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The impact of sleep duration and quality on the health of the pediatric population: consequences for mental health, eating disorders, and other diseases

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Abstract

Background

Sleep is a fundamental biological process, the duration and quality of which are of key importance for the proper development and health of the pediatric population. Understanding the complex relationship between sleep and the pathogenesis of diseases during childhood is essential for developing effective preventive and interventional strategies aimed at supporting the holistic health of the youngest patients.

Aim

This review aims to provide an analysis of available scientific evidence regarding the impact of insufficient sleep duration and poor sleep quality on a wide range of health indicators in children and adolescents. Special emphasis is placed on the consequences for mental health and eating disorders.

Materials and methods

This review synthesizes current evidence by examining literature available on PubMed, Google Scholar, and academic sources using keywords related to the sleep duration, sleep quality, pediatric population, mental health and eating disorders. Experimental, cohort, systematic reviews and clinical studies were evaluated.

Results

We highlight sex- and age-dependent vulnerabilities and discuss the multifactorial interplay between sleep behavior, lifestyle factors, and disease risk. Specifically, interventions aimed at improving sleep hygiene, reducing screen exposure before bedtime, and addressing sleep disorders such as obstructive sleep apnea show promise in mitigating risks associated with obesity, hypertension, emotional disorders, and chronic inflammation.

Conclusion

Sleep duration and sleep quality contributes to pediatric obesity, insulin resistance, cardiometabolic risk, mental health, inflammatory processes and headache disorders. Limitations in current research, including methodological heterogeneity and reliance on subjective sleep measures, underscore the need for prospective studies. We recommend future research focus on mechanistic pathways linking sleep disturbances to specific health outcomes.

Keywords: sleep duration, sleep quality, pediatric population, mental health, eating disorders, obesity, insulin resistance, cardiometabolic risk

1. Introduction

Sleep is crucial for children's physical, cognitive, and emotional development. Both the duration and quality of sleep affect health, cognitive functions, behavior, and the risk of somatic problems, such as obesity or metabolic disorders. Moreover, insufficient sleep frequently coincides with longer screen exposure and sedentary behavior. Silistre et al. found that screen

time was significantly higher in children with inadequate sleep, supporting the idea that sleep curtailment is part of an obesogenic lifestyle pattern [1].

Research indicates that sleep duration in children shows high interpersonal variability, with relative individual stability over time. Jenni et al. showed that while sleep time differs among children, each individual has a relatively constant need for sleep, and short-term fluctuations are natural [2].

Sleep quality depends on many factors: genetics, lifestyle, environmental conditions, family habits, and co-existing diseases. A key risk factor for sleep disorders is the use of electronic devices before bedtime, which correlates with a reduction in sleep time [3]. Studies show that insufficient sleep quality leads to daytime sleepiness, cognitive problems, and emotional and behavioral disorders. It is increasingly emphasized that both sleep duration and quality should be considered together, as the quantity of sleep alone does not guarantee its effectiveness or positive impact on health [4,5].

Knowledge of these dependencies has practical significance: it allows for adjusting recommendations to the child's individual needs, monitoring sleep disorders, and implementing appropriate preventive interventions.

Aim

The aim of this study is to synthesize scientific evidence on how insufficient sleep duration and poor sleep quality affect various health outcomes in children. It particularly emphasizes consequences for mental health (such as anxiety and mood disorders), eating disorders, and other conditions like cardiometabolic, endocrine, and immune disorders. This review explores mechanistic links, including neuroendocrine dysregulation and immune activation, while highlighting vulnerabilities by sex and age, and advocating for preventive strategies like improved sleep hygiene. The goal supports holistic pediatric health by integrating sleep assessment into disease prevention. The gathered data were meticulously evaluated to determine recurring patterns, methodological constraints, and new directions in research.

2. Research materials and methods

2.1. Data collection and analysis

A comprehensive literature review was conducted using the PubMed and Google Scholar databases. The search strategy included terms such as sleep duration, sleep quality, pediatric population, mental health and eating disorders. The analysis concentrated on studies examining the impact of sleep quality and duration on various aspects of health among children.

3. Research results

3.1. Obesity

Obesity in children and adolescents is a rapidly growing public health concern globally, with prevalence rates escalating significantly across various regions. Emerging evidence underscores the pivotal role of sleep quality and duration as important factors associated with

the risk of overweight and obesity in these populations. The study by Pileggi et al. demonstrated a significant association between chronic short sleep duration and higher body mass index (BMI) in children around 10 years old, independent of other factors such as diet, physical activity, and parental obesity [6].

The study by Elizabeth et al. assessed the relationship between sleep quality and obesity prevalence among school-age children in Indonesia, revealing that poor sleep quality substantially increases the likelihood of obesity.

The authors reported that children with poor sleep quality were 8.6 times more likely to be obese compared to those with good sleep quality, with habitual sleep efficiency identified as the most dominant component related to obesity risk (Odds Ratio (OR) = 12.36). Mechanistically, poor sleep quality disrupts hormonal regulation, leading to increased appetite and energy intake, thereby contributing to the development of obesity. Furthermore, this study highlighted interrelations between dietary intake, physical activity, sleep efficiency, and obesity risk, emphasizing the multifactorial nature of obesity etiology in children [7].

Supporting these findings, the systematic review and meta-analysis by Han et al. focused on short sleep duration as a risk factor for overweight and obesity in children and adolescents across diverse populations. Their analysis revealed that short sleep duration was associated with a significant increase in the odds of being overweight or obese (OR = 1.17), particularly pronounced in the age groups 6 years and 6 to 10 years, and notably among boys. The authors noted geographical and sex-related differences, with stronger associations observed in Asian populations and male children. These findings implicate insufficient sleep duration as an independent risk factor for elevated body mass index (BMI) in pediatric populations. The observed associations may be driven by neuroendocrinological disruptions that affect appetite regulation and energy balance. Han et al. also emphasized the complexity of sleep's impact on obesity, calling for more objective measures of sleep parameters and consideration of confounding lifestyle factors in future research [8].

Epidemiological investigations indicate that short sleep duration is associated with increased risk of obesity, as reflected by elevated body mass index (BMI) and waist circumference, the latter being a more precise marker of abdominal adiposity and visceral fat accumulation. Mechanistically, sleep deprivation disrupts endocrine regulation involving appetite hormones such as leptin and ghrelin, leading to increased energy intake and weight gain. Additionally, behavioral factors contribute, where extended wakefulness provides more opportunities to consume calories, while increased fatigue reduces physical activity levels [9].

The literature review by Munyati and Wahyudi synthesizes findings from over 100 studies and concludes that shorter sleep duration is significantly associated with higher body mass index (BMI), increased prevalence of overweight and obesity, and greater central adiposity in school-aged children [10]. Similarly, Silistre et al. conducted a cross-sectional analysis of 1002 primary school children aged 6–12 years in Istanbul and found that obese children had significantly shorter mean sleep duration than their normal-weight peers [1].

3.2. Insulin resistance

A substantial body of evidence demonstrates a close correlation between sleep duration and insulin resistance, particularly in populations at risk for metabolic syndrome. Short sleep duration disrupts normal hormonal homeostasis, impacting key regulators such as leptin, ghrelin, insulin, cortisol, and growth hormone, while also increasing sympathetic activity and catecholamine levels. These endocrine and autonomic changes collectively foster energy imbalance and promote both obesity and insulin resistance, with robust metabolic consequences observable even in adolescents and young adults [11].

Mechanistically, sleep deprivation interferes with insulin signaling pathways through both central and peripheral effects. Sleep loss impairs hypothalamic glucose sensing, increases circulating anti-insulin hormones (e.g., cortisol), and promotes systemic inflammation by raising cytokine levels such as TNF-α and IL-6. Additionally, disrupted sleep alters other metabolic hormones and neurohormones, modulating appetite and energy storage while impairing glucose uptake and utilization in key tissues. Chronic sleep deprivation is associated with increased adiposity, impaired glucose metabolism, elevated inflammatory milieu, and ultimately, a heightened risk for type 2 diabetes mellitus and other metabolic disorders [12].

Recent cross-sectional research in obese adolescents with metabolic syndrome reveals a significant negative correlation between sleep duration and insulin resistance, as measured by the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). Specifically, individuals obtaining fewer than eight hours of sleep nightly exhibited higher fasting insulin levels and elevated HOMA-IR values compared to their peers with longer sleep durations. Statistical analysis confirmed this inverse relationship ($rs = -0.581$, $p < 0.001$), emphasizing that shorter sleep duration predicts greater insulin resistance within this cohort. These findings align with previous investigations noting increased insulin levels, reduced insulin sensitivity, and elevated diabetes risk in individuals with chronic sleep restriction [11].

In pediatric populations, longitudinal and cross-sectional studies reveal that shorter nocturnal sleep duration correlates with greater abdominal obesity and insulin resistance. Path analyses have elucidated that the relationship between sleep duration and insulin resistance is predominantly mediated by abdominal obesity, rather than being direct. That is, longer sleep duration contributes to reduced waist circumference, which in turn lessens insulin resistance markers, such as HOMA-IR. These effects, while notable, exhibit modest magnitude in large samples, emphasizing that sleep is a contributory rather than exclusive factor in obesity development [13].

3.3. Cardiology

An increasing body of evidence indicates that both sleep duration and sleep quality during childhood and adolescence significantly influence blood pressure and overall cardiovascular risk. A retrospective clinical study involving 539 children (mean age 14.6 years) who underwent ambulatory blood pressure monitoring (ABPM) demonstrated that each

additional hour of sleep (mean 9.1 h/night) was associated with a significantly lower risk of daytime hypertension (OR = 0.88; 95% CI, 0.79-0.99), while later bedtime correlated with higher systolic blood pressure (SBP; β = 0.07; 95% CI, 0.02-0.13) [14].

A population-based study in China (N = 1,085 children) revealed a nonlinear relationship between sleep duration and blood pressure: the optimal sleep range was 8-10 hours per night, while both shorter (< 8 h) and longer sleep (> 10 h) were associated with elevated systolic blood pressure (SBP-Z; β = -0.10, 95% CI: -0.17 to -0.03 and β = 0.34, 95% CI: 0.04–0.64, respectively) [15].

In the large Brazilian ERICA cohort (N = 65,643 adolescents aged 12-17 years), each additional hour of sleep was significantly associated with lower SBP in boys (p = 0.004), whereas a slight but significant increase in SBP was observed in girls (p = 0.009) after adjusting for confounding factors, suggesting potential sex differences in the sleep-blood pressure relationship [16]. Longitudinal studies among children aged 8-11 years (N = 723) indicated that shorter actigraphy-measured sleep was associated with a worse cardiometabolic profile (metabolic syndrome score), independent of physical activity and body mass index [17].

Mechanistically, short and disturbed sleep affects the autonomic nervous system, leading to increased sympathetic activity, circadian rhythm disruption, and reduced nocturnal blood pressure dipping, along with hormonal alterations, including elevated cortisol levels and impaired glucose regulation. Prolonged effects may include early structural vascular changes, increased arterial stiffness, and a higher risk of hypertension and cardiovascular disease in adulthood. A meta-analysis encompassing over one million participants found that short sleep (< 7 h) significantly increased the risk of developing hypertension (HR = 1.07; 95% CI, 1.06-1.09), with extremely short sleep (< 5 h) associated with even higher risk (HR = 1.11; 95% CI, 1.08-1.14), whereas long sleep (> 8 h) did not significantly increase risk (HR = 1.01; 95% CI, 1.00-1.03).

Collectively, the literature indicates that maintaining optimal sleep duration (8-10 h) and high sleep quality represents a critical component of early prevention strategies for hypertension and cardiovascular disease in children and adolescents. Interventions such as establishing regular bedtimes, limiting screen exposure before sleep, and implementing strategies to improve sleep quality may serve as effective non-pharmacological measures to reduce the risk of early cardiometabolic dysfunction and long-term hypertension.

3.4. Anxiety disorders

An expanding body of empirical research demonstrates that both sleep duration and the structural aspects of sleep-particularly bedtime and sleep onset latency (SOL)-play a crucial role in the development and severity of anxiety symptoms in children and adolescents. In a very large sample of preschool-aged children (N ≈ 69,138), Qiu et al. reported statistically significant

associations between later bedtimes and an increased likelihood of anxiety symptoms: children who fell asleep between 21:01 and 22:00 had a 48% higher probability of anxiety compared with those who went to bed before 21:00 (AOR = 1.48; 95% CI: 1.26-1.76), whereas children who fell asleep after 23:00 exhibited nearly a threefold increase in anxiety risk (AOR = 2.86; 95% CI: 2.21-3.69, $p < 0.001$). Moreover, short sleep duration (< 9 hours) was associated with a 36% increase in anxiety symptoms relative to children who slept at least 10 hours (AOR = 1.36; 95% CI: 1.23-1.51). These findings suggest that both insufficient sleep and delayed sleep timing constitute significant risk factors for anxiety already in early childhood.

Complementary evidence comes from an investigation by Haugland et al., involving 313 adolescents (12-16 years, mean age 14.0; 84% female) seeking treatment for anxiety within primary care. The study found that sleep disturbances were highly prevalent in this population: 38.1% met criteria for insomnia, 34.8% reported short sleep duration (< 7 h), and 83.1% experienced a sleep onset latency of at least 30 minutes. Linear regression analyses revealed that overall anxiety severity was strongly associated with prolonged SOL ($\beta = 0.25$, $p < 0.001$), and this relationship remained significant after adjusting for age and gender ($\beta = 0.23$, $p < 0.001$). Furthermore, anxiety symptoms were inversely correlated with total sleep duration ($\beta = -0.18$, $p < 0.01$), although this association was attenuated when depressive symptoms were included in the model ($\beta = -0.15$, $p < 0.01$), indicating that comorbid depression may mediate part of the relationship between sleep and anxiety [18].

Mechanistically, delayed sleep timing and reduced sleep duration may disrupt circadian rhythms and neuroendocrine regulation-such as stress hormone secretion-leading to heightened limbic activation (particularly in the amygdala) and diminished emotional regulation capacity, thereby exacerbating anxiety symptoms. Qiu et al. additionally highlight that late bedtimes may directly dysregulate circadian-controlled hormone release, contributing to increased vulnerability to anxiety in children [19].

Taken together, these findings underscore an important clinical implication: preventive and therapeutic strategies should address not only the alleviation of anxiety symptoms but also the optimization of sleep hygiene-most notably promoting earlier bedtimes and ensuring adequate sleep duration. This approach may be especially beneficial for children and adolescents in whom anxiety co-occurs with chronic sleep disturbances, as both studies indicate that sleep problems and anxiety often co-exist independently of depressive symptomatology.

3.5. Mental health

Empirical research indicates that both sleep duration and sleep quality in early childhood exert a significant influence on the development of emotional, behavioral, and cognitive

functions later in life. A prospective cohort study including 799 children, which employed actigraphy for objective sleep assessment, demonstrated that shorter sleep duration at ages 6 and 8 was associated with a significant increase in emotional disorder symptoms at ages 8 and 10 ($\beta = -0.44$; 95% CI [−0.80, −0.08], $p = 0.02$ and $\beta = -0.47$; 95% CI [−0.83, −0.11], $p = 0.01$), and in boys, also with heightened behavioral problems ($\beta = -0.49$; 95% CI [−0.90, −0.07], $p = 0.02$) [20]. These findings highlight the strong predictive value of sleep duration for the emergence of psychopathological symptoms, with effects particularly pronounced in boys, suggesting potential sex differences in vulnerability to the consequences of shortened sleep.

Systematic reviews of longitudinal studies, encompassing seven cohort analyses, have shown that early childhood sleep disturbances-including behavioral insomnia, parasomnias, obstructive sleep apnea, and nighttime awakenings-significantly predicted the occurrence of anxiety disorders, depression, and ADHD during adolescence [21]. These studies further indicated that both sleep durations below recommended norms (i.e., <9 hours per night for children aged 6–12 years) and frequent sleep disruptions increased the risk of emotion dysregulation, impulsivity, and social difficulties.

Preliminary findings from cohort studies published as preprints suggest that sleep restriction elevates the risk of depressive symptoms and worsened daily mental well-being: the Average Treatment Effect (ATE) for depressive symptoms was +5.6% ($p < 0.001$), and for the number of days with poor mental health approximately +2.24 days per month ($p < 0.001$) [22]. These results indicate that even modest reductions in sleep can have cumulative effects on children's mental health, particularly if they persist chronically.

Multimethod studies have also shown that subjective assessments of sleep quality, reported by both parents and children, correlate with greater severity of psychopathological symptoms, whereas objective sleep measures such as total sleep time or the number of awakenings recorded by actigraphy do not always predict all dimensions of mental health [23]. Such discrepancies highlight the complexity of the relationship between subjective sleep quality and psychological functioning, suggesting that perceived sleep may play a mediating role in the development of emotional and behavioral symptoms.

From a neurobiological perspective, shortened and disrupted sleep affects the hypothalamic-pituitary-adrenal (HPA) axis, increasing cortisol levels and stress sensitivity, and impacts the limbic system, including the hippocampus and amygdala, leading to impairments in memory consolidation and emotion regulation. Additionally, alterations in dopaminergic and serotonergic neurotransmission may promote impulsivity, hyperactivity, and aggressive tendencies.

Collectively, the evidence indicates that maintaining optimal sleep duration and quality during childhood may serve as a protective factor against the development of emotional and behavioral disorders. Interventions aimed at improving sleep hygiene-including consistent bedtimes, limiting screen exposure before sleep, parental education, and behavioral strategies for sleep-onset difficulties-may hold significant preventive potential, reducing the risk of depression, anxiety, ADHD, and other disorders throughout childhood and adolescence.

3.6. Inflammation

A growing body of evidence demonstrates that both sleep duration and sleep quality are major determinants of immune activation in children and adolescents, and that sleep disturbances-whether quantitative or related to disruptions in sleep architecture-are strongly associated with elevated inflammatory markers, which play a central role in the pathogenesis of numerous chronic diseases. In a large analysis by Alqaderi et al., involving 352 adolescents aged 16-19 years, later sleep onset time was significantly correlated with increased interleukin-6 (IL-6) levels, with each 1-hour delay in bedtime associated with an approximate 0.05 pg/mL increase in IL-6 ($p = 0.01$). Additionally, adolescents with substantial sleep debt (≥ 2 h) showed higher concentrations of C-reactive protein (CRP; by approximately 0.61 µg/mL, $p = 0.02$) and salivary IL-6 (by 0.38 pg/mL, $p = 0.01$), indicating that both chronic sleep restriction and circadian misalignment have a measurable impact on inflammatory activation. Notably, the authors demonstrated full mediation by BMI in the associations between delayed sleep and levels of CRP and IL-6, suggesting that part of the inflammatory burden produced by sleep disruption may be conveyed through metabolic impairment, including adipokine dysregulation and oxidative stress typical of excess body mass [24].

Comparable findings were reported by Strumberger et al., who, in a pediatric sample presenting with clinically relevant sleep disturbances, observed a positive association between the severity of sleep problems and plasma CRP concentrations. This relationship remained significant even after adjusting for BMI, psychosocial stress, and depressive symptoms, indicating that sleep disruption in children may serve as an independent driver of systemic inflammation. Particularly robust evidence stems from research on children with obstructive sleep apnea syndrome (OSAS), where marked elevations in both pro- and anti-inflammatory cytokines have been documented, reflecting chronic immune activation triggered by sleep fragmentation and intermittent hypoxia. In one such study, children with OSAS displayed significantly elevated levels of YKL-40 (29.28 vs. 25.13 ng/mL, $p = 0.027$), IL-6 (1.91 vs. 0 pg/mL, $p = 0.002$), IL-8 (66.65 vs. 28.29 pg/mL, $p < 0.001$), and IL-10 (7.08 vs. 4.86 pg/mL, $p = 0.008$) compared with controls, underscoring the proinflammatory impact of disturbed nocturnal respiratory patterns [25].

The mechanisms underlying these associations include circadian desynchronization, heightened activation of the hypothalamic-pituitary-adrenal (HPA) axis, altered cortisol secretion profiles, increased oxidative stress, and upregulation of the NF-κB signaling pathway-all of which contribute to a chronic low-grade inflammatory state. Long-term exposure to this inflammatory burden during childhood may significantly elevate the risk of developing cardiovascular disease, hypertension, insulin resistance, and obesity in adulthood, highlighting substantial public health implications. Taken together, these findings reinforce the importance of prioritizing sleep hygiene, stabilizing circadian rhythms, and ensuring early detection and treatment of sleep disorders-including OSAS-as essential components of preventive strategies aimed at mitigating inflammatory and metabolic risk in pediatric populations. This perspective

is further supported by the landmark study by Larkin et al., which found that adolescents with more severe sleep-disordered breathing (SDB) had significantly higher levels of high-sensitivity C-reactive protein (CRP). In their polysomnography-based cohort (n = 143, ages 13-18), mean CRP values (adjusted for BMI, age, sex, and race) rose in a dose-response manner across SDB severity groups: 0.50 mg/L for AHI < 1, 0.43 mg/L for AHI 1–4.9, 0.97 mg/L for AHI 5–14.9, and 1.66 mg/L for AHI ≥ 15 ($p = 0.0049$). They concluded that even in adolescents without known cardiovascular disease, moderate to severe SDB may confer systemic inflammatory burden, representing an early cardiovascular risk factor beyond the influence of obesity [26].

3.7. Headaches

Multiple studies have evidenced a strong association between sleep disturbances and headache disorders, particularly migraine, in pediatric populations. Sleep-related issues are common complaints among children with migraines, yet the complex mechanisms linking these conditions remain incompletely understood. Both insufficient sleep duration and poor sleep quality have been identified as frequent triggers for headache attacks across age groups. Childhood migraineurs often exhibit disturbed sleep patterns, including delayed sleep onset, irregular sleep schedules, frequent nocturnal awakenings, nightmares, hypnic jerks, and restless sleep, which correlate with increased headache frequency and duration [27, 28].

Sleep hygiene-the set of behavioral and environmental practices conducive to sustained, restorative sleep-has been investigated as a modifiable factor in managing pediatric migraine. A randomized controlled study demonstrated that improving sleep hygiene significantly reduced the mean duration and frequency of migraine attacks in affected children, without pharmacological intervention, whereas severity of attacks appeared less influenced by sleep behavior and more associated with underlying sleep disorders. This suggests that circadian rhythm disruption may exacerbate migraine frequency and duration, whereas sleep structure abnormalities contribute to attack severity [27].

Comorbid sleep disorders prevalent among children with headaches include insomnia, sleep apnea, sleep bruxism, restless legs syndrome, and parasomnias such as sleep terrors and somnambulism. These conditions share neurochemical and anatomical pathways with migraine pathophysiology, particularly involving serotonergic and dopaminergic systems. For instance, sleep apnea has been linked to increased morning headache prevalence, while restless legs syndrome shows higher occurrence among migraineurs compared to controls, potentially worsening headache chronicity and disability [28].

The bidirectional relationship between sleep and headache is underscored by findings that pain disorders disrupt sleep continuity and sleep deprivation heightens pain sensitivity, establishing a vicious cycle that may promote migraine chronification. Education on sleep hygiene, behavioral interventions, and targeted treatment of sleep disorders have been recommended as integral components of managing pediatric migraines to enhance therapeutic outcomes and quality of life [27, 28].

3.8. Atopic dermatitis

Atopic dermatitis (AD) is a prevalent chronic inflammatory skin disorder that significantly impacts sleep quality in affected individuals across all ages. Sleep disturbances are reported in approximately 47–80% of children and 33–90% of adults with AD, markedly higher than in the general population. Despite the chronic relapsing nature of AD, sleep duration tends to be comparable between individuals with and without AD; however, sleep quality is distinctly impaired in those with AD [29].

Children with active AD more frequently experience multiple sleep-quality disturbances, including difficulty falling asleep, frequent nocturnal awakenings, early morning awakenings, and nightmares. These disturbances lead to sleep fragmentation, reduced sleep efficiency, and increased wake time after sleep onset, which have been objectively confirmed by polysomnography and actigraphy studies. Notably, even children in remission or with inactive disease continue to exhibit poorer sleep quality, suggesting persistent alterations in sleep regulation beyond acute symptomatology [29, 30].

The underlying mechanisms for sleep disturbance in AD include nocturnal pruritus and scratching behaviors, which disrupt sleep continuity. However, scratching alone accounts for a minor proportion of awakenings, indicating additional factors at play. Neuroimmune dysregulation involving sensory hypersensitivity, inflammatory cytokines (e.g., IL-31, IL-4, IL-13), circadian variations in inflammatory mediators, and melatonin secretion disruptions have been proposed as contributory pathways affecting sleep architecture in AD patients. The complex bidirectional relationship between sleep disturbance and AD severity underscores the need for comprehensive management strategies that address both dermatologic and sleep-related symptoms [29].

In clinical practice, impaired sleep quality constitutes a notable comorbidity in patients with AD and warrants routine assessment. Current challenges include limited objective sleep measurement in this population and the need for validated sleep outcome measures tailored for AD. Improved interventions targeting both AD inflammation and sleep disturbance, alongside behavioral therapies for insomnia, are essential to mitigate the adverse effects of sleep disruption on quality of life, cognitive function, and overall health in AD patients [29, 30].

3.9. Memory

Sleep exerts a profound influence on memory consolidation processes in children, particularly facilitating the stabilization and enhancement of word learning from infancy

through early childhood, as demonstrated across experimental paradigms reviewed in the provided literature. In a systematic analysis of 16 studies encompassing participants aged birth to 3 years, post-learning naps consistently supported veridical retention of novel word-form-meaning associations, with behavioral measures such as pointing tasks and eye-tracking revealing superior recall in nappers compared to wake groups; moreover, electrophysiological evidence, including event-related potentials like the N400 mismatch effect, underscored sleep's role in category generalization to novel exemplars, evident in infants as young as 9 months. Longitudinal investigations further illuminated how sleep consolidation-marked by a decreasing daytime-to-nighttime sleep ratio-predicts accelerated vocabulary growth, while parental reports and actigraphy confirmed that habitual nappers exhibit enhanced lexical stabilization over delays [31]. Complementing these infancy-focused findings, research on school-aged children highlights the involvement of sleep spindles during stage 2 non-rapid eye movement sleep, where fast spindles (≥13 Hz) negatively correlated with sensorimotor performance and positively with narrative memory, and overall spindle central frequency inversely related to working memory and planning abilities, potentially reflecting neural pruning for synaptic efficiency in the developing brain [32]. Collectively, these observations affirm sleep's domain-general contributions to lexical development, though persistent research gaps, such as the interplay between naps and nocturnal sleep or the modulation by preexisting phonological knowledge, warrant targeted future inquiry to fully delineate underlying neurophysiological mechanisms [31,32].

4. Discussion

From a mechanistic perspective, the findings underscore the central role of neuroendocrine and immune dysregulation as plausible etiological links between sleep disturbance and long-term health outcomes. Disruptions in leptin–ghrelin balance, activation of the hypothalamic-pituitary-adrenal axis, alterations in cortisol secretion, and upregulation of proinflammatory cytokines (e.g., IL-6, IL-8, CRP) form a convergent pathophysiological framework that may simultaneously promote obesity, insulin resistance, endothelial dysfunction, and increased pain sensitivity. Parallel alterations within limbic and prefrontal networks, impacting emotion regulation, reward processing, and cognitive control, offer a coherent explanatory model for the observed links between short or fragmented sleep and internalizing as well as externalizing psychopathology. Importantly, several studies emphasize sex- and age-dependent effects, suggesting developmental windows of particular vulnerability and the need for more granular, longitudinal approaches.

Clinically and from a public health perspective, the reviewed data argue strongly for incorporating systematic assessment and promotion of healthy sleep into pediatric prevention and intervention strategies. Interventions targeting regular bedtimes, reduction of evening

screen exposure, and structured sleep hygiene have demonstrated clinically meaningful improvements, for example in migraine frequency and cardiometabolic risk indices, even in the absence of pharmacological treatment. At the same time, the literature reveals important methodological limitations, including predominant reliance on cross-sectional designs, heterogeneity in sleep and outcome measures, and frequent use of subjective reports without parallel objective recordings. Future research should prioritize prospective, multi-method designs (combining actigraphy, polysomnography, and validated questionnaires), refine causal modelling of mediators such as adiposity and inflammation, and test integrated behavioral and medical interventions in high-risk pediatric groups (e.g., children with obesity, atopic dermatitis, or chronic headaches). Collectively, current evidence supports the conceptualization of adequate sleep in childhood as a core health behavior, comparable in importance to nutrition and physical activity, with substantial implications for lifelong cardiometabolic, immune, neurological, and psychological trajectories.

5. Conclusions

In conclusion, this comprehensive review elucidates the profound implications of suboptimal sleep duration and quality on the multifaceted health trajectories of children and adolescents, establishing these factors as critical, modifiable determinants across somatic, metabolic, neuropsychiatric, and immunological domains. Synthesizing epidemiological, clinical, and mechanistic evidence, the analysis reveals consistent associations between insufficient sleep and heightened risks of pediatric obesity, insulin resistance, cardiometabolic perturbations such as hypertension, chronic low-grade inflammation, psychopathological conditions including anxiety and mood disorders, as well as somatic afflictions like migraines and atopic dermatitis - often mediated by neuroendocrine dysregulation (e.g., leptin-ghrelin imbalance, hypothalamic-pituitary-adrenal axis hyperactivity) and proinflammatory cytokine upregulation, with notable sex- and age-dependent vulnerabilities.

These bidirectional relationships persist independently of traditional confounders like diet and physical inactivity, underscoring sleep's foundational role akin to nutrition and exercise in holistic pediatric development and lifelong disease prevention.

Notwithstanding the robustness of these findings, inherent methodological limitations - such as predominant cross-sectional designs, heterogeneous sleep assessment modalities (subjective vs. objective measures like actigraphy or polysomnography), and incomplete mediation analyses - necessitate cautious interpretation and highlight the imperative for prospective, multimethod longitudinal studies to delineate causal pathways and refine risk stratification. Clinically, the evidence advocates for the systematic integration of sleep hygiene protocols into pediatric care, encompassing regular bedtimes, pre-bedtime screen curtailment, parental education, and targeted interventions for disorders like obstructive sleep apnea, which have demonstrated efficacy in attenuating obesity, emotional dysregulation, and inflammatory burdens without pharmacological reliance.

Ultimately, prioritizing optimal sleep as a public health cornerstone promises substantial dividends in mitigating intergenerational cardiometabolic, neurological, and psychological

morbidity, while future research should prioritize tailored, evidence-based strategies for high-risk cohorts to translate these insights into actionable paradigms for enhanced child well-being.

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Author contributions

Conceptualization, J.B. and J. B.; Methodology, M.M.; Software, H.D.; Validation, J.B., J.Ż., P.B., J.B., H.D, A.O., M.P., E.K., M.M. ; Formal Analysis, J.B., J.Ż., P.B., J.B., H.D, A.O., M.P., E.K., M.M.; Investigation, J.B. and J.B.; Resources, J.B., J.Ż., P.B., J.B., H.D, A.O., M.P., E.K., M.M.; Data Curation, J.B., J.Ż., P.B., J.B., H.D, A.O., M.P., E.K., M.M.; Writing - Original Draft Preparation, J.B., J.Ż., P.B., J.B., H.D, A.O., M.P., E.K., M.M.; Writing - Review & Editing, J.B. and J.B.; Visualization, P.B.; Supervision, J.Ż.; Project Administration, M.P, E.K.

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Conflict of interest

The authors declare there are no conflicts of interest.

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