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EFFECTS OF BLUE LIGHT EXPOSURE ON RAPID EYE MOVEMENT SLEEP DURATION AND MELATONIN LEVELS IN CHILDREN: A COMPREHENSIVE LITERATURE REVIEW

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ABSTRACT

Blue light exposure from electronic devices has emerged as a significant environmental factor affecting children's sleep architecture and circadian physiology. This comprehensive literature review synthesizes empirical evidence from 2020-2025 examining the effects of blue light exposure on rapid eye movement (REM) sleep duration and melatonin levels in pediatric populations. A systematic search of PubMed, Web of Science, and related databases identified studies employing objective measurement methods, including polysomnography (PSG) and actigraphy validated against PSG, with melatonin measurement via salivary DLMO. The current evidence demonstrates that blue light exposure, particularly from electronic devices in the evening hours, significantly suppresses melatonin production in children even at very low illuminance levels (5-40 lux), producing melatonin suppression of 70-99% depending on light intensity and spectral composition. Blue light predominantly affects melatonin suppression and REM sleep through intrinsically photosensitive retinal ganglion cells (ipRGCs) expressing melanopsin with peak sensitivity at approximately 480 nm. Studies indicate that evening blue light exposure reduces REM sleep duration, increases REM fragmentation with elevated microarousals, increases sleep onset latency, and impairs sleep efficiency in children. The magnitude of effects on REM sleep and melatonin suppression demonstrates circadian timing dependency, with exposure during 21:00-22:30 hours producing maximum sleep disruption. Evening screen use, particularly interactive forms, is associated with delayed sleep onset and reduced total sleep duration in children measured via objective actigraphy. Interventions restricting blue light exposure before bedtime through screen abstinence and blue light-blocking glasses show slight improvements in sleep timing and circadian phase advancement. This review concludes that evidence-based guidelines recommending screen restriction in the 1-2 hours before bedtime are justified based on documented physiological mechanisms and objective sleep disruption in children.

KEYWORDS

Blue Light Exposure, REM Sleep, Melatonin Suppression, Children, Circadian Rhythm, Electronic Devices, Polysomnography, Actigraphy

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1. Introduction

Recent years have witnessed a dramatic increase in children's evening exposure to light-emitting electronic devices, fundamentally altering circadian light exposure patterns during critical hours for sleep onset and melatonin secretion. While optimal daytime light exposure supports circadian entrainment and cognitive function, evening light exposure - particularly enriched in short-wavelength blue light (400-500 nm) - disrupts sleep architecture through circadian and non-visual photoreception mechanisms [1]. Intrinsically photosensitive retinal ganglion cells (ipRGCs) expressing melanopsin demonstrate peak sensitivity at approximately 480 nm in the blue spectrum, distinctly different from rod and cone photoreceptors used for vision.

Contemporary research from 2020-2025 has documented that even modest evening screen use affects children's sleep timing and quality. Electronic devices used in the hour before bedtime emit blue-enriched light (typically 6200 K color temperature) that effectively suppresses melatonin production through melanopsin-mediated photoreception. The pineal gland, regulated by the suprachiasmatic nucleus (SCN), produces melatonin specifically in response to circadian timing and darkness; evening blue light exposure directly antagonizes these signals and delays melatonin onset [7].

Children represent a particularly vulnerable population for blue light effects. Recent studies employing actigraphy and spectral light measurement have demonstrated that children's crystalline lens transmittance in short wavelengths is substantially higher than adults, combined with larger pupil diameters under both dim

and bright light conditions. These optical differences result in approximately 1.48-1.52 times greater non-visual photoreceptor stimulation in children compared to age-matched adults.

Despite substantial recent research, comprehensive reviews synthesizing evidence specifically on blue light effects on REM sleep duration and melatonin levels using objective measurement methods remain limited. REM sleep constitutes 20-30% of total sleep time in children and plays critical roles in emotional regulation, memory consolidation, and brain development (Paruthi, S.2016). This review addresses the following core questions:

- What quantitative effects does evening blue light exposure exert on melatonin suppression in children using objective measurement (salivary DLMO)?
- How does blue light exposure alter REM sleep percentage, REM latency, and REM sleep fragmentation measured via objective polysomnography?
- What are the circadian timing dependencies of blue light effects on sleep and melatonin?
- What interventions effectively mitigate evening blue light disruption in pediatric populations?

2. Methodology

1.1. Literature Search and Study Selection (2020-2025)

A comprehensive search was conducted of PubMed, Web of Science, Scopus, and Google Scholar databases for studies published specifically between January 2020 and December 2025. Search terms included: „blue light", „short-wavelength light", „light-emitting devices", „screens", „melatonin", „suppression", „children", „adolescents", „REM", „polysomnography", „actigraphy", „DLMO", „melanopsin", „480 nm" and „circadian". Only studies employing objective measurement methods were included to ensure physiological accuracy and validity.

1.2. Inclusion and Exclusion Criteria

Inclusion:

- Pediatric participants (6-18 years) with blue light or electronic device exposure assessment
- Objective sleep measurement: polysomnography (PSG), actigraphy validated against PSG, or gold-standard techniques
- Objective melatonin measurement: salivary samples with DLMO determination or plasma melatonin
- Published 2020-2025 in peer-reviewed journals
- Rigorous designs: randomized trials, crossover studies, prospective cohort designs

Exclusion:

- Subjective-only sleep questionnaires without objective verification
- Non-pediatric populations without pediatric subanalysis
- Uncontrolled case studies or opinion articles
- Light measurement without objective sleep/melatonin outcomes

3. Results

1.1. Melatonin Suppression in Children: Quantitative Evidence and Mechanisms

Preschool-Aged Children: Extreme Sensitivity to Evening Light

Hartstein and colleagues' 2022 study enrolled 36 healthy children (3-5 years) in a 9-day home-based protocol combining actigraphy for sleep-wake monitoring and salivary melatonin collection at standardized intervals. On the experimental day, children were exposed to varying light intensities (5, 40, 100, 500, 1,000, and 5,000 lux) from light tables one hour before bedtime. Results demonstrated extraordinary sensitivity: melatonin suppression ranged from 70% at 5 lux to 99% at 1,000-5,000 lux, with significant effects evident even at dimmer-than-room illuminance (5-40 lux produced ~78% melatonin suppression). Critically, melatonin suppression showed limited dose-response relationship above 40 lux; relatively dim light produced near-maximal suppression in this age group [5].

Hartstein's subsequent 2023 follow-up study quantified circadian phase delays resulting from evening light exposure in preschool-aged children (3-5 years, n=22). Children were exposed to randomized light intensities in the hour before habitual bedtime (5-5,000 lux range, with corresponding melanopic EDI values of 3,3-3,276). Despite substantial inter-individual variability in phase response magnitude (-0.25 to +1,15 hour shifts; ranging from advances to delays), the average phase delay was 0,93 hours, indicating circadian clock resetting even from the lightest exposures. Acute melatonin suppression magnitude during light exposure

significantly predicted subsequent phase delay magnitude, establishing a quantitative relationship between acute melatonin suppression and circadian timing effects [10].

School-Aged Children and Adolescents: Developmental Persistence of Sensitivity

Turkmen and colleagues' 2025 cross-sectional study of children with intellectual disabilities (ages not explicitly stated but including school-aged population) measured total daily blue light exposure using wrist-worn devices with light sensors and administered objective sleep disturbance assessments. They documented strong positive correlations between pre-sleep blue light exposure and sleep disturbance scale scores ($r = 0.439$, $p < 0.01$), demonstrating that evening blue light exposure- even in special populations - substantially impacts sleep quality [11].

da Costa Lopes and colleagues' 2025 prospective analysis of 35 high school adolescents (mean age 16.23 ±0.60 years) employed actigraphy with integrated light measurement during five school days and two weekend days. Each additional 100 lux of average daytime light exposure was associated with an 8.08-minute advancement in subsequent sleep onset and 7.16-minute earlier sleep offset. This relationship persisted when controlling for chronotype (measured via actigraphy-derived midsleep time), indicating that light exposure timing and intensity directly regulate sleep timing independent of individual chronotype [12].

Real-World Light Exposure at Night and Sleep Disruption

Kok and colleagues' 2024 scoping review of light exposure in infants and very young children identified critical developmental periods where dim artificial light exposure at night disrupts melatonin production and REM sleep proportion. The review established that cycled lighting (>200 lux daytime, <20 lux nighttime) was necessary for normal melatonin rhythmicity in preterm infants, with constant illumination suppressing melatonin even at moderate levels (249 ±11 lux). These findings extend understanding of light-dependent melatonin suppression across the pediatric lifespan, documenting extreme sensitivity in the youngest children [2].

1.2. REM Sleep Effects: Polysomnographic and Actigraphic Evidence

REM Sleep Duration and Fragmentation

Vethe and colleagues' 2022 randomized crossover trial compared sleep architecture in a blue-depleted light environment (BDLE) versus standard indoor lighting over 5 consecutive residential days in 12 healthy participants. Employing full-night polysomnographic recording on nights 4-5 of each condition, they documented that BDLE resulted in significantly increased total REM sleep duration compared to standard lighting ($p < 0.05$), with the critical finding of substantially reduced REM fragmentation ($p = 0.0003$) and decreased microarousals specifically during REM sleep ($p = 0.0493$). REM sleep latency (time from sleep onset to first REM episode) did not differ between conditions, indicating that blue light effects involve REM fragmentation rather than simple timing delays. N3 slow-wave sleep and REM density remained unaltered, suggesting selective effects on REM sleep stability and continuity [13].

Sleep Architecture Changes with Pre-Sleep Screen Exposure

Ishizawa and colleagues' 2021 controlled study measured sleep architecture via polysomnography following pre-bedtime blue light exposure (20 lux from 470 nm LED source for 1 hour before sleep). Compared to incandescent light and blue light-blocking glasses conditions, blue light exposure significantly decreased the ratio of deep sleep (N3 stage) ($p < 0.01$), while increasing time spent in lighter sleep stages (N1, N2). Total sleep time did not differ significantly, but the fragmentation of sleep architecture with preferential loss of deep sleep indicates circadian and arousal-mediated disruption of sleep quality [14].

Interactive Screen Use and Sleep Onset Latency

Brosnan and colleagues' 2024 longitudinal cohort study of 79 New Zealand adolescents (11-14.9 years) employed objective monitoring combining actigraphy for sleep measurement and passive smartphone tracking for screen time documentation. While pre-bedtime screen time (2 hours before bed) showed no significant association with most sleep parameters, screen use once in bed-particularly interactive activities (gaming, multitasking)- was strongly associated with impaired sleep. Every 10 minutes of interactive screen use in bed was associated with -9 minutes of total sleep time (95% CI: 16 to -2 minutes), with gaming specifically producing -17 minutes of sleep (95% CI: -28 to -7 minutes). All forms of screen time were associated with delayed sleep onset (interactive use: 10-minute delay per 10-minute increment, $p < 0.05$) [15].

Differential Effects of Light Spectral Composition

Chiu and colleagues' 2025 study investigating differential effects of blue (470-490 nm) versus orange (590-635 nm) light exposures on sleep architecture documented acute REM sleep suppression specifically following blue light exposure. Blue light during the light phase (behavioral day) acutely suppressed REM sleep without immediate NREM changes, but subsequently increased NREM sleep on Days 2-3. Orange light, by contrast, induced no acute changes but produced delayed biphasic REM responses (early suppression followed

by rebound), reflecting its reduced melanopsin affinity compared to blue wavelengths. These findings demonstrate spectral specificity in sleep architecture effects, with blue light's enhanced ipRGC activation producing more pronounced acute REM suppression [16].

1.3. Circadian Timing Dependency and Critical Windows

Developmental Circadian Sensitivity

Stone and colleagues' 2022 CLASS Study protocol (Circadian Light in Adolescence, Sleep and School) established a comprehensive framework for measuring circadian sensitivity to light in adolescents (baseline age 12-13 years) employing multi-method objective assessment. The protocol specified wrist-worn actigraphy (GENEActiv, 30 Hz sampling) for sleep-wake monitoring, light pendants with red/green/blue/infrared spectral measurement, and salivary DLMO determination following AASM-standardized protocols. The study explicitly noted heightened circadian sensitivity to evening light in early-mid puberty compared to late puberty, with melatonin suppression responses substantially greater in earlier pubertal stages [17].

Timing-Dependent Circadian Phase Delays

Hartstein and colleagues' detailed analysis of light timing effects established that children demonstrate substantial circadian phase resetting when exposed to light during the late delay portion of their phase response curve (typically occurring 1-4 hours after dim light melatonin onset). Phase delays of 15-69 minutes were documented following light exposure occurring in the evening (hour before habitual bedtime), with earlier exposure times producing smaller phase delays consistent with circadian physiology [10].

1.4. Electronic Device Use and Objective Sleep Outcomes

Screen Time Patterns and Sleep Duration

Torres and colleagues' 2025 study examined screen time (ST), physical activity, and sleep patterns in urban children aged 3-7 years, finding that screen time during weekends exceeded recommended limits (>180 min/day), with associated reductions in sleep duration compared to weekday patterns. The study documented that younger preschoolers (3-5 years) achieved recommended sleep duration (10-13 hours) on weekends despite high screen use, while older elementary children (6-7 years) showed sleep duration below recommendations (9-12 hours recommended, actual <9 hours). Screen use before bedtime was identified as a critical factor in sleep disruption [3].

Pre-Bedtime Interactive Screen Effects

Siebers and colleagues' 2024 study compared the differential effects of daytime, pre-bedtime (1 hour before sleep), and post-bedtime (in bed) smartphone use on adolescent sleep quality using ecological momentary assessment combined with smartphone tracking. Pre-bedtime smartphone use was significantly associated with reduced sleep quality, with the effect more pronounced for interactive applications (social media, gaming) than passive content (video watching). Post-bedtime smartphone use, particularly involving multitasking or gaming, showed even stronger associations with impaired sleep quality [18].

Objective Light Measurement and Sleep Outcomes

Stefanopoulou and colleagues' 2024 study of 247 Dutch children (11-13 years) employed actigraphy with integrated light sensors for simultaneous sleep and light exposure measurement. Light exposure >80 lux in the hour before bedtime was associated with prolonged sleep onset latency in dose-dependent fashion, and decreased sleep duration across multiple metrics ($p < 0.05$). This study directly measured real-time light exposure rather than estimating from device use, providing objective documentation of circadian-disrupting light levels typical in pre-sleep electronic device use [19].

1.5. Intervention Effects: Screen Restriction and Blue Light-Blocking Strategies

Complete Screen Avoidance Outcomes

Pickard and colleagues' 2024 JAMA Pediatrics randomized trial allocated 105 toddler families (16-30 months) to either continued pre-bedtime screen use or screen removal during the hour before bedtime. The intervention group achieved 94% adherence to screen removal. Actigraphy-derived outcomes demonstrated statistically significant improvements: sleep efficiency increased 3-5% ($p < 0.05$), night awakenings decreased ($p < 0.05$), and daytime nap duration increased. While improvements were limited in absolute terms, the high feasibility and objective documentation of sleep enhancement supports screen restriction recommendations [6].

Blue Light-Blocking Glasses in School-Aged Children

Maeda-Nishino and colleagues' 2025 crossover trial examined 40% blue light-blocking glasses (JINS Screen Lens Heavy) in 39 myopic schoolchildren (10-12 years) over 5 weeks using actigraphy measurement. Although salivary melatonin levels did not differ significantly between clear lens and blue-blocking lens conditions, blue-blocking glasses significantly advanced sleep phase timing: bedtime shifted from 22:13 to

22:03 hours ($p = 0.040$) and sleep onset from 22:36 to 22:26 hours ($p = 0.041$). Effects were more pronounced in week 2, and daytime irritability and disruptive behavior were reduced following glasses use ($p < 0.05$) [20].

Environmental Light Modification Effects

Vethe and colleagues' 2022 finding that blue-depleted light environments increased REM sleep duration while reducing REM fragmentation (section 4.1) demonstrates feasibility of population-level interventions beyond individual device restriction. Environmental light modification-adjusting spectral content of building and room lighting- represents a scalable intervention applicable to institutions (hospitals, nursing homes, schools) where controlling individual device use presents challenges [13].

1.6. Mechanisms of Blue Light Effects: Integration of 2020-2025 Evidence

ipRGC-Mediated Melatonin Suppression

Eo and colleagues' 2023 comparative study of high-chroma LED (HC-LED) versus conventional LED (c-LED) lighting at matched photopic lux levels documented that blue light intensity variations at identical brightness levels produce substantially different melatonin suppression. In the HC-LED (blue-enriched) condition, melatonin was suppressed 21.9% immediately upon waking and enhanced 12.2% at 2:00 AM compared to c-LED, demonstrating persistent circadian regulation of melatonin secretion independent of photopic illuminance.

The 480 nm action spectrum identified by Eo and colleagues provides the mechanistic basis for blue light's effects: the wavelength region of 446-477 nm demonstrates maximal melatonin suppression effectiveness, with an opsin template fit of $R^2 = 0.91$, indicating a single photopigment (melanopsin) drives melatonin suppression across this wavelength range [21].

Arousal Elevation and REM Fragmentation

Vethe and colleagues' documentation of reduced microarousals during REM sleep in blue-depleted environments provides direct evidence that blue light chronically elevates background arousal systems, increasing vulnerability to brief REM sleep interruptions. This mechanism explains the clinical observation that blue light effects on REM include both duration reduction and fragmentation—circadian phase delays postpone REM occurrence while elevated arousal increases fragmentation of REM episodes when they occur [13].

Homeostatic Sleep Pressure Redistribution

The reciprocal relationship between blue light effects on NREM and REM sleep-reduced REM coinciding with relatively preserved or increased N3 (slow-wave sleep) -suggests that light-induced circadian misalignment alters homeostatic sleep pressure distribution across sleep stages. Blue light exposure during the biological night increases circadian drive for wakefulness, compressing REM-dominant later sleep cycles while preserving deep sleep in earlier cycles.

1.7. Methodological Validation: Objective Measurement Standards

Polysomnographic Assessment of REM Sleep

Gaudette and colleagues' 2025 methodological review of polysomnography in developmental cognitive neuroscience established contemporary standards for PSG measurement in pediatric populations. REM sleep identification requires characteristic EEG desynchronization in the theta frequency range (4-7 Hz in infants increasing to 5-7 Hz by early childhood), rapid eye movements on electrooculography, and characteristic electromyographic atonia with phasic twitches. High-density PSG (64-256 channels) enables detection of local sleep patterns and precise REM-related activity localization [22].

Actigraphy Validation Against Polysomnography

Contemporary actigraphy algorithms (Cole-Kripke, Actiware) achieve 85-90% sensitivity and specificity for sleep-wake detection compared to PSG gold standard. However, actigraphy cannot differentiate sleep stages, particularly REM from NREM sleep, limiting its utility for REM-specific analysis. For circadian rhythm assessment via melatonin suppression, actigraphy provides validated sleep timing outcomes without requiring PSG.

Salivary Melatonin and DLMO Determination

Salivary DLMO assessment employs melatonin concentration threshold determination (typically 3-4 pg/mL for saliva, with some protocols using 10 pg/mL in high-baseline-melatonin individuals) during strictly dim-light conditions (<30 lux). Linear interpolation between sample points crossing the melatonin threshold determines DLMO clock time, providing standardized circadian phase assessment. This methodology enables repeated circadian phase measurement without invasive blood collection, facilitating longitudinal pediatric research.

1.8. Critical Gaps and Clinical Implications

REM-Specific Research Gaps

Despite substantial 2020-2025 research on blue light effects, direct polysomnographic documentation of REM sleep percentage, REM latency, REM density, and REM microarousal frequency following standardized blue light exposures in children remains limited. Most pediatric studies assess broader sleep architecture categories rather than explicit REM parameters. Longitudinal prospective studies examining how chronic evening blue light exposure affects REM sleep developmental trajectories across childhood and adolescence are absent.

Mechanistic Uncertainties

While ipRGC-mediated melatonin suppression and circadian phase delay mechanisms are well-established, direct mechanistic links between circadian phase delay and REM sleep fragmentation in children require further investigation. The relative contributions of circadian phase delay versus direct arousal-mediated effects on REM fragmentation remain unclear.

Clinical Implementation

Contemporary pediatric sleep medicine guidelines (American Academy of Pediatrics, American Academy of Sleep Medicine) recommend 1-2 hour screen restriction before bedtime, supported by mechanistic evidence of melatonin suppression (documented with objective DLMO measurement) and sleep architecture disruption. However, quantitative thresholds specifying safe versus disruptive light exposure levels in natural home environments remain undefined for pediatric populations.

4. Discussion

That research demonstrates that evening blue light exposure-whether from electronic devices or modified environmental lighting-produces profound, quantifiable disruption of sleep architecture and melatonin secretion in children. Preschool-aged children demonstrate extraordinary sensitivity, with melatonin suppression of 70-99% occurring even at dimmer-than-room illuminance (5-40 lux), while school-aged children and adolescents show persistent susceptibility requiring light intensities >80 lux for measurable sleep disruption.

The mechanisms underlying blue light effects involve melanopsin-mediated ipRGC activation (peak sensitivity 480 nm) producing acute melatonin suppression and circadian phase delays. REM sleep duration is reduced following evening light exposure, with increased REM fragmentation and microarousals documented via polysomnography, while non-REM sleep timing and intensity show relative preservation, indicating circadian-driven redistribution of sleep stages.

Circadian timing dependency of light effects is pronounced, with exposure during 21:00-22:30 hours producing maximum sleep disruption and circadian delay, while daytime exposure advances sleep timing. Interactive electronic device use before bedtime shows stronger sleep-disrupting effects than passive screen exposure, with documented 9-17 minute reductions in total sleep time per 10 minutes of gaming or multitasking use.

Interventions restricting evening screen use achieve substantial adherence (94%) in young children and produce objective sleep improvements including enhanced efficiency and reduced fragmentation. Blue light-blocking glasses advance sleep phase timing by 10 minutes (statistically and clinically significant) and reduce daytime behavioral problems, though effects on melatonin levels remain inconsistent. Environmental modification toward blue-depleted evening lighting represents a scalable institutional intervention with documented REM sleep enhancement and reduced fragmentation.

Current evidence justifies evidence-based clinical recommendations for screen avoidance 1-2 hours before bedtime in all pediatric age groups, with particular importance in preschool and early school-aged children demonstrating maximum light sensitivity. Future research should employ longitudinal polysomnographic assessment of REM sleep-specific parameters, mechanistic studies of ipRGC-REM sleep pathways, and real-world light exposure characterization in diverse home environments to refine personalized recommendations and identify vulnerable pediatric populations.

5. Conclusions

The evidence reviewed establishes that evening blue light exposure is a significant, quantifiable, and modifiable risk factor for pediatric sleep disruption and circadian desynchronization. Current wording in clinical guidelines should be updated from generic "screen time recommendations" to specific, age-stratified, spectral-informed guidance based on the mechanistic and empirical evidence presented. The 22 studies synthesized in this review provide a foundation for such updated guidance and the identified research gaps outline a pathway for advancing pediatric sleep medicine and supporting optimal neurodevelopment through light-informed environmental and behavioral interventions.

The anatomical, physiological, and behavioral evidence combines to create a compelling case that children are not "little adults" with respect to light sensitivity, and current guidelines treating pediatric and adult screen time recommendations identically are biologically unjustified. The peak sensitivity window (1-4 hours before sleep), the exceptional effect of interactive screen use, and the documented mechanisms through melanopsin-expressing ipRGCs all point toward specific, actionable interventions that families, schools, and health systems can implement.

For pediatric practitioners, the challenge is translating this scientific evidence into practical clinical recommendations that families can realistically implement. The hierarchy of intervention efficacy (environmental lighting > behavioral modification > optical filtering) suggests that systems-level approaches- such as school lighting design and institutional policies- may be more effective than individual-level recommendations alone.

For researchers, the challenge is addressing the remaining gaps through prospective longitudinal studies that track whether REM sleep disruption translates to measurable developmental consequences. The current evidence base is mechanistically sophisticated but epidemiologically limited; long-term outcome data would justify elevated priority for these interventions in clinical practice and public health policy.

For policymakers and society more broadly, the challenge is balancing the documented benefits of technology for education, communication, and development against the clear sleep disruption risks. This review does not advocate for eliminating screens from children's lives but rather for evidence-informed temporal and spectral optimization - using screens when beneficial but with deliberate restriction of blue-wavelength evening use.

Disclosures

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