indicator, validating the study's focus on dynamic physiological fluctuations.

Table 2

Bivariate Correlations Between Main Study Variables

Variable	1	2	3	4	5
1. Follow-up PHQ-A	–				
2. Rumination (RRS)	.58**	–			
3. Sleep Variability (TST-SD)	.41**	.28**	–		
4. HRV (RMSSD)	−.36**	−.19**	−.22**	–	
5. Baseline PHQ-A	.72**	.45**	.31**	−.28**	–

** $p < .001$.

The core analysis evaluated the predictive capability of the proposed multimodal deep learning architecture against several unimodal baseline models. The models were evaluated using 5-fold cross-validation, and performance was quantified using the coefficient of determination (R^2), Mean Absolute Error (MAE), and Root Mean Squared Error (RMSE).

The baseline Multi-Layer Perceptron (MLP) trained solely on static data (demographics, baseline PHQ-A, and rumination) achieved moderate predictive success ($R^2 = .61$, $RMSE = 3.03$). The unimodal Long Short-Term Memory (LSTM) networks trained exclusively on temporal physiological data (either sleep variability or HRV sequences alone) yielded lower standalone predictive power ($R^2 = .38$ and $R^2 = .32$, respectively). However, the late-fusion Multimodal Deep Learning Model, which dynamically integrated both the static psychometric representations and the dynamic physiological temporal sequences, drastically outperformed all unimodal approaches. The final multimodal architecture achieved a highly robust predictive accuracy ($R^2 = .79$), accounting for nearly 80%of the variance in adolescent depressive symptom severity at the two-week follow-up, with substantially minimized error rates ($MAE = 1.65$, $RMSE = 2.22$).

Table 3

Model Performance Metrics Across Different Architectures

Model Architecture	Input Modality	R^2	MAE	RMSE
Static MLP	Demographics + RRS + Baseline PHQ-A	.61	2.31	3.03
Sleep LSTM	Continuous Actigraphy (Sleep)	.38	3.15	3.82
HRV LSTM	Continuous PPG (HRV)	.32	3.40	4.01
Early Fusion NN	All Modalities (Flattened)	.68	2.05	2.75
Multimodal Late-Fusion	All Modalities (Temporal + Static)	.79	1.65	2.22

To elucidate the specific contributions of the physiological and psychological modalities within the highest-performing late-fusion network, a systematic ablation study was conducted. Modalities were iteratively withheld from the training and validation phases to observe the subsequent degradation in model performance.

The ablation results, summarized in Table 4, confirmed that the integration of continuous physiological variability significantly augmented the predictive capacity provided by psychological questionnaires alone. Removing cognitive rumination resulted in the largest singular performance drop ($\Delta R^2 = -.14$), highlighting its critical role in the psychopathology of adolescent depression. However, the removal of sleep variability ($\Delta R^2 = -.11$) and HRV sequences ($\Delta R^2 = -.09$) also precipitated severe degradations in the model's accuracy. Furthermore, excluding the temporal dynamics (by replacing sequential daily physiological data with simple 14-day averages) resulted in an $R^2$of .65, which was a marked reduction from the optimal .79. This specific finding unequivocally demonstrated that the deep learning model successfully extracted highly prognostic temporal patterns from the day-

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to-day variability of sleep and autonomic functioning, which traditional aggregated averages failed to capture.

Table 4

Ablation Study Results Demonstrating Modality Contributions

Modality Excluded / Altered	Resulting R^2	Change in $R^2(\Delta)$	Resulting RMSE
None (Full Multimodal Model)	.79	–	2.22
Withhold Rumination (RRS)	.65	−.14	2.87
Withhold Sleep Variability	.68	−.11	2.75
Withhold HRV Sequences	.70	−.09	2.66
Use Averages Instead of Sequences	.65	−.14	2.88
Withhold Baseline PHQ-A	.72	−.07	2.56

4. Discussion

The primary objective of this study was to develop and evaluate a multimodal deep learning architecture capable of predicting the severity of adolescent depressive symptoms by integrating cognitive rumination scores with continuous, time-series data of sleep variability and heart-rate variability. The findings of this investigation robustly demonstrate that a late-fusion deep learning model, which dynamically amalgamates static psychometric vulnerability factors with objective temporal physiological sequences, significantly outperforms traditional unimodal predictive approaches. Specifically, the multimodal architecture achieved a highly robust predictive accuracy of $R^2 = .79$, accounting for nearly 80%of the variance in depressive symptom severity at the two-week follow-up. These results underscore the multidimensional nature of adolescent psychopathology and highlight the profound clinical utility of integrating wearable biosensor data with validated psychological assessments to formulate precise psychiatric prognostications.

Consistent with our hypotheses, cognitive rumination emerged as a highly potent predictor of subsequent depressive symptom severity. Our bivariate analyses revealed a strong positive correlation between baseline rumination scores and follow-up depression ($r = .58$). Furthermore, the ablation study demonstrated that the removal of the rumination modality from the deep learning network precipitated the most substantial degradation in predictive accuracy ($\Delta R^2 = -.14$). This finding aligns seamlessly with established psychological literature emphasizing the central role of rumination in the etiology and maintenance of depressive disorders (Wang et al., 2024). The neurobiological underpinnings of this perseverative cognitive style involve maladaptive negative self-referential processing, which significantly impairs an adolescent's capacity for emotional regulation (Murray et al., 2024). When youth are trapped in a cycle of repetitive negative thinking, their psychological vulnerability escalates, often leading to severe clinical outcomes such as non-suicidal self-injury (He et al., 2025) and the exacerbation of suicidal ideation through heightened self-criticism (Lo & Cheng, 2024). The pervasive toxicity of rumination is further compounded when it occurs in social or dyadic contexts. For instance, co-rumination consistently magnifies depressive symptoms (Dong et al., 2025) and predicts long-term anxiety and relationship dissatisfaction (Whitewolf, 2025). The cognitive inflexibility inherent in rumination also serves as a critical longitudinal mediator, linking depressive symptoms to behavioral compensations like problematic smartphone use (Li et al., 2024), and anchoring individuals in depressed moods across various clinical populations (Luo et al., 2025; Rahmanian, 2025). Interventions that specifically target these ruminative loops, such as rumination-focused cognitive behavioral therapy, have proven essential in reducing cross-network brain connectivity associated with adolescent depression (Langenecker et al., 2024), further validating the necessity of monitoring and addressing this cognitive diathesis as demonstrated by our model's reliance on this feature. Furthermore, the interaction between excessive mind-wandering and rumination frequently mediates severe psychiatric comorbidities, highlighting its foundational role in adolescent mental health decline (Kandeğer et al., 2024). Environmental factors, such as peer victimization and poor parental nurturing, often trigger this depressive rumination, solidifying its place as a primary psychological vulnerability (Masuya et al., 2025). Modulating these cognitive responses, even through advanced somatic treatments like repetitive transcranial magnetic stimulation, remains a critical therapeutic frontier (Saeidi-Nejad et al., 2025).

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Beyond the cognitive domain, our objective physiological monitoring revealed that dynamic sleep variability is a critical, independent prognostic marker for adolescent depression. While the average total sleep time across the monitoring period did not significantly correlate with depression severity ($r = -.08$), the intra-individual standard deviation of sleep duration—representing night-to-night sleep variability—exhibited a strong positive association with depressive outcomes ($r = .41$). The network ablation study confirmed that temporal sleep variability sequences were indispensable for optimal model performance. This finding challenges the traditional reliance on aggregated sleep duration metrics, suggesting that circadian instability and erratic sleep architectures are more sensitive indicators of affective dysregulation. This aligns with longitudinal evidence indicating that deteriorating sleep quality trajectories dramatically increase the risk of clinical depression (Yang et al., 2025). The physiological toll of such sleep disturbances is profound, often accelerating biological aging pathways through the mediating mechanism of depressive symptoms (Zhang et al., 2025). In the contemporary digital age, irregular sleep patterns in adolescents are frequently exacerbated by excessive nighttime smartphone use, a behavioral pattern strongly linked to loneliness and depression (Lai et al., 2025). Conversely, stable and high-quality sleep acts as a robust protective factor, shielding vulnerable individuals from the depressive symptoms typically associated with social isolation (Jiang et al., 2025). Restoring sleep architecture is therefore a primary objective in advanced psychiatric treatments, as improvements in sleep often dictate the success of interventions ranging from dynamic interpersonal therapy to pharmacotherapy (Renani & Zare, 2025). By capturing the day-to-day erraticism of sleep rather than a static average, our deep learning model successfully operationalized this circadian instability, proving that physiological unpredictability is a hallmark of the depressed adolescent brain. As highlighted by recent interventions, even behavioral modifications like physical exercise can moderate the pathway between poor sleep quality and severe outcomes like suicidal ideation, further emphasizing the modifiable nature of this physiological risk factor (Liu et al., 2025). Furthermore, the cognitive deficits observed in depression, similar to sluggish cognitive tempo, are intimately tied to restorative sleep, reinforcing the necessity of monitoring sleep variability to protect executive functioning (Abdolmohamadi & Ghadiri, 2023).

In parallel with sleep instability, the continuous monitoring of autonomic nervous system function via nocturnal heart-rate variability (HRV) provided a vital physiological window into the adolescents' psychological distress. Our results indicated a significant inverse relationship between parasympathetic tone, as indexed by nocturnal RMSSD, and depressive symptom severity ($r = -.36$). Depressed adolescents exhibited blunted HRV, indicative of a rigid autonomic state that lacks the physiological flexibility required to adapt to environmental or psychological stressors. The systemic implications of this autonomic dysregulation are severe, as depression is an established independent risk factor for the accelerated development of cardiovascular disease, particularly when onset occurs during youth (Goldstein et al., 2015; Hare et al., 2014). The withdrawal of vagal tone not only reflects current psychological distress but also predicts declining physical and mental health-related quality of life (Fan et al., 2023). In populations with compromised cardiovascular health, maladaptive psychological coping and subsequent depression are inextricably linked to this autonomic rigidity (Bakhtiyarovich et al., 2023). Interventions that successfully alleviate depressive symptoms, such as targeted cognitive behavior therapy, concurrently improve these physiological markers, highlighting the bidirectional neurovisceral integration in affective disorders (Freedland et al., 2015). The inclusion of sequential HRV data in our deep learning architecture allowed the model to detect micro-fluctuations in autonomic arousal that precede conscious reports of distress. The ablation results ($\Delta R^2 = -.09$) confirmed that excluding these temporal HRV sequences significantly compromised the model's predictive precision.

The most profound methodological insight of this study lies in the synergistic power of the multimodal late-fusion deep learning architecture. While unimodal models utilizing only continuous actigraphy ($R^2 = .38$) or continuous photoplethysmography ($R^2 = .32$) demonstrated limited predictive validity, their integration with static cognitive vulnerability data elevated the predictive accuracy to exceptional levels. Crucially, when the temporal sequences of sleep and HRV were collapsed into simple 14-day averages, the model's performance dropped substantially ($\Delta R^2 = -.14$). This mathematically validates the assertion that the dynamic interaction over time between cognitive rumination and physiological instability contains critical prognostic information that is entirely lost in traditional cross-sectional or aggregated analyses. The algorithm successfully identified how specific patterns of autonomic

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withdrawal interacting with sleep erraticism exacerbate the underlying vulnerability of a ruminative cognitive style, resulting in a highly accurate prediction of subjective depressive severity Y.

5. Conclusion

In conclusion, this study demonstrates the profound clinical utility of integrating static psychological vulnerability markers with continuous physiological time-series data to predict adolescent depressive symptom severity. By leveraging a multimodal late-fusion deep learning architecture, our model achieved a robust predictive accuracy of $R^2 = .79$, significantly outperforming traditional unimodal approaches. The findings confirm that while cognitive rumination remains a primary driver of depressive affect, the dynamic intra-individual erraticism of sleep and the autonomic rigidity reflected in blunted heart-rate variability are indispensable objective biomarkers. Crucially, the substantial drop in model performance when temporal sequences were replaced with static averages highlights the necessity of analyzing physiological fluctuations over time rather than relying on aggregated cross-sectional metrics. Ultimately, fusing validated psychometric assessments with objective wearable biosensor data provides a transformative, scalable framework for proactive risk stratification, paving the way for personalized, early interventions in adolescent mental health care.

6. Limitations & Suggestions

Despite the robust predictive capabilities demonstrated by the multimodal deep learning architecture, several methodological limitations must be acknowledged. First, the sample was geographically restricted to urban and semi-urban adolescents in Lagos, Nigeria, which may limit the generalizability of the findings to rural populations or adolescents in different cultural and socio-economic contexts. Second, while consumer-grade wearable biosensors offer the distinct advantage of continuous, naturalistic data collection, they are inherently susceptible to motion artifacts and data loss, which, despite rigorous preprocessing and interpolation, may introduce noise into the temporal sequences. Third, the reliance on self-report questionnaires for the assessment of rumination and baseline depressive symptoms introduces the potential for response biases, such as social desirability or state-dependent memory recall, which could marginally skew the static input features of the predictive model. Finally, the observational period was restricted to two weeks; while sufficient for capturing short-term physiological variability, it precludes the examination of long-term developmental trajectories and the chronicity of depressive episodes over months or years.

To build upon the foundation established by this study, future research should prioritize longitudinal designs that track adolescent cohorts over extended developmental periods, potentially spanning several months to years, to capture the seasonal and academic fluctuations in both physiological variability and affective states. Furthermore, researchers should explore the integration of additional continuous physiological modalities, such as electrodermal activity or continuous skin temperature, to provide a more comprehensive multidimensional profile of autonomic arousal and circadian rhythms. Investigating alternative deep learning architectures, such as Transformer-based models with self-attention mechanisms, may also yield improvements in identifying the most critical temporal physiological sequences that precede acute depressive exacerbations. Finally, future studies should aim to validate these multimodal predictive models across diverse international cohorts and distinct clinical subpopulations to ensure cross-cultural reliability and algorithmic fairness.

The successful prediction of depressive symptom severity using commercially available wearables and standard psychological assessments presents transformative opportunities for clinical practice and school-based mental health initiatives. Mental health practitioners can integrate this multimodal approach into early warning systems within educational institutions, identifying at-risk adolescents based on subtle physiological shifts and cognitive screening before full-syndromal clinical depression manifests. By recognizing the equal importance of physiological variability and cognitive habits, clinicians can design highly personalized, pre-emptive interventions. For instance, an adolescent flagged by the algorithm for high sleep erraticism and elevated rumination could be immediately routed into targeted psychoeducation programs focusing on sleep hygiene stabilization and cognitive defusion techniques. Ultimately, shifting the psychiatric paradigm from reactive, episodic treatment to proactive, continuous, and objective monitoring has the potential to drastically reduce the burden of adolescent depression and mitigate its long-term psychological and physiological consequences.

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Declaration of Interest

The authors of this article declared no conflict of interest.

Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants.

Transparency of Data

In accordance with the principles of transparency and open research, we declare that all data and materials used in this study are available upon request.

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Authors' Contributions

All authors equally contributed to this article.

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