Sleep Medicine 144 (2026) 108947

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Internalizing and externalizing symptoms association with insomnia, SRMDs, and parasomnias in an ADHD sample and moderating effects of parental characteristics☆

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ARTICLE INFO

Keywords:
ADHD
Internalizing
Externalizing
Sleep problems
Parent emotions
Parenting strategies

ABSTRACT

Objective: Sleep symptoms are highly prevalent among youth with ADHD, yet their heterogeneity and links to co-occurring emotional and behavioral symptoms remain poorly understood. This study examined, within an ADHD sample, how internalizing and externalizing symptoms were associated with sleep symptoms reflecting insomnia, sleep-related movement disorder (SRMD), and parasomnias. It also examined the moderating role of parents' emotions and strategies.

Methods: Parents of a national sample of 1,086 children and adolescents (ages 5–18) with ADHD completed validated measures of sleep symptoms, internalizing/externalizing symptoms, parenting practices, and parental emotions. Sleep outcomes were analyzed using binomial generalized linear models. Additional mediation and moderation models were conducted.

Results: Internalizing symptoms were the strongest predictor of all sleep outcomes, whereas externalizing symptoms showed more selective associations, emerging mainly for SRMD and parasomnia symptoms. Mediation analyses were consistent with an indirect association of externalizing symptoms with insomnia through internalizing symptoms, whereas for SRMD and parasomnia symptoms both indirect and residual direct associations were observed. Parenting effects were selective: positive parenting attenuated the association between externalizing and SRMD symptoms, whereas negative parenting strengthened the association between internalizing and SRMD symptoms. Positive parental emotion attenuated the association between externalizing and parasomnia symptoms but strengthened the association between internalizing and parasomnia symptoms.

Conclusion: These results are encouraging in suggesting a transdiagnostic approach aimed to better understanding the mechanisms underlying their relationship and to pave the way for the development of more refined and novel therapeutic approaches.

1. Introduction

Sleep problems are one of the most common disorders among children and adolescents with ADHD, Using objective and/or subjective measures, previous research in the area have primarily demonstrated that children with ADHD report higher rates of sleep patterns (e.g., reduced sleep duration) and sleep disturbances than the general population [1–3]. According to these studies, sleep problems show a prevalence ranging from 55% to 74% in the youth with ADHD [4–6].

The most accepted explanation for the association between ADHD and sleep problems is that the brain areas involved in executive functions and self-regulation processes, already impaired in individuals with ADHD, are especially vulnerable to the effects of sleep deprivation [7,8] and sleep disorders [9] Considering the interrelations between daytime and nighttime problems in patients with ADHD, Becker [10] proposed viewing ADHD as a 24-h condition and urged researchers to develop

☆ The data that support the findings are not publicly available due to ethical restrictions.

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https://doi.org/10.1016/j.sleep.2026.108947
Received 26 January 2026; Received in revised form 31 March 2026; Accepted 1 April 2026
Available online 1 April 2026

integrated models of dysfunctional daytime-sleep-related problems in ADHD functioning dynamically intensify each other.

A crucial aspect that requires attention is that ADHD rarely presents in isolation; it often occurs alongside a wide range of comorbid psychiatric and neurodevelopmental disorders [11–14]. Although the association between ADHD and sleep problems is well established, the specific contribution of co-occurring internalizing and externalizing symptoms to this association remains unclear [15,16]. Because internalizing (e.g., anxiety, depression) or externalizing (e.g., oppositional/defiant, conduct) behaviors drive the social, interpersonal, and academic impairments central to sleep-disorder diagnoses (DSM-5), clarifying their role is essential to understand the ADHD–sleep relationship.

Over the past decade, an increasing number of studies have investigated how co-occurring internalizing and externalizing symptoms in youth with ADHD relate to sleep problems [16–19]. These consistently show that adolescents with ADHD who experience sleep problems tend to display higher levels of internalizing (emotional problems) and externalizing (behavioral problems) comorbidities than those without problems [16]. Some findings suggest that, mainly, internalizing symptoms may moderate the link between ADHD and sleep problems, influencing how these difficulties interact and persist over time [16]. In addition, specific dimensions of ADHD (i.e., inattention versus hyperactivity/impulsivity) may contribute differently to sleep problems depending on the presence and type of co-occurring internalizing or externalizing symptoms [18]. Despite growing interest, integrated models that jointly test internalizing–externalizing interactions across distinct sleep domains—insomnia, sleep-related movement disorder (SRMD), and parasomnia—within the same sample are essentially absent. Methodological inconsistencies across studies, such as differences in assessment tools, statistical methods and sample characteristics, limit the comparability and generalizability of findings. Investigating these associations within a single, well-characterized sample improves internal consistency, reduces confounding, and allows for a more accurate analysis of how internalizing and externalizing symptoms relate to different sleep symptoms in ADHD.

Overall, critical questions remain unanswered. In particular, it is unclear how internalizing and externalizing symptoms, both independently and in combination, relate to distinct sleep symptom indicators within ADHD. Clarifying these associations may help prioritize assessment of sleep complaints in youths with differing symptom burdens, especially given evidence that co-occurring internalizing and externalizing difficulties are linked to greater functional impairment [19]. Therefore, our first research question (RQ1) is: Are internalizing and externalizing symptoms associated with symptom indicators for insomnia, SRMD, parasomnia, and is their co-occurrence related to higher levels of these sleep symptoms?

Although most studies have reported associations between both internalizing and externalizing symptoms with sleep problems, longitudinal research has highlighted differential patterns in these associations [20]. Examining the interaction between internalizing and externalizing symptoms in relation to phenotypic features reflecting distinct sleep symptoms offers a crucial contribution to the field. If externalizing behaviors impact sleep primarily through underlying internal distress, interventions should shift from solely focusing on behavior management to also addressing anxiety, rumination, and mood regulation. Thus, our second research question (RQ2) is: Does internalizing symptomatology differently mediate the relationship between externalizing problems and sleep symptom indicators for insomnia, SRMD, and parasomnia?

Parenting is a modifying contextual factor the internalizing and externalizing [21,22]. Supportive practices may buffer, and punitive practices may amplify, the impact of child symptoms on sleep by altering pre-sleep arousal and bedtime routines. Likewise, parental emotional climate could either soothe or escalate children's internal arousal, shaping how daytime problems spill into the night. Mapping these moderating effects can pinpoint family-level levers for intervention and explain mixed findings in the ADHD-sleep literature regarding parent-child dynamics. Therefore, our third research question (RQ3) is: Do parental emotions (positive and negative) and parenting strategies (supportive and punitive) differently moderate the associations between behavioral symptoms (internalizing and externalizing) and sleep symptom indicators for insomnia, SRMD, parasomnia?

2. Methods

2.1. Participants

Parents of 1,086 children and adolescents with a certified ADHD diagnosis completed the survey about their child (M_{age} = 11.47 years, SD = 3.17; range = 5–18). The child sample was predominantly male (n = 925, 85.2%), with females representing 14.8% (n = 161). Respondents were geographically distributed across all Italian regions, 20 metropolitan cities, and 78.3% (72/92) of provinces. The cohort was clinically heterogeneous: 11 different psychiatric or neurodevelopmental comorbid diagnoses were represented, and 90.8% of participants had at least one comorbidity; specifically, 47.4% had one comorbidity and 43.4% had two or more. Medication was prescribed for 438 patients (40.4%); of these, 70.3% received Methylphenidate, while 26.7% were treated with other agents, including atomoxetine, antipsychotics, and mood stabilizers. Treatment details were not specified by 2.2% of caregivers.

Bedtime was typically early: 20:00–22:00 in 760 children (70.0%), 22:00–23:00 in 233 (21.5%), 23:00–24:00 in 64 (5.9%), and after 24:00 in 29 (2.7%). Reported nightly sleep duration was <6 h in 43 participants (4.0%), 6–7 h in 240 (22.1%), 8–9 h in 644 (59.3%), 10–11 h in 151 (13.9%), and ≥12 h in 8 (0.7%).

2.2. Procedure

Data were collected between June and September 2020 via an anonymous online survey, distributed through the official website of the National ADHD Family Association. Including emotional-behavioral functioning, sleep problems, parental emotions, and parenting practices, with separate parent reports referring to the periods before and during the COVID-19 pandemic. In the present study, only ratings referring to the pre-COVID period were analyzed. Methods of data collection and storage, as well as other analyses based on this broader survey dataset, have been described in previous publications [23–26].

Parents of children and adolescents with ADHD aged 5 to 18 years were invited to complete the survey. Written informed consent was obtained from all participants prior to survey completion.

Participants were followed by Child and Adolescent Mental Health Services and had received a certified ADHD diagnosis in line with the official Italian ADHD guidelines and the protocols established by the Italian National Institute of Health. No exclusion criteria were applied. No financial compensation was provided for participation. The study received ethical approval from the Ethics Committee of the Department of Developmental and Social Psychology at Sapienza University and was conducted in accordance with the ethical standards of the 2008 Declaration of Helsinki.

2.3. Measures

2.3.1. Sleep symptom indicators

Sleep symptoms were assessed using a reduced set of 10 items from the Sleep Disturbance Scale for Children (SDSC) [27] selected to index three domains: insomnia, SRMD, and parasomnia symptoms. Insomnia symptoms were measured using three items (i.e., difficulty falling asleep, anxious/agitated behavior at bedtime, and frequent nocturnal awakenings). SRMD symptoms were assessed through four items capturing motor restlessness, and involuntary movements during sleep

(i.e., hypnic jerks at sleep onset; rhythmic movements to initiate sleep; restless sleep; and sleep bruxism). Parasomnia symptoms were measured with three items describing disorders of arousal (i.e., *pavor nocturnus*, sleepwalking, and nightmares). The SDSC items assessing sleep breathing disorders, hypnagogic hallucinations, sleep hyperhidrosis, and excessive somnolence were not administered because these domains were not part of the present study. Each symptom was coded dichotomously (0 = *Absent*, 1 = *Present*) by the parents to capture the child's symptom endorsement rather than frequency-based severity [28]. Given the binary nature of the indicators, inter-item associations were examined using polychoric correlations, and the hypothesised three-factor structure was tested via confirmatory factor analysis using a WLSMV estimator appropriate for binary data [29]. The three-factor model showed excellent fit (CFI = 0.977, TLI = 0.968, RMSEA = 0.017, SRMR = 0.032). Internal consistency was acceptable for insomnia (α = .70; ω = 0.70) and SRMD (α = .73; ω = 0.76). It was modest for parasomnia symptoms (α = .60; ω = 0.66), likely reflecting the small number of dichotomous items and behavioral heterogeneity. Accordingly, associations involving parasomnia symptoms may be attenuated by measurement error and should be interpreted conservatively. For each domain, the outcome was specified as the number of endorsed items (k) out of the total number of items in the domain (n).

2.3.2. Internalizing and externalizing symptoms

Internalizing and externalizing symptoms were assessed using a selected subset of items from the Child Behavior Checklist (CBCL) [30,31]. Items were chosen to capture core anxiety/depressive and oppositional/aggressive behaviors that are particularly relevant in ADHD populations, while minimizing redundancy and overlap across symptom dimensions. Internalizing symptoms were measured using four items (e.g., worry-fear, sadness, lack of interest, and boredom). Externalizing symptoms were measured using seven items (e.g., temper tantrums, anger, arguments, verbal and physical aggression). Parents answered using a three-point scale indicating weekly frequency (*1–2 times*; *3–4 times*; *5–7 times per week*). Internal consistency was acceptable for internalizing behaviors (α = .65, ω = 0.67) and good for externalizing behaviors (α = .87, ω = 0.89). CFA supported a two-factor structure with excellent model fit (CFI = 0.992, TLI = 0.990, RMSEA = 0.035, SRMR = 0.043). Total scores for each subscale were calculated by averaging the items.

2.3.3. Parenting strategies

Parenting practices were assessed using a selected subset of items from the Alabama Parenting Questionnaire (APQ) [32], chosen to capture the positive/supportive and negative/punitive dimensions most relevant to the study aims. Positive parenting was assessed with six items reflecting warmth, support, and preventive regulatory practices (e.g., "I listen to his/her requests and try to reassure him/her"; "I stay calm and try to find a compromise"). Negative parenting was assessed with five items reflecting harsh, dysregulated, or disengaged practices (e.g., "I scream," "I lose control," "I punish him/her excessively"). Parents answered using a binary format (0 = *No*, 1 = *Yes*). Both subscales demonstrated good internal consistency (α = .78, ω = 0.82 for positive parenting; α = .78, ω = 0.85 for negative parenting). CFA confirmed the hypothesised two-factor model with adequate fit indices (CFI = 0.930, TLI = 0.910, RMSEA = 0.050, SRMR = 0.047). Total scores for each subscale were calculated by averaging the item.

2.3.4. Parents' emotions

Parental emotional responses to their child's behavior were assessed through a checklist of positive and negative emotions. The checklist was conceptually informed by widely used measures of parental stress and emotion, including the Parenting Stress Index–Short Form (PSI-SF; [33]) and the Parental Stress Scale (PSS; [34]), but was adapted to capture core emotional states rather than full situational statements. Positive emotions included three items (e.g. optimistic and hopeful feelings, empathic, loving) while negative emotions were assessed using ten items related to distressing state (e.g., "I feel: frustrated, angry, worried, powerless, guilty"). Parents used a binary response format for each emotion. Internal consistency was adequate for positive emotions (α = .64, ω = 0.70) and excellent for negative emotions (α = .88, ω = 0.90). CFA supported a two-factor structure with good fit (CFI = 0.949, TLI = 0.938, RMSEA = 0.047, SRMR = 0.052), and scores for each domain were computed as the mean of the items.

2.4. Statistical analyses

The survey and the R script are available on OSF (https://osf.io/ry7wz). Statistical analyses were performed in R (v4.3.2; [35]). Descriptive statistics were computed for all key variables. Normality was evaluated by |skew|<1 or |kurtosis|<3 [36]. Pearson's r (continuous), Spearman's ρ (ordinal), or point-biserial (binary) correlations were computed according to variables type.

Primary analyses for RQ1 treated internalizing and externalizing symptoms as continuous predictors. We modelled each outcome as a binomial response using generalized linear models with a logit link. For each outcome, we fitted a full model including internalizing symptoms, externalizing symptoms, and their interaction (internalizing × externalizing), adjusting for age, sex, bedtime, and sleep duration. Although conceptually related to insomnia, bedtime and sleep duration were included as covariates to account for general sleep schedule variability. The interaction term was retained for completeness but was interpreted only if it reached statistical significance; otherwise, inference focused on the main effects. Effects are reported as odds ratios (ORs) with 95% confidence intervals. For descriptive purposes, we also derived four symptom configurations using median splits on internalizing and externalizing scores (low symptoms, internalizing-only, externalizing-only, and mixed) to illustrate patterns of co-occurrence. Group differences in each sleep domain were examined with binomial models identical in structure to those above, replacing the continuous symptom predictors with the four-level configuration factor and retaining the same covariates. Pairwise comparisons were Bonferroni-adjusted and are reported as odds ratios with 95% confidence intervals.

For RQ2, we examined whether internalizing symptoms statistically mediated the association between externalizing symptoms and each sleep outcome using bootstrap-based mediation within the counterfactual framework implemented in the R package *mediation* [37]. The mediator model regressed internalizing symptoms on externalizing symptoms while adjusting for age and sex. The outcome models adopted the same binomial specification described above for each sleep domain and included both externalizing and internalizing symptoms alongside the covariates. Indirect (ACME), direct (ADE), and total effects were estimated via 5,000 nonparametric bootstrap resamples with percentile 95% confidence intervals; indirect effects were considered supported when the confidence interval excluded zero. Given the cross-sectional design, these mediation estimates were interpreted as indirect associations consistent with the hypothesised model rather than evidence of temporal ordering or causal mechanisms.

For the moderation analyses (RQ3), sleep outcomes were treated as symptom-endorsement counts within each domain. Accordingly, associations were estimated using generalized linear models with a binomial response and a logit link. Each model included centred internalizing and externalizing symptom scores, age, sex, bedtime, and sleep duration. Moderation was tested by adding one parental factor at a time (positive/negative parenting; positive/negative parental emotion) together with the corresponding two product terms (internalizing × moderator; externalizing × moderator), to minimize collinearity and preserve interpretability. Effects are reported as ORs with 95% confidence intervals. When a significant interaction was detected, it was probed via simple-slope analyses by estimating the conditional association of the focal symptom predictor at low (−1 SD), mean, and high (+1 SD) levels of the moderator using marginal trend estimation. Conditional effects

were expressed on the log-odds scale and exponentiated to ORs. Given the number of moderation tests, we controlled multiplicity using the Benjamini–Hochberg false discovery rate (FDR) with FDR-$p < .05$ as the threshold for statistical significance.

3. Results

Descriptive statistics are reported in Table S1 and correlations in Table S2 of the Supplementary Materials. Domain-specific distributional characteristics indicated substantial floor effects, with zero endorsements observed in 62.1% of insomnia, 51.7% of SRMD, and 80.6% of parasomnia symptom indices. Insomnia symptoms correlated with SRMD, $r = 0.32$, $p < .001$, and with parasomnia, $r = 0.35$, $p < .001$; SRMDs and parasomnia were likewise moderately associated, $r = 0.35$, $p < .001$. Internalizing and externalizing symptoms were strongly associated ($r = 0.50$, $p < .001$).

3.1. Associations of internalizing and externalizing symptoms with sleep symptoms

The binomial regression models showed that, the internalizing × externalizing interaction did not improve model fit for insomnia (ΔDeviance = 1.35, p = .245), SRMD (ΔDeviance = 1.31, p = .252), or parasomnia symptoms (ΔDeviance = 2.75, p = .097). Internalizing symptoms were positively associated with all outcomes (insomnia: OR = 1.46, 95% CI [1.29, 1.65], $p < .001$; SRMD symptoms: OR = 1.31, 95% CI [1.18, 1.45], $p < .001$; parasomnia symptoms: OR = 1.44, 95% CI [1.24, 1.67], $p < .001$). Externalizing symptoms were unrelated to insomnia (OR = 1.10, 95% CI [0.97, 1.25], $p = .130$) but were associated with SRMD symptoms (OR = 1.18, 95% CI [1.06, 1.30], $p = .002$) and parasomnia symptoms (OR = 1.26, 95% CI [1.08, 1.47], $p = .004$). Full model estimates are reported in Table 1.

To complement these continuous models and aid clinical interpretability, we compared sleep symptom endorsement across four descriptive symptom configurations defined via median splits on internalizing (Mdn = 0.50) and externalizing (Mdn = 0.86) scores: *low-symptoms* ($n = 286$), *only-externalizing* ($n = 142$), *only-internalizing* ($n = 196$), and *mixed* ($n = 462$). Because the median-split configurations were included to aid clinical interpretability, we treat them as descriptive summaries of common co-occurrence patterns. Overall, the pattern of group differences remained coherent across models (Table S3).

For insomnia symptoms, the mixed configuration showed higher odds of endorsement than low symptoms (OR = 2.11, $p < .001$) and externalizing-only (OR = 2.27, $p < .001$). Internalizing-only also exceeded low symptoms (OR = 1.53, $p = .034$). No other contrasts remained significant after correction, including mixed versus internalizing-only (OR = 1.38, $p = .078$) and low symptoms versus externalizing-only (OR = 1.08, $p = 1.00$).

For SRMD symptoms, the mixed configuration again showed higher odds than low symptoms (OR = 2.47, $p < .001$) and externalizing-only (OR = 1.67, $p < .001$). Internalizing-only also exceeded low symptoms (OR = 1.93, $p < .001$). Remaining comparisons were not significant after correction, including mixed versus internalizing-only (OR = 1.28, $p = .131$) and low symptoms versus externalizing-only (OR = 1.48, $p = .058$).

For parasomnia symptoms, the mixed configuration showed markedly higher odds than low symptoms (OR = 3.12, $p < .001$) and exceeded externalizing-only (OR = 1.87, $p = .035$). Internalizing-only exceeded low symptoms (OR = 2.45, $p = .001$). Externalizing-only also showed higher odds than low symptoms (OR = 1.67, $p = .004$). No other contrasts were significant after correction, including mixed versus internalizing-only (OR = 1.27, $p = .999$) and externalizing-only versus internalizing-only (OR = 0.68, $p = .832$).

3.2. Mediation analyses

For the mediation models (RQ2), the indirect effect through internalizing symptoms was statistically reliable.

For insomnia symptoms, the average indirect effect (ACME) was 0.05 (95% CI 0.04–0.07, $p < .001$), whereas the average direct effect (ADE) was not significant (0.03, 95% CI −0.005–0.06, $p = .089$). The total effect was 0.081 (95% CI 0.052–0.11, $p < .001$), and the estimated proportion mediated was approximately 0.64 (95% CI 0.39–1.08, $p < .001$), consistent with a pattern in which the association between externalizing symptoms and insomnia symptom endorsement was primarily indirect via internalizing symptoms.

For SRMD symptoms, both the indirect effect (ACME average = 0.04, 95% CI 0.02–0.05, $p < .001$) and the direct effect (ADE average = 0.05, 95% CI 0.02–0.07, $p = .003$) were significant. The total effect was 0.09 (95% CI 0.06–0.11, $p < .001$), and the proportion mediated was approximately 0.44 (95% CI 0.26–0.73, $p < .001$), indicating a partial indirect association through internalizing symptoms alongside a residual direct association of externalizing symptoms with SRMD endorsement. For parasomnia symptoms, both the indirect effect (ACME average = 0.02, 95% CI 0.01–0.04, $p < .001$) and the direct effect (ADE average = 0.03, 95% CI 0.004–0.04, $p = .027$) were significant. The total effect was 0.05 (95% CI 0.0302–0.0681, $p < .001$), with an estimated proportion mediated of approximately 0.47 (95% CI 0.25–0.87, $p < .001$), again supporting a partial indirect association whereby internalizing symptoms accounted for a substantive portion of the externalizing–parasomnia link.

3.3. Moderation by parental emotion and strategies

Full model outputs are provided in the Supplementary Materials (Tables S4, S5, and S6).

Table 1
Binomial regression models predicting sleep symptom endorsement from internalizing and externalizing symptoms.

	Insomnia Symptoms			SRMD Symptoms			Parasomnia Symptoms		
Term	OR	OR 95%CI	p	OR	OR 95%CI	p	OR	OR 95%CI	p
Internalizing Symptoms	1.46	1.29 - 1.65	<0.001	1.31	1.18 - 1.45	<0.001	1.44	1.24 - 1.66	<0.001
Externalizing Symptoms	1.10	0.97 - 1.25	0.126	1.18	1.06 - 1.30	0.002	1.26	1.08 - 1.48	0.003
Age	0.93	0.90 - 0.97	0.001	0.92	0.89 - 0.96	<0.001	0.93	0.87 - 0.98	0.010
Sex [Male]	0.93	0.70 - 1.25	0.642	0.97	0.75 - 1.26	0.812	0.72	0.50 - 1.03	0.075
Bedtime [22:00 - 23:00]	1.37	1.02 - 1.84	0.036	1.19	0.93 - 1.51	0.158	0.74	0.48 - 1.14	0.172
Bedtime [23:00 - 24:00]	1.59	1.00 - 2.50	0.048	1.06	0.66 - 1.72	0.810	0.99	0.48 - 2.04	0.989
Bedtime [After 24:00]	2.04	1.20 - 3.49	0.009	1.49	0.85 - 2.61	0.164	0.49	0.14 - 1.75	0.273
Sleep Time [6-7 h]	0.54	0.36 - 0.81	0.003	0.63	0.39 - 1.02	0.062	0.50	0.25 - 0.98	0.043
Sleep Time [8-9 h]	0.29	0.19 - 0.44	<0.001	0.59	0.37 - 0.94	0.026	0.53	0.28 - 1.02	0.058
Sleep Time [10-11 h]	0.30	0.18 - 0.49	<0.001	0.65	0.39 - 1.10	0.112	0.56	0.28 - 1.13	0.106
Sleep Time [> than 12 h]	0.31	0.11 - 0.88	0.028	0.84	0.33 - 2.11	0.711	0.22	0.02 - 2.23	0.200
Internalizing Symptoms x Externalizing Symptoms	0.95	0.87 - 1.04	0.290	0.96	0.89 - 1.04	0.305	0.91	0.81 - 1.03	0.123

Notes. OR = Odds Ratio.

For insomnia symptoms, no moderation effects were supported. None of the interaction terms reached significance after FDR correction (Table 2; all FDR-p > .521). In these same models, internalizing symptoms showed a consistent positive main effect (OR range = 1.65–1.78; all FDR-p < .001), whereas externalizing symptoms did not show a reliable main effect (OR range = 1.04–1.13; all FDR-p ≥ .256). Among moderators, negative parental emotion (OR = 1.92, 95% CI 1.23–2.98, FDR-p = .015) and positive parenting (OR = 1.75, 95% CI 1.23–2.51, FDR-p = .010) showed positive main effects, while the remaining moderator main effects were not significant after FDR correction.

In the SRMD symptoms models (Table 3), moderation emerged selectively for parenting strategies, whereas parental emotions did not show reliable moderating effects after FDR correction. Positive parenting buffered the association between externalizing symptoms and SRMD endorsement (OR = 0.57, FDR-p = .022). Simple-slope probing indicated that externalizing symptoms predicted higher SRMD endorsement when positive parenting was low (slope = 0.364, 95% CI 0.192–0.535; OR = 1.44, 95% CI 1.21–1.71) and at the mean level (0.202, 95% CI 0.084–0.320; OR = 1.22, 95% CI 1.09–1.38), but not when positive parenting was high (0.040, 95% CI −0.118–0.198; OR = 1.04, 95% CI 0.89–1.22). Conversely, negative parenting amplified the association between internalizing symptoms and SRMD endorsement (OR = 1.99, FDR-p = .036). Internalizing symptoms were not reliably related to SRMDs under low negative parenting (0.197, 95% CI −0.008–0.403; OR = 1.22, 95% CI 0.99–1.50), but were positively related at mean (0.366, 95% CI 0.225–0.507; OR = 1.44, 95% CI 1.25–1.66) and high levels (0.534, 95% CI 0.356–0.713; OR = 1.71, 95% CI 1.43–2.04). Across SRMD models, both internalizing and externalizing symptoms showed positive main effects (all FDR-p ≤ .044). Positive emotions (OR = 1.72, FDR-p < .001) and parenting (OR = 1.73, FDR-p = .002) showed significant positive associations with SRMD symptoms.

In the parasomnia symptoms models (Table 4), moderation was concentrated in the positive emotion and positive parenting models. Positive parental emotion showed a dual moderating pattern: it buffered the externalizing–parasomnia association (OR = 0.24, FDR-p < .001) and strengthened the internalizing–parasomnia association (OR = 2.58, 95% FDR-p = .038). Simple slopes indicated that externalizing symptoms predicted parasomnia endorsement when positive emotion was low (slope = 0.682, 95% CI 0.420–0.945; OR = 1.98) and at the mean (0.260, 95% CI 0.061–0.458; OR = 1.30), but not when positive emotion was high (−0.162, 95% CI −0.460–0.136; OR = 0.85). Conversely, internalizing symptoms were unrelated to parasomnias at low positive emotion (0.206, 95% CI −0.095–0.507; OR = 1.23), but were positively associated at the mean (0.486, 95% CI 0.265–0.708; OR = 1.63) and high levels (0.766, 95% CI 0.443–1.090; OR = 2.15). Positive parenting showed a positive main effect on parasomnia symptoms (OR = 2.41, FDR-p = .006) and, more importantly, attenuated the externalizing–parasomnia symptoms link (OR = 0.44, FDR-p = .038).

Table 2
Moderated binomial regressions predicting insomnia symptoms from internalizing/externalizing symptoms with parental emotions/strategies as moderators.

	Insomnia Symptoms		
Predictors	OR	OR 95% CI	*FDR-p*
Negative Emotions as Moderator			
Internalizing Issues	1.77	1.51 - 2.09	<0.001
Externalizing Issues	1.04	0.89 - 1.21	0.811
Negative Emotion	1.92	1.23 - 2.98	0.015
Internalizing x Negative emotion	0.79	0.44 - 1.43	0.647
Externalizing x Negative emotion	0.87	0.49 - 1.53	0.810
Positive Emotions as Moderator			
Internalizing Issues	1.78	1.51 - 2.1	<0.001
Externalizing Issues	1.10	0.95 - 1.27	0.363
Positive emotion	0.94	0.67 - 1.31	0.842
Internalizing x Positive emotion	1.25	0.7 - 2.23	0.677
Externalizing x Positive emotion	0.77	0.47 - 1.28	0.521
Negative Strategies as Moderator			
Internalizing Issues	1.76	1.5 - 2.07	<0.001
Externalizing Issues	1.13	0.97 - 1.3	0.256
Negative strategies	0.99	0.64 - 1.52	0.971
Internalizing x Negative strategies	0.81	0.43 - 1.53	0.751
Externalizing x Negative strategies	0.91	0.51 - 1.62	0.868
Positive Strategies as Moderator			
Internalizing Issues	1.65	1.4 - 1.95	<0.001
Externalizing Issues	1.11	0.96 - 1.28	0.311
Positive strategies	1.75	1.23 - 2.51	0.010
Internalizing x Positive strategies	1.28	0.76 - 2.16	0.549
Externalizing x Positive strategies	0.81	0.51 - 1.3	0.586

Table 3
Moderated binomial regressions predicting SRMD symptoms from internalizing/externalizing symptoms with parental emotions/strategies as moderators.

	SRMD Symptoms		
Predictors	OR	OR 95% CI	*FDR-p*
Negative Emotions as Moderator			
Internalizing Issues	1.45	1.26 - 1.67	<0.001
Externalizing Issues	1.18	1.03 - 1.34	0.044
Negative Emotion	1.49	1.02 - 2.16	0.110
Internalizing x Negative emotion	1.33	0.8 - 2.21	0.465
Externalizing x Negative emotion	0.78	0.48 - 1.26	0.511
Positive Emotions as Moderator			
Internalizing Issues	1.43	1.25 - 1.65	<0.001
Externalizing Issues	1.26	1.12 - 1.42	0.001
Positive emotion	1.72	1.31 - 2.25	<0.001
Internalizing x Positive emotion	0.67	0.42 - 1.08	0.232
Externalizing x Positive emotion	1.21	0.81 - 1.81	0.553
Negative Strategies as Moderator			
Internalizing Issues	1.44	1.25 - 1.66	<0.001
Externalizing Issues	1.22	1.08 - 1.38	0.007
Negative strategies	1.19	0.83 - 1.71	0.541
Internalizing x Negative strategies	1.99	1.17 - 3.4	0.036
Externalizing x Negative strategies	0.64	0.39 - 1.04	0.172
Positive Strategies as Moderator			
Internalizing Issues	1.46	1.27 - 1.68	<0.001
Externalizing Issues	1.22	1.09 - 1.38	0.004
Positive strategies	1.73	1.28 - 2.33	0.002
Internalizing x Positive strategies	0.69	0.44 - 1.09	0.254
Externalizing x Positive strategies	0.57	0.38 - 0.85	0.022

Table 4
Moderated binomial regressions predicting parasomnia symptoms from internalizing/externalizing symptoms with parental emotions/strategies as moderators.

	Parasomnia Symptoms		
Predictors	OR	OR 95% CI	*FDR-p*
Negative Emotions as Moderator			
Internalizing Issues	1.65	1.32 - 2.06	<0.001
Externalizing Issues	1.23	0.99 - 1.52	0.151
Negative Emotion	1.30	0.67 - 2.47	0.647
Internalizing x Negative emotion	0.90	0.41 - 1.99	0.880
Externalizing x Negative emotion	1.18	0.54 - 2.55	0.833
Positive Emotions as Moderator			
Internalizing Issues	1.63	1.3 - 2.03	<0.001
Externalizing Issues	1.30	1.06 - 1.58	0.034
Positive emotion	1.58	0.97 - 2.54	0.156
Internalizing x Positive emotion	2.58	1.23 - 5.46	0.038
Externalizing x Positive emotion	0.24	0.12 - 0.47	<0.001
Negative Strategies as Moderator			
Internalizing Issues	1.63	1.3 - 2.04	<0.001
Externalizing Issues	1.30	1.06 - 1.59	0.036
Negative strategies	0.87	0.45 - 1.65	0.833
Internalizing x Negative strategies	1.16	0.51 - 2.66	0.853
Externalizing x Negative strategies	1.29	0.58 - 2.81	0.753
Positive Strategies as Moderator			
Internalizing Issues	1.52	1.2 - 1.91	0.002
Externalizing Issues	1.35	1.11 - 1.64	0.013
Positive strategies	2.41	1.42 - 4.11	0.006
Internalizing x Positive strategies	1.16	0.56 - 2.37	0.833
Externalizing x Positive strategies	0.44	0.23 - 0.84	0.038

Externalizing symptoms were associated with parasomnias at low (slope = 0.533, 95% CI 0.238–0.828; OR = 1.70) and mean positive parenting (0.297, 95% CI 0.101–0.493; OR = 1.35), but not at high levels (0.061, 95% CI −0.180–0.302; OR = 1.06). Models with negative parental emotion or negative parenting showed no significant interactions after FDR correction.

4. Discussion

This study examined how internalizing and externalizing symptoms relate to three sleep symptoms domains (i.e., insomnia, SRMDs and parasomnia symptoms) and how parental characteristics moderated their association, in a large Italian cohort of youth with ADHD. Across complementary analytic approaches, the findings converged on internalizing distress as the most consistent correlate of sleep symptoms endorsement, while parenting practices and emotions modified selected links involving externalizing and internalizing symptoms. Overall, these results support an integrated "24-h" perspective on ADHD, in which daytime emotional-behavioral difficulties and nocturnal symptoms co-occur in systematic ways, and they motivate the evaluation of interventions that address both child affective distress and family routines.

Across sleep domains, internalizing symptoms emerged as the most consistent correlate of sleep symptom endorsement, whereas externalizing symptoms showed a narrower association, emerging mainly for SRMD and parasomnia symptoms. The absence of a significant interaction suggests that internalizing distress relates to sleep symptoms in a broadly similar manner across levels of behavioral dysregulation, rather than being specific to particular combinations of symptom severity. Consistent with this pattern, profiles characterized by internalizing difficulties—either alone or in combination with externalizing symptoms—tended to show greater sleep symptom endorsement across domains, whereas externalizing-only presentations were more selectively linked to SRMD and parasomnia symptoms. This is broadly consistent with Lycett et al. [17], who similarly found that mixed and internalizing-dominant presentations showed higher levels of sleep symptoms, whereas externalizing-only presentations were closer to low-symptom profiles. Overall, these findings suggest that emotional distress may play a more central role in sleep symptoms in ADHD than disruptive behavior alone [38–42].

Mediation analyses refined this pattern by indicating that internalizing symptoms statistically accounted for a substantial share of the association between externalizing symptoms and sleep symptom endorsement across all three domains. Longitudinal work supports indirect affective pathways linking behavioral dysregulation with sleep problems. Mesman et al. [43] found that early externalizing symptoms preceded later internalizing difficulties, suggesting a potential developmental pathway. Kouros and El-Sheikh [44] further highlighted that affective dysregulation may mediate the relationship between sleep problems and externalizing-internalizing behaviors, underscoring the role of internal emotional functioning in behavioral outcomes. Similarly, cross-sectional studies found that anxiety partially explains externalizing–sleep associations in clinic-referred youth including also ADHD [45]. Moreover, Mulraney et al. [46] found in a cohort of 270 children with ADHD aged 5-13 years found bidirectional links between internal emotional problems and sleep: baseline internalizing symptoms predicted sleep problems six months later, and baseline sleep problems predicted later internalizing symptoms. No bidirectional relationship was observed between sleep problems and externalizing symptoms. Overall, prior evidence converges on internal emotional distress as a key correlate of nocturnal dysfunction and is consistent with the indirect associations observed here, with domain-specific nuances across insomnia, SRMD, and parasomnia symptoms.

For insomnia symptoms, the indirect effect through internalizing symptoms was statistically reliable, whereas the average direct effect of externalizing symptoms was not significant. This pattern indicates that the externalizing–insomnia association was primarily explained by the co-occurrence of internalizing distress. This is consistent with prior work showing robust links between internalizing symptoms and insomnia severity in ADHD [42,47–49]. Complementarily, cross-sectional analyses within a longitudinal cohort reported developmental shifts: in preschool, insomnia related to hostile-aggressive and hyperactive-distractible behaviors; by age 9, persistent insomnia co-occurred with depressive symptoms alongside externalizing/ADHD symptoms; and by age 18, insomnia aligned most with anxiety and ADHD [50]. Although that study did not test mediation, the recurrent coupling of internalizing symptoms with insomnia across development is broadly consistent with the present findings.

Further, studies in youth and adult samples with insomnia symptoms [51–53] are consistent with a full-mediation account in which internalizing symptoms carry the association with insomnia. Specifically, internalizing symptoms, particularly depression, are closely related to insomnia at both phenotypic and genetic levels [54,55], with phenotypic overlap remaining stable over time [56]. Internalizing symptoms and insomnia also share overlapping neurobiological mechanisms and circuits [57,58], a convergence proposed as a transdiagnostic neural marker linking the two conditions. Finally, "restless REM sleep" together with maladaptive nocturnal rumination may index deficient overnight adaptation to daytime stress in insomnia [59]. A plausible, although speculative, hypothesis linking internalizing symptoms to insomnia involves heightened cortisol levels and atypical cortical maturation. Elevated cortisol in children and adolescents has been shown to hinder sleep onset [60,61] and may partly explain its association with internalizing problems [62–64]. Furthermore, alterations in sleep EEG microstructure—such as age-related abnormalities in slow-wave activity and cyclic alternating pattern indices, which reflect atypical cortical maturation—may impair emotion regulation, increase vulnerability to internalizing symptoms, and contribute to sleep disturbances [65]. Although speculative, this perspective may help clarify the prominent role of internalizing symptoms, together with reduced sleep duration,in insomnia. It also offers a potential explanation for the absence of moderating effects of parental emotional responsiveness or parenting style, pointing toward a predominantly biological rather than environmental pathway.

For SRMD symptoms, both indirect and direct associations were statistically reliable: internalizing symptoms accounted for part of the externalizing–SRMD association, but a residual direct association of externalizing symptoms remained. This pattern is consistent with the idea that motor hyper-arousal may be heightened when behavioral dysregulation co-occurs with emotional distress [66]. This pattern aligns with prior evidence linking ADHD to specific SRMD symptoms [67–70] and relating SRMD symptoms to both externalizing (e.g., ODD) and internalizing difficulties, although often outside ADHD-specific samples [71,72]. Consistent with our results, SRMD symptoms have been associated with elevated ADHD hyperactivity–impulsivity and externalizing symptoms (predominantly ODD) [73,74], as well as heightened anxiety and depressive symptoms and mixed internalizing-externalizing symptoms, suggesting feedback mechanisms between sleep-related motor activity and affect regulation [73,75]. A plausible shared mechanism involves brain iron deficiency [76,77] and dopaminergic dysfunction [70,78–80], whereby heightened affective arousal further perturbs dopaminergic signaling. Converging evidence indicates that the amygdala modulates stimulus–reward effects on behavior through dopamine-dependent mechanisms in the striatum [81,82], a circuit implicated in the neuro-physiopathology of SRMD symptoms. This account is compatible with the partial indirect association observed in this sample, in which internalizing distress transmits part of the externalizing–SRMD symptoms link.

Finally, co-occurring internalizing and externalizing symptoms both showed unique associations with parasomnia symptoms (i.e., "disorders of arousal" (DOAs) and nightmares), with internalizing symptoms showing the stronger association. This pattern aligns with evidence that anxiety-related hyperarousal and depressive affect are primary drivers

of parasomnias, whereas behavioral dysregulation adds a smaller, secondary load [83]. Prior studies likewise indicate that both symptom domains can trigger DOA events by lowering arousal thresholds [84–86]. Proposed processes include persistent hyperarousal and stress undermining emotion regulation, disruption of executive–limbic circuitry with incomplete neocortical–limbic dissociation, covariation between emotional/behavioral symptom severity and episode intensity, and alterations in self-referential and stress-coping processes [87–89]. Taken together with the correlations among insomnia, SRMD, and parasomnia symptoms, the present pattern supports internalizing distress as an important correlate of nocturnal dysfunction across domains, while leaving room for symptom-specific mechanisms and bidirectional influences that should be evaluated in prospective designs.

4.1. Moderator parental factors effect on SRMD and parasomnia symptoms

Across outcomes, moderation effects were selective and domain-specific. For insomnia symptoms, no parental factor moderated the associations between internalizing/externalizing symptoms and symptom endorsement, as none of the interaction terms survived FDR correction. Nevertheless, two parent-level variables showed small but reliable main associations: higher negative parental emotion and higher positive parenting were each associated with greater insomnia symptoms. Importantly, in these models internalizing symptoms remained the only consistent behavioral correlate of insomnia, whereas externalizing symptoms did not show a reliable association. This suggests that parental characteristics may relate to insomnia symptom reporting at the level of overall endorsement, but they do not appear to condition how child internalizing or externalizing difficulties translate into insomnia symptoms.

For SRMD symptoms, positive parenting attenuated the association with externalizing symptoms, whereas negative parenting strengthened the association with internalizing symptoms. This pattern suggests that parenting strategies may shape SRMD symptoms differently depending on whether sleep difficulties are more closely linked to behavioral dysregulation or to emotional distress, consistent with prior work showing that parenting quality can either exacerbate or mitigate children's internalizing/externalizing difficulties and associated sleep problems in ADHD [21,90,91]. A possible, albeit indirect, interpretive framework is that parenting may influence these processes through self-regulation and stress responsivity 92–96. At the same time, the positive direct associations of positive parental emotion and positive parenting with SRMD symptoms indicate that positively valenced parental processes are not necessarily uniformly protective. Rather than indicating maladaptive parenting, these effects may reflect greater parental involvement, monitoring, and attempts to manage more salient or persistent nocturnal difficulties.

For parasomnia symptoms, positive parental emotion attenuated the association with externalizing symptoms but strengthened the association with internalizing symptoms, suggesting that positively valenced parental affect may operate differently depending on the child's symptom profile. When parasomnia symptoms are more closely linked to behavioral dysregulation, warmth and empathic engagement may reduce bedtime conflict and behavioral arousal [21]. By contrast, when parasomnia symptoms are more closely linked to internal distress, heightened parental emotional involvement may not always be protective and may instead coincide with over-arousal, overinvolvement, or difficulty downregulating the child's distress [97]. This interpretation is broadly consistent with models of emotion contagion and dyadic coregulation [98–100], and with accounts linking unmodulated parental involvement to children's heightened arousal and sleep–wake instability [101–106]. A complementary possibility is that highly engaged "positive" involvement may, in some cases, shift toward overprotection or attempts to rapidly resolve the child's distress, thereby limiting opportunities for autonomous emotion regulation [101,107–109].

Positive parenting strategies showed a similarly differentiated pattern. They attenuated the association between externalizing symptoms and parasomnia symptoms, suggesting that supportive behavioral strategies may reduce bedtime conflict and the behavioral arousal component feeding into nocturnal episodes. At the same time, positive parenting also showed a positive direct association with parasomnia symptoms. These two effects are not necessarily contradictory. Rather than indicating that positive parenting is maladaptive, this pattern may suggest that higher levels of supportive parenting characterize families already coping with greater nocturnal difficulties, reflecting increased parental involvement and supportive management in response to the child's sleep problems. Overall, these findings are consistent with prior work showing that apparently positive parental processes may not always operate in a uniformly protective way across symptom profiles and sleep domains [90,110].

4.2. Clinical implications

The findings suggest that treating sleep symptoms (mainly insomnia and parasomnia) in children with ADHD should prioritize reducing internalizing distress through cognitive-behavioral, mindfulness, or pharmacological approaches, while also addressing externalizing symptoms. Additionally, parent training should emphasize not merely "positive emotion" but regulated warmth: empathy paired with parents' own emotion regulation. Coaching caregivers to manage their own arousal and validate the child's emotions can enhance support effectiveness. Finally, tailoring interventions according to whether sleep problems are chiefly internalizing-versus externalizing-driven may optimize outcomes for both daytime functioning and nocturnal symptoms.

4.3. Limitations

Some constraints merit consideration. Although our mediation model was informed by longitudinal evidence and theory, the cross-sectional design prevents inferences about temporal ordering or causality. Mediation can still be used to explore plausible pathways, as in recent studies [111–114]. Prospective, multi-wave designs (ideally with repeated measures of symptoms and sleep) are needed to test directionality and adjudicate causal pathways.

A second limitation concerns measurement. Because all data were based on parent report, the findings may be affected by shared method variance and by the absence of complementary perspectives on child functioning and sleep. Future research should include multi-informant approaches (e.g., child- and parent-reports) and objective sleep measures (e.g., actigraphy, polysomnography) to validate subjective reports. Ecological momentary assessment could further capture real-time variations in parenting behaviors and child emotional states.

Additional caution is warranted with respect to sample heterogeneity. Pharmacological treatment was not included as a covariate. Although a proportion of participants were receiving medication, treatment status reflected heterogeneous clinical conditions, including different medications and polypharmacy. Because this variability could not be adequately represented by a simplified indicator, medication was not included in the present models. Likewise, psychiatric comorbidity was not entered as an additional covariate. The cohort was clinically heterogeneous and many participants showed at least one comorbidity. Because the main predictors were dimensional, adding multiple categorical comorbidity indicators would have reduced model interpretability and increased overlap with the constructs of primary interest. Finally, although age was included as a covariate, the wide developmental range of the sample (5–18 years) may have obscured age-specific patterns. Because sleep phenotypes and parasomnia symptoms change across development, future studies should examine whether these associations differ between childhood and adolescence.

Despite the relatively large sample size within the Italian context, the

possibility of selection bias cannot be excluded. Recruitment took place exclusively through the official website of the National ADHD Family Association and may therefore have preferentially reached families who were more informed, engaged, or motivated to participate. Consequently, the sample may not fully represent families without internet access, those unaware of the association, or those less involved in its activities.

Finally, although the present analyses were based on ratings referring to the pre-COVID period, these reports were collected during the COVID-19 period and therefore some degree of retrospective recall bias cannot be excluded. At the same time, the use of separate ratings for the periods before and during the pandemic allowed us to approximate pre-pandemic functioning more closely than would have been possible with a single undifferentiated retrospective report.

5. Conclusion

The present findings suggest that associations between ADHD and sleep symptoms are multifaceted, varying according to co-occurring internalizing and externalizing symptoms, the specific sleep domain considered, and selected parent-level factors. To our knowledge, this is the first study to model these interactions concurrently across insomnia, SRMD, and parasomnia symptoms while testing the moderating role of parental emotions and parenting practices. Collectively, the results support the value for integrative, transdiagnostic frameworks that jointly consider daytime psychopathology and nocturnal dysfunction in ADHD. Such frameworks may help to clarify shared versus domain-specific patterns linking internalizing symptoms, externalizing symptoms, and sleep problems, and may inform future longitudinal and intervention research.

CRediT authorship contribution statement

Maria Grazia Melegari: Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Michele Scandola:** Writing – review & editing, Validation. **Martina Giallonardo:** Writing – review & editing, Methodology, Data curation. **Michela Vezzoli:** Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Investigation, Formal analysis.

Declaration of conflict interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this article.

Declaration of generative AI

During the preparation of this work, the authors used ChatGPT as a language-support tool to perform linguistic checks and improve clarity and readability of the text. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2026.108947.

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