

Identifying Novel Endophenotypes of Adolescent Insomnia and their Association with Emotional Dysregulation

Eleni. Kouris¹, Yara. Mahfouz^{2*}, Andrei. Dumitrescu³

¹ Department of Educational Psychology, University of Crete, Heraklion, Greece

² Department of Counseling Psychology, Ain Shams University, Cairo, Egypt

³ Department of Applied Psychology, University of Bucharest, Bucharest, Romania

* Corresponding author email address: yara.mahfouz@asu.edu.eg

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ABSTRACT

Objective: This study aimed to identify unobservable endophenotypes of adolescent insomnia utilizing latent variable modeling and to evaluate their differential associations with specific facets of emotional dysregulation.

Methods and Materials: A cross-sectional design was employed, encompassing a community sample of $n = 846$ adolescents in Egypt. Sleep parameters were rigorously assessed using continuous objective actigraphy worn in the home environment, paired with the subjective self-report Insomnia Severity Index. Emotional dysregulation was quantified using the multidimensional Difficulties in Emotion Regulation Scale. To uncover hidden sub-populations, Latent Profile Analysis was conducted in R using the combined objective and subjective sleep metrics. Subsequently, a Multivariate Analysis of Covariance was utilized to determine how the derived sleep profiles differentially predicted impairments across the various emotional dysregulation subscales, controlling for demographic covariates.

Findings: The Latent Profile Analysis successfully identified 3 distinct insomnia endophenotypes within the adolescent sample: Normative Sleepers, Sleep Onset Delay, and High Fragmentation. The multivariate analyses revealed highly specific associations between these profiles and emotional functioning. Adolescents classified within the Sleep Onset Delay profile demonstrated specialized emotional deficits, specifically characterized by a lack of emotional clarity and severe difficulties engaging in goal-directed behaviors ($p < 0.01$). Conversely, the High Fragmentation profile—marked by extensive wake after sleep onset and reduced total sleep time—was associated with the most severe global impairments in emotion regulation, manifesting primarily as profound deficits in impulse control and a high nonacceptance of emotional responses ($p < 0.001$).

Conclusion: Adolescent insomnia is not a monolithic disorder but a heterogeneous construct comprising distinct endophenotypes linked to specific emotional vulnerabilities. Precision behavioral medicine that tailors interventions to exact sleep architectural disruptions may significantly optimize clinical outcomes and mitigate psychopathological risks.

Keywords: Adolescent Insomnia; Endophenotypes; Latent Profile Analysis; Emotional Dysregulation

1. Introduction

Adolescence represents a critical developmental period characterized by profound biological, cognitive, and psychosocial transitions. Concurrent with these developmental milestones is a significant vulnerability to sleep disturbances, with adolescent insomnia emerging as a pervasive public health concern. Epidemiological data suggest that a substantial proportion of adolescents experience chronic sleep deprivation and clinically significant insomnia symptoms, which profoundly disrupt their developmental trajectory (Uccella et al., 2023). The clinical presentation of insomnia during this sensitive period is rarely isolated; it frequently co-occurs with an array of somatic and psychiatric conditions. For instance, adolescent insomnia has been identified as a robust antecedent and concomitant risk factor for the first lifetime onsets of major depressive disorder, generalized anxiety, and suicidality (Soltani et al., 2023). Furthermore, the somatic manifestations of chronic sleep disruption are diverse, encompassing conditions such as irritable bowel syndrome, which significantly diminishes the health-related quality of life among affected youths (Yang, 2021). The phenotypic expression of psychopathology, particularly depression, often differs between adolescents and adults, with sleep architecture alterations serving as a core differentiating symptom profile (Rice et al., 2019). Consequently, understanding the complex etiology and heterogeneous manifestation of adolescent insomnia is imperative for mitigating its long-term detrimental effects on somatic and psychological well-being.

The etiology of adolescent insomnia is multifactorial, heavily influenced by contemporary lifestyle factors, environmental stressors, and the pervasive integration of digital technology into daily routines. The modern adolescent landscape is characterized by constant connectivity, leading to phenomena such as nomophobia—the severe fear of being detached from mobile connectivity—and the addictive use of social media platforms. Longitudinal analyses have demonstrated that these digital dependencies are bidirectionally linked to the exacerbation of insomnia symptoms, primarily through heightened pre-sleep somatic arousal and delayed circadian phase alignment (Lin et al., 2021). Beyond screen time, internet addiction broadly correlates with a complex network of maladaptive behaviors, including decreased physical activity and elevated suicidal ideation, with insomnia acting as a central bridging node within this psychopathological

network (Lu et al., 2025). Environmental adversities and traumatic life events further compound sleep vulnerabilities. Profound psychosocial stressors, such as parental death during adolescence, precipitate severe grief trajectories and protracted sleep architecture disruptions (Farella Guzzo & Gobbi, 2021). Similarly, insidious forms of trauma, such as emotional neglect, significantly predict non-suicidal self-injury among youths, a relationship that is substantially mediated by the concurrent presence of social anxiety symptoms and chronic insomnia (Hou et al., 2023).

Psychological distress and specific personality typologies also play a pivotal role in the maintenance of insomnia. The measurement of negative emotional states, structurally validated by instruments differentiating between depression, anxiety, and stress dimensions (Lovibond & Lovibond, 1995), consistently highlights the primacy of hyperarousal in sleep disruption. Excess cognitive and somatic anxiety during late adolescence acts as a potent catalyst for the acute onset of insomnia, overwhelming the individual's homeostatic sleep drive (Bao & Bihn, 2021). Moreover, the relationship between daily psychological stress and physical health is frequently mediated by behavioral alterations; for instance, high stress environments are associated with maladaptive dietary habits and emotional eating behaviors, which indirectly exacerbate insomnia severity (Choi et al., 2021). Intrinsic personality traits further modulate this risk. The interaction between conscientiousness, emotional stability, and sleep quality is deeply influenced by intervening variables of anxiety and depression (Akram et al., 2019). Maladaptive perfectionism, characterized by excessive self-criticism and fear of failure, significantly heightens vulnerability to stress, sequentially increasing the risk of insomnia, with notable gender disparities in the phenotypic expression of these traits (Richardson & Gradisar, 2020). The interplay of neuroticism, insecure attachment styles, and sleep continuity is heavily mediated by cognitive emotion regulation deficits and perfectionistic tendencies (Sohrabian et al., 2020). Interestingly, while insomnia is overwhelmingly associated with deficits, some emerging literature suggests complex cognitive trade-offs, where sleep disturbances may inadvertently interact with the need for cognition to influence domains such as creativity in specific adolescent populations (Ren et al., 2024).

Central to the pathophysiological maintenance of adolescent insomnia is the construct of emotional dysregulation. Emotion dysregulation encompasses a spectrum of difficulties, including an inability to flexibly respond to and manage the valence, intensity, and

chronometry of emotional experiences. Longitudinal investigations in large community samples underscore that pre-existing emotion dysregulation acts as a robust predictor for the future incidence of insomnia, establishing a bidirectional feedback loop where sleep fragmentation subsequently erodes emotional regulatory capacity (Jansson-Fröjmark et al., 2015). Direct clinical comparisons reveal that individuals suffering from primary insomnia exhibit significantly higher levels of experiential avoidance and general emotional dysregulation compared to healthy, non-clinical populations (Zakiei et al., 2020). The intersection of sleep and emotion becomes particularly pronounced when examining severe psychopathological constructs, such as Borderline Personality Disorder (BPD). Specific sub-components of insomnia, particularly sleep onset delay and wake after sleep onset, exert differential influences on the exacerbation of emotion dysregulation within BPD populations (Fitzpatrick et al., 2020). Furthermore, borderline personality traits intensify overall insomnia severity through a sequential mediation pathway involving maladaptive cognitive emotion regulation strategies and heightened pre-sleep cognitive arousal (Park, 2025).

The recognition of emotion dysregulation as a core maintaining mechanism of insomnia has catalyzed the development and refinement of targeted therapeutic interventions. Digital health interventions, such as smartphone application-delivered Cognitive Behavioral Therapy for Insomnia (CBT-I), have demonstrated considerable efficacy in treating adolescent sleep disturbances, though treatment response trajectories and baseline sleep disruption patterns often reveal significant gender differences (Li et al., 2020). Further exploratory analyses of these digital modalities confirm that while CBT-I effectively normalizes sleep architecture, tailoring interventions to account for gender-specific emotional responses may optimize long-term clinical outcomes (Li et al., 2021). Beyond traditional CBT-I, therapies explicitly designed to target emotional processing have shown promise. Emotion-focused therapy, traditionally utilized for interpersonal trauma such as promoting resilience in individuals affected by relational infidelity (Boroumandrad 2020), highlights the broader utility of emotional schema restructuring. Somatic and energy-psychology interventions, such as Emotional Freedom Techniques (EFT), have also demonstrated significant efficacy in reducing physiological hyperarousal and promoting resiliency in highly stressed populations, such as veterans at risk for post-traumatic stress disorder, indirectly benefiting sleep continuity (Church et

al., 2016). Most pertinently, transdiagnostic treatments explicitly rooted in emotion regulation skills training have proven highly effective in single-case and small-cohort experimental designs at significantly reducing the severity of primary insomnia disorder (Hatamian et al., 2023).

Despite the robust literature linking generalized insomnia to emotional dysregulation, a critical gap remains in understanding the heterogeneity of insomnia itself. Insomnia is rarely a monolithic condition; rather, it manifests through varied objective and subjective sleep parameters, such as isolated sleep onset latency (*SOL*), prolonged wake after sleep onset (*WASO*), or isolated subjective dissatisfaction despite normative objective sleep metrics. Traditional analytical approaches often rely on aggregated sum scores or unitary diagnostic labels, failing to capture the multivariate, interactive nature of sleep architecture. Identifying distinct unobservable subgroups, or endophenotypes, based on concurrent objective and subjective sleep data can clarify the mixed findings in the current literature. For instance, an adolescent whose insomnia is characterized primarily by physiological hyperarousal and prolonged *SOL* may exhibit vastly different emotional regulatory deficits compared to an adolescent suffering from severe sleep fragmentation and low total sleep time (*TST*). Understanding these nuanced profiles is essential for transitioning toward precision behavioral medicine, where interventions can be specifically matched to an individual's distinct sleep and emotional dysregulation endophenotype. Therefore, the overarching aim of this study is to utilize person-centered latent variable modeling to identify novel, unobservable endophenotypes of adolescent insomnia based on combined actigraphy and subjective self-report data, and subsequently evaluate how these distinct sleep profiles are differentially associated with specific facets of emotional dysregulation.

2. Methods and Materials

2.1. Study Design and Participants

This study employed a cross-sectional, multi-site observational design to investigate the underlying endophenotypes of insomnia and their specific associations with emotional dysregulation among adolescents. The research was conducted in Egypt, specifically targeting diverse urban and semi-urban educational districts within the governorates of Cairo and Alexandria to ensure a socioeconomically representative cohort. The final sample comprised exactly 846 adolescents, aged between 13 and 18 years, who were recruited through a stratified cluster

sampling technique from twelve public and private high schools. To be included in the study, participants were required to be native Arabic speakers, currently enrolled in full-time education, and able to provide written informed assent alongside documented parental or legal guardian consent. Exclusion criteria were rigorously applied to minimize confounding variables, leading to the exclusion of individuals with a documented history of severe neurological disorders, intellectual disabilities, active substance abuse, or those currently undergoing pharmacological treatment with psychotropic medications known to alter sleep architecture. The study protocol was meticulously reviewed and approved by the regional institutional review board, ensuring strict adherence to the ethical principles outlined in the Declaration of Helsinki regarding human subject research.

2.2. Measures

To comprehensively capture the multifaceted nature of adolescent insomnia, its potential endophenotypes, and associated emotional dysregulation, a combination of validated psychometric instruments and objective physiological measures was utilized. Subjective sleep disturbances were initially quantified using the Arabic-validated version of the Insomnia Severity Index, a self-report questionnaire assessing the nature, severity, and impact of insomnia over the preceding month. To identify novel sleep-related endophenotypes, objective sleep parameters were continuously monitored over a consecutive fourteen-day period using wrist-worn actigraphy devices. Participants were instructed to wear the actigraphs on their non-dominant wrist and maintain a concurrent daily sleep diary to corroborate sleep onset and wake times. These objective measures yielded precise data on total sleep time, sleep onset latency, wake after sleep onset, and overall sleep efficiency. Furthermore, to evaluate the core construct of emotional dysregulation, participants completed the Difficulties in Emotion Regulation Scale. This comprehensive instrument measures multiple facets of emotion dysregulation, including nonacceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity. All psychometric tools were administered in Arabic, having previously demonstrated robust internal consistency and test-retest reliability within Middle Eastern adolescent populations.

2.3. Data Analysis

All statistical procedures were executed using the R statistical programming environment, specifically leveraging packages designed for advanced multivariate analysis and structural equation modeling. Initial data screening was conducted to assess for missing values, outliers, and the assumption of normality among continuous variables. Missing data, which accounted for less than 4% of the total dataset, were handled using multiple imputation by chained equations to maximize statistical power and reduce non-response bias. To identify the novel endophenotypes of adolescent insomnia, Latent Profile Analysis was applied to the objective actigraphy data and the subjective insomnia scores. This person-centered analytical approach allowed for the categorization of the sample into distinct, mutually exclusive latent classes based on their unobservable sleep trait patterns. Model fit for the Latent Profile Analysis was evaluated using the Akaike Information Criterion, the Bayesian Information Criterion, and the Lo-Mendell-Rubin adjusted likelihood ratio test. Once the distinct insomnia endophenotypes were established, generalized linear models and multivariate analysis of covariance were utilized to examine the associations between these newly identified profiles and the various sub-domains of emotional dysregulation, controlling for demographic covariates such as age, gender, and socioeconomic status. Throughout all statistical computations, a two-tailed threshold for statistical significance was established at $\alpha = 0.05$, and effect sizes were calculated using Cohen's *d* and partial eta squared to determine the practical magnitude of the observed associations.

3. Findings and Results

The initial phase of the data analysis focused on evaluating the demographic and baseline clinical characteristics of the final analytical sample, which consisted of $N = 846$ Egyptian adolescents. The cohort exhibited a relatively balanced gender distribution, with $n = 450$ (53.19%) females and $n = 396$ (46.81%) males. The mean age of the participants was $M = 15.42$ years with a standard deviation of $SD = 1.63$ years. Socioeconomic status, derived from parental education and occupational indices, indicated that $n = 215$ (25.41%) participants belonged to a low socioeconomic bracket, $n = 482$ (56.97%) to a middle bracket, and $n = 149$ (17.61%) to a high socioeconomic bracket. Baseline assessments of subjective sleep and emotional dysregulation revealed a

mean Insomnia Severity Index score of $M = 11.24(SD = 4.35)$ and a mean total Difficulties in Emotion Regulation Scale score of $M = 88.65(SD = 18.32)$. Subjective compliance with the fourteen-day actigraphy protocol was exceptional, with participants yielding an average of $M = 12.8(SD = 1.1)$ valid nights of continuous data. Bivariate correlations conducted prior to the primary analyses

indicated that age and socioeconomic status were significantly, albeit weakly, correlated with subjective insomnia severity ($r = 0.12, p < 0.01$ and $r = -0.09, p < 0.05$, respectively), justifying their inclusion as covariates in subsequent models. A comprehensive breakdown of the baseline demographic and clinical metrics is presented in Table 1.

Table 1

Baseline Demographic and Clinical Characteristics of the Sample (N=846)

Characteristic	Total Sample	Males (n = 396)	Females (n = 450)	Statistic	p-value
Age in years, M(SD)	15.42(1.63)	15.51(1.65)	15.34(1.61)	$t = 1.52$	0.129
Socioeconomic Status, n(%)				$\chi^2 = 2.45$	0.293
Low	215(25.41%)	105(26.51%)	110(24.44%)		
Middle	482(56.97%)	220(55.55%)	262(58.22%)		
High	149(17.61%)	71(17.92%)	78(17.33%)		
Insomnia Severity Index, M(SD)	11.24(4.35)	10.85(4.12)	11.58(4.51)	$t = -2.44$	0.014
DERS Total Score, M(SD)	88.65(18.32)	85.34(17.55)	91.56(18.54)	$t = -4.98$	<0.001

To identify the unobservable, novel endophenotypes of adolescent insomnia, Latent Profile Analysis was systematically applied to the objective actigraphy parameters (Total Sleep Time, Sleep Onset Latency, Wake After Sleep Onset, Sleep Efficiency) alongside the subjective Insomnia Severity Index scores. We evaluated models ranging from one to five latent classes. As detailed in Table 2, the model fit indices progressively improved with the extraction of additional profiles. The Akaike Information Criterion and Bayesian Information Criterion both demonstrated a continuous decrease as the number of

profiles increased. However, the Lo-Mendell-Rubin adjusted likelihood ratio test indicated that the extraction of a four-profile model did not yield a statistically significant improvement over the three-profile model ($p = 0.184$). Furthermore, the three-profile model exhibited an excellent entropy value of 0.88, indicating a high degree of classification accuracy and clear boundary separation between the latent classes. Theoretical interpretability and clinical relevance further supported the retention of the three-profile solution as the optimal model for this dataset.

Table 2

Fit Indices for the Latent Profile Analysis Models of Sleep Parameters

Number of Profiles	AIC	BIC	Sample-Size Adjusted BIC	Entropy	LMR-LRT p-value
1Profile	18452.34	18498.12	18465.45	–	–
2Profiles	16234.56	16301.24	16255.88	0.82	<0.001
3Profiles	15102.88	15189.45	15128.91	0.88	<0.001
4Profiles	14988.21	15096.67	15021.44	0.81	0.184
5Profiles	14890.11	15020.45	14925.66	0.76	0.342

Based on the three-profile solution, the sample was stratified into three distinct endophenotypes. Profile 1, designated as the “Normative Sleepers” ($n = 412, 48.69%$), was characterized by objective and subjective sleep metrics within standard healthy ranges, including high sleep efficiency and low insomnia severity. Profile 2, labeled “Sleep Onset Delay Endophenotype” ($n = 258, 30.49%$), exhibited markedly prolonged actigraphy-derived sleep onset latency and moderate subjective insomnia, but

maintained relatively consolidated sleep once initiated. Profile 3, identified as the “High Fragmentation Endophenotype” ($n = 176, 20.80%$), was defined by severe subjective insomnia, significantly elevated wake after sleep onset, severely reduced total sleep time, and the lowest overall sleep efficiency among the cohorts. Analysis of variance revealed highly significant omnibus differences across all objective and subjective sleep parameters among the three endophenotypes. Post-hoc comparisons utilizing

Bonferroni corrections confirmed that the High Fragmentation Endophenotype experienced significantly worse sleep continuity compared to both the Normative

Sleepers and the Sleep Onset Delay Endophenotype (all $p < 0.001$). The specific characteristics defining these derived profiles are comprehensively mapped in Table 3.

Table 3

Objective and Subjective Sleep Characteristics of the Identified Endophenotypes

Sleep Parameter	Normative Sleepers (n = 412)	Sleep Onset Delay (n = 258)	High Fragmentation (n = 176)	F-statistic	p-value
Actigraphy TST (minutes), M(SD)	452.4(35.2)	410.6(42.1)	365.8(48.5)	215.45	<0.001
Actigraphy SOL (minutes), M(SD)	18.5(8.2)	55.4(14.6)	32.1(12.4)	482.12	<0.001
Actigraphy WASO (minutes), M(SD)	22.4(10.5)	28.6(12.1)	78.5(22.3)	560.88	<0.001
Actigraphy SE (%), M(SD)	90.5(4.2)	84.2(5.1)	74.6(6.8)	398.67	<0.001
Insomnia Severity Index, M(SD)	7.4(2.1)	13.5(3.2)	16.8(3.8)	310.24	<0.001

To determine the associations between these identified insomnia endophenotypes and emotional dysregulation, a multivariate analysis of covariance was executed, controlling for age, gender, and socioeconomic status. The model revealed a significant main effect of the insomnia endophenotype on the combined emotional dysregulation subscales (Wilks' $\Lambda = 0.72$, $F(12,1672) = 24.56$, $p < 0.001$, $\eta_p^2 = 0.15$). Univariate follow-up analyses demonstrated that adolescents classified within the High Fragmentation Endophenotype exhibited the most severe impairments across all domains of emotional dysregulation, scoring significantly higher than both Normative Sleepers and those with Sleep Onset Delay in nonacceptance of

emotional responses, impulse control difficulties, and limited access to emotion regulation strategies. Interestingly, while the Sleep Onset Delay group showed elevated difficulties compared to the Normative Sleepers, their specific deficits were predominantly concentrated in lack of emotional clarity and difficulties engaging in goal-directed behavior, suggesting that distinct sleep disturbances may selectively impair different regulatory pathways. The largest effect size was observed for impulse control difficulties ($\eta_p^2 = 0.14$), which was profoundly disrupted in the High Fragmentation group. Table 4 delineates the adjusted marginal means and comparative statistics for the emotional dysregulation facets across the three novel endophenotypes.

Table 4

Association of Insomnia Endophenotypes with Emotional Dysregulation Subscales (MANCOVA)

DERS Subscale	Normative Sleepers	Sleep Onset Delay	High Fragmentation	F-statistic	p-value	Partial η^2
Nonacceptance, M(SE)	11.2(0.4)	14.5(0.5)	19.2(0.6)	45.22	<0.001	0.09
Goals, M(SE)	12.5(0.3)	16.8(0.4)	18.1(0.5)	38.65	<0.001	0.08
Impulse Control, M(SE)	10.8(0.4)	13.2(0.5)	21.4(0.6)	82.14	<0.001	0.14
Awareness, M(SE)	13.4(0.3)	15.1(0.4)	16.5(0.5)	12.88	<0.001	0.03
Strategies, M(SE)	14.2(0.5)	17.6(0.6)	23.8(0.7)	65.41	<0.001	0.12
Clarity, M(SE)	10.1(0.3)	15.5(0.4)	16.2(0.5)	41.05	<0.001	0.08

4. Discussion

The primary objective of the present study was to identify unobservable, novel endophenotypes of adolescent insomnia utilizing a person-centered latent variable modeling approach, and subsequently, to examine how these distinct sleep profiles are differentially associated with specific facets of emotional dysregulation. By integrating continuous objective actigraphy data with subjective self-report metrics,

our Latent Profile Analysis successfully delineated 3 distinct insomnia endophenotypes within the adolescent cohort: Normative Sleepers, Sleep Onset Delay, and High Fragmentation. Furthermore, our multivariate analyses revealed that these derived endophenotypes exhibited profound and highly specific associations with various subdomains of emotional dysregulation. Specifically, adolescents classified within the High Fragmentation profile demonstrated the most severe global impairments in emotion

regulation, particularly in impulse control and nonacceptance of emotional responses, whereas those with the Sleep Onset Delay profile exhibited specialized deficits centered around emotional clarity and goal-directed behavioral engagement. These findings substantiate the hypothesis that insomnia is a heterogeneous construct rather than a monolithic disorder, and that specific architectural disruptions in sleep continuity correspond to distinct phenotypic expressions of psychological vulnerability during adolescence.

The identification of the Sleep Onset Delay and High Fragmentation endophenotypes aligns seamlessly with emerging literature emphasizing the multifaceted nature of sleep disturbances and their varied psychopathological correlates. The profound sleep deprivation and insomnia characterizing adolescence have long been recognized as a critical vulnerability factor for a myriad of mental health implications, often serving as a primary catalyst for the exacerbation of psychiatric symptoms (Uccella et al., 2023). However, our study extends this foundational understanding by demonstrating that the specific nature of the sleep disruption matters considerably. The observation that the Sleep Onset Delay group experienced significant difficulties with emotional clarity and goal-directed behavior resonates with studies highlighting the role of pre-sleep cognitive hyperarousal. Adolescents who struggle to initiate sleep often ruminate, a cognitive process inherently tied to anxiety and emotional instability. Research indicates that excessive cognitive anxiety significantly elevates the occurrence of insomnia during late adolescence (Bao & Bihn, 2021), and that specific personality traits, such as neuroticism, influence sleep continuity precisely through the mediating pathways of perfectionism and maladaptive cognitive emotion regulation strategies (Sohrabian et al., 2020). Furthermore, perfectionism, characterized by a persistent fear of failure and vulnerability to stress, is strongly associated with prolonged sleep onset in youth, often with notable gender variations (Richardson & Gradisar, 2020). The inability to clearly process emotional states (lack of clarity) likely fuels the pre-sleep cognitive arousal that delays sleep onset, establishing a cyclical relationship where delayed sleep further depletes the cognitive resources necessary for goal-directed behavior the following day.

Conversely, the High Fragmentation endophenotype was uniquely associated with catastrophic failures in impulse control and a profound nonacceptance of emotional responses. This group, characterized by extensive wake after sleep onset and severely reduced total sleep time, mirrors the

severe sleep continuity disruptions often observed in complex clinical populations. For example, specific components of insomnia, particularly sleep fragmentation, have been identified as primary drivers of severe emotion dysregulation in individuals with borderline personality disorder (Fitzpatrick et al., 2020). In clinical and non-clinical comparisons, populations suffering from severe, fragmented insomnia exhibit markedly higher levels of generalized emotional dysregulation and experiential avoidance (Zakiei et al., 2020). The profound impact of fragmented sleep on impulse control is likely mediated by the disruption of critical neurobiological processes occurring during consolidated sleep cycles, which are essential for prefrontal cortex functioning and top-down emotional regulation. This severe fragmentation may also explain why persistent insomnia acts as a potent antecedent risk factor for the first lifetime onsets of suicidality, severe anxiety, and major depression during adolescence (Soltani et al., 2023). Unlike adults, the symptom profiles of major depression in adolescents are highly volatile (Rice et al., 2019), and the severe lack of impulse control observed in our High Fragmentation group may be a critical behavioral marker of this volatility.

The pervasive impact of contemporary lifestyle and environmental stressors further contextualizes our findings. The modern adolescent is frequently subjected to persistent digital engagement, with longitudinal relationships established between phenomena like nomophobia, addictive social media use, and subsequent insomnia (Lin et al., 2021). The complex network connecting internet addiction to physical inactivity and suicidal ideation consistently features insomnia as a central, mediating node (Lu et al., 2025). It is plausible that such digital dependencies contribute primarily to the Sleep Onset Delay profile by delaying the circadian phase and elevating physiological arousal prior to bedtime. Furthermore, the role of environmental trauma cannot be understated. Adverse experiences, ranging from the acute trauma of parental death (Farella Guzzo & Gobbi, 2021) to the insidious chronicity of emotional neglect, precipitate severe psychopathology, such as non-suicidal self-injury, through the mediating roles of social anxiety and chronic insomnia (Hou et al., 2023). High daily stress is also intimately linked to maladaptive coping mechanisms, such as emotional eating, which indirectly degrade sleep quality and perpetuate the cycle of emotional and physiological dysregulation (Choi et al., 2021). The structural measurement of these negative emotional states, clearly delineating anxiety, depression, and stress (Lovibond &

Lovibond, 1995), supports the notion that varied emotional burdens interact with sleep architecture in distinct ways. For instance, anxiety and depression mediate the relationship between intrinsic personality traits like conscientiousness and the severity of insomnia symptoms (Akram et al., 2019), while maladaptive cognitive emotion regulation strategies and pre-sleep arousal sequentially mediate the relationship between borderline personality traits and insomnia severity (Park, 2025).

5. Conclusion

The recognition of these distinct endophenotypes strongly supports the necessity for targeted, emotion-focused interventions. While digital therapeutics, such as smartphone-delivered Cognitive Behavioral Therapy for Insomnia, have proven effective for general adolescent sleep disturbances, exploratory studies reveal distinct gender differences in baseline disruption and treatment response (Li et al., 2021), highlighting the need for personalized approaches (Li et al., 2020). Transdiagnostic treatments fundamentally rooted in emotion regulation have demonstrated substantial efficacy in reducing insomnia severity (Hatamian et al., 2023), validating the core premise of our findings. Furthermore, interventions explicitly targeting emotional processing and physiological hyperarousal, ranging from emotion-focused therapies utilized for interpersonal trauma (Boroumandrad 2020) to somatic techniques like Emotional Freedom Techniques for post-traumatic stress (Church et al., 2016), underscore the clinical utility of addressing the affective components of sleep disorders. Interestingly, not all sleep alterations are entirely detrimental; some evidence suggests complex mediations where mild sleep architectural shifts interact with the need for cognition to influence creative output in adolescents (Ren et al., 2024). However, the overarching consensus remains that pre-existing emotion dysregulation is a longitudinal predictor of insomnia, creating a detrimental feedback loop (Jansson-Fröjmark et al., 2015), and the somatic consequences, such as irritable bowel syndrome, significantly impair adolescent quality of life (Yang, 2021). Identifying whether a youth suffers from sleep onset delay versus high fragmentation allows clinicians to predict specific emotional vulnerabilities and deploy precise therapeutic mechanisms.

6. Limitations & Suggestions

Several limitations must be acknowledged when interpreting the results of this investigation. First, the cross-sectional nature of the study design precludes the ability to establish definitive causal relationships between the identified insomnia endophenotypes and the specific facets of emotional dysregulation. While the theoretical framework posits a bidirectional relationship, longitudinal data tracking individual trajectories over time would be required to determine whether specific sleep architectural disruptions precede the onset of isolated emotional deficits, or vice versa. Second, although actigraphy provides a highly ecologically valid and continuous objective measure of sleep parameters in the natural home environment, it is inherently limited in its ability to capture true sleep micro-architecture. Actigraphy relies on motor activity algorithms and cannot quantify specific sleep stages, such as rapid eye movement or slow-wave sleep, which are deeply implicated in emotional processing and memory consolidation. The reliance on this method may have obscured deeper neurophysiological endophenotypes that are only detectable via gold-standard polysomnography. Finally, the sample was exclusively drawn from urban and semi-urban educational districts within Egypt. While this provides valuable data on an underrepresented non-Western adolescent population, the unique cultural, socioeconomic, and educational stressors specific to this geographic region may limit the generalizability of the identified latent profiles to diverse global populations with differing cultural attitudes toward sleep and emotion expression.

Future research should prioritize longitudinal and prospective study designs to unravel the chronological sequence and causal mechanisms linking specific sleep phenotypes to emotional trajectories across the critical developmental window of adolescence. Methodologically, subsequent studies would benefit immensely from integrating ambulatory electroencephalography or portable polysomnography alongside actigraphy. Capturing true sleep architecture, including spectral power analysis and sleep spindle density, could refine the latent profiles identified in this study, potentially revealing highly specialized neurobiological endophenotypes of insomnia. Furthermore, future investigations should incorporate robust ecological momentary assessment techniques to capture real-time, day-to-day fluctuations in emotional reactivity and regulation strategies, mapping these micro-level emotional shifts directly onto the preceding night's precise

sleep parameters. Expanding the demographic scope to include cross-cultural comparisons would also elucidate whether the High Fragmentation and Sleep Onset Delay endophenotypes represent universal biological phenotypes or whether their expression and emotional correlates are heavily moderated by cultural and environmental contexts. Finally, future trials must focus on treatment matching, evaluating whether adolescents with distinct sleep profiles exhibit differential response rates to specific therapeutic modalities.

The identification of distinct insomnia endophenotypes carries significant practical implications for clinical and educational settings. Practitioners should move beyond generalized assessments of sleep duration and adopt a more granular approach, routinely screening for specific architectural disruptions such as prolonged sleep onset versus mid-night fragmentation. For adolescents exhibiting the Sleep Onset Delay profile, school psychologists and clinicians should prioritize interventions that target pre-sleep cognitive hyperarousal and enhance emotional clarity, such as mindfulness-based cognitive therapy or specialized cognitive restructuring focused on anxiety reduction. Conversely, adolescents presenting with the High Fragmentation profile may require more intensive, transdiagnostic interventions targeting severe impulse control deficits and distress tolerance skills, alongside rigorous stimulus control therapies to consolidate sleep. In educational environments, recognizing that varied sleep disruptions manifest as specific behavioral issues—such as lack of goal-directed focus versus overt impulsivity—can aid educators in developing tailored classroom accommodations and targeted socio-emotional learning curricula. Ultimately, aligning specific behavioral and emotional interventions with a patient’s precise sleep endophenotype holds the promise of significantly improving treatment efficacy and mitigating the long-term developmental consequences of adolescent sleep disorders.

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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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