

Review

Evening light exposure and sleep phase shifts in adolescence and childhood: Literature review

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ABSTRACT

Sleep regulation in children and adolescents relies on the interaction between homeostatic and circadian processes. The internal biological clock, located within the suprachiasmatic nuclei, is primarily synchronized by light. This light is detected by intrinsically photosensitive retinal ganglion cells (ipRGCs), which are highly sensitive to short-wavelength blue light. Evening exposure to LED lighting and electronic screens can suppress melatonin secretion, promoting sleep phase delay. Children and adolescents are exceptionally vulnerable to this effect due to both anatomical and developmental factors. Combined with cognitive arousal from social media and the Fear of Missing Out (FOMO), this creates a "perfect storm," resulting in behavioral bedtime procrastination and a natural shift toward an evening chronotype. While it is crucial to distinguish this common developmental and behavioral shift from a clinical disorder, cumulative circadian disruption and social jetlag remain significant risk factors for Delayed Sleep-Wake Phase Disorder (DSWPD). Furthermore, chronic sleep restriction exhibits observational associations with poorer academic performance, potential long-term metabolic disturbances (such as obesity and insulin resistance), and an elevated risk of mood disorders and depression. Available evidence highlights the critical importance of light hygiene, including limiting evening screen exposure and promoting morning natural light. Medical interventions, such as melatonin and chronotherapy, should be carefully reserved for severe clinical cases. Finally, future longitudinal studies utilizing personal light dosimetry are needed, alongside systemic solutions like delayed school start times, to protect pediatric sleep health.

KEYWORDS

children, circadian rhythm, adolescence, evening light exposure, sleep phase

Introduction

Sleep is a fundamental biological process that is essential for the normal physical, psychological, and cognitive development of children and adolescents [1,2]. During periods of rapid growth and maturation, adequate sleep duration and quality are critical for synaptic plasticity, and emotional regulation [1,3]. Chronic sleep deprivation in this population has been consistently associated with poorer academic performance, metabolic disturbances, and an increased risk of anxiety disorders and depression [4,5,6,7].

However, the modern human environment has undergone profound technological and urban transformations [8,9]. The natural light-dark cycle has largely been replaced by continuous exposure to artificial lighting and LED-emitting electronic devices, including smartphones, tablets, and computers [9,10,11]. The widespread availability of mobile technologies has resulted in

children and adolescents spending considerable amounts of time using screens during the evening and nighttime hours, directly disrupting normal sleep architecture [12,13,14,15,16].

Understanding the mechanisms by which artificial light disrupts human sleep requires reference to Borbély's classical two-process model of sleep regulation. According to this model, sleep onset and sleep duration are determined by the interaction of two independent processes:

- Homeostatic Process (Process S): Represents the progressive accumulation of sleep pressure during wakefulness, which gradually increases with time spent awake and dissipates during sleep.
- Circadian Process (Process C): Represents the endogenous biological clock generated by the suprachiasmatic nuclei (SCN) of the hypothalamus, which regulates the cyclical propensity for sleep and wakefulness independently of the duration of prior wakefulness [8,17].

The principal neurohormonal regulator of the circadian process is melatonin, a hormone synthesized by the pineal gland [8,17]. Under conditions of darkness, melatonin secretion rises rapidly during the evening, signaling the onset of the biological night and preparing the organism for sleep [8,17,18].

The most important environmental synchronizer (zeitgeber) of the endogenous circadian rhythm is light reaching the retina [8,17,19]. Specialized intrinsically photosensitive retinal ganglion cells (ipRGCs), which contain the photopigment melanopsin, exhibit peak sensitivity to short-wavelength blue light within the range of approximately 460-480 nm [19,20,21]. However, the circadian response to light is not determined by wavelength alone; it is a complex function of light intensity, duration, timing of exposure, and the individual's prior photic history. When evening exposure to this wavelength is sufficiently intense and prolonged, as is often the case with LED-based electronic displays, it generates a false dawn signal to the suprachiasmatic nuclei, resulting in rapid suppression of melatonin secretion and disruption of clock gene transcription [10,21,22,23,24,25].

Methods

This study was conducted as a narrative literature review to synthesize current evidence regarding the impact of evening light exposure on sleep phase shifts and circadian biology in children and adolescents. A comprehensive electronic literature search was performed using the PubMed database between November and December 2025 for peer-reviewed literature published between December 2003 and December 2025. The search strategy employed combinations of Medical Subject Headings terms and relevant keywords, structured as follows: ("children" OR "adolescents" OR "pediatric") AND ("evening light" OR "blue light" OR "screen time") AND ("sleep phase shift" OR "melatonin suppression" OR "delayed sleep phase disorder"). Additionally,

reference lists of relevant review articles and primary studies were manually screened. To be included, studies had to be peer-reviewed original research or systematic reviews and meta-analyses investigating pediatric or adolescent cohorts exposed to evening artificial light or screen-based media, as well as adult studies providing essential comparative or mechanistic insight into circadian physiology. Studies conducted exclusively on animal or in vitro models, non-peer-reviewed literature, and publications completely unrelated to sleep and circadian biology were excluded. The study selection followed a three-step process consisting of title and abstract screening, full-text eligibility assessment, and thematic categorization according to age groups, exposure parameters, and circadian mechanisms. Ultimately, a total of 37 publications met all inclusion criteria and were included in the qualitative narrative synthesis.

Results

Neurobiological basis: Light and the biological clock

For decades, it was believed that light perception in mammals was mediated exclusively by the classical photoreceptors, namely rods and cones. A breakthrough in neurobiology came with the discovery of a third class of photoreceptors, the intrinsically photosensitive retinal ganglion cells (ipRGCs) [8,20]. These cells express the unique photopigment melanopsin, which exhibits peak sensitivity to short-wavelength light, particularly within the 460-480 nm range corresponding to the blue-light spectrum [19,20].

Unlike rods and cones, which are responsible for rapid image detection and detailed visual perception, ipRGCs specialize in encoding ambient light intensity over prolonged periods [8,17]. The axons of ipRGCs form the retinohypothalamic tract (RHT), a monosynaptic pathway that branches from the optic nerve and projects directly to the hypothalamus while bypassing the cortical visual processing centers entirely [8]. Consequently, circadian plasticity and synchronization occur independently of conscious image-forming vision, explaining why even blind individuals with an intact ipRGC-RHT pathway are capable of maintaining normal circadian rhythms [17]. While melanopsin has a peak sensitivity to wavelengths between 460 and 480 nm, the circadian system integrates multiple properties of the light stimulus. Artificial light sources enriched in blue wavelengths, including smartphone and tablet displays as well as LED lighting, can be interpreted by the RHT as a powerful signal of biological morning, provided that the intensity, duration, and timing of exposure are sufficient to overcome the buffering effects of daytime light exposure [10,11].

The primary target of the retinohypothalamic tract is the suprachiasmatic nuclei (SCN) of the hypothalamus, which serve as the central circadian pacemaker in mammals [8,17]. The SCN comprises approximately 20,000 neurons that generate autonomous circadian oscillations through

intracellular transcriptional-translational feedback loops involving clock genes, including CLOCK, BMAL1, PER, and CRY [8,23]. Although these endogenous cycles last slightly longer than 24 hours in humans, the SCN requires daily adjustment to synchronize the organism with the astronomical day-night cycle. This process, known as entrainment, is driven primarily by light, the most important external time cue (zeitgeber) [17,26].

When blue light activates ipRGCs, neural impulses travel through the RHT to SCN neurons, inducing the release of the neurotransmitters glutamate and pituitary adenylate cyclase-activating polypeptide (PACAP) [8]. This signaling cascade promotes calcium influx into SCN neurons and stimulates the expression of the Period (PER1 and PER2) genes [23]. Depending on the timing of light exposure, these molecular events induce a phase shift of the intracellular circadian clock. The SCN subsequently coordinates peripheral oscillators located throughout the body, including those in the liver, skeletal muscles, and cardiovascular system, thereby maintaining physiological synchronization across tissues [5,6,8].

A key mechanism through which the SCN regulates the sleep-wake cycle is the control of melatonin secretion by the pineal gland. Under conditions of darkness, the SCN initiates neural signaling that stimulates melatonin synthesis, thereby chemically signaling the onset of the biological night [8,18]. In contrast, direct light stimulation of the SCN during the evening or nighttime activates an inhibitory sympathetic pathway, resulting in the rapid suppression of melatonin production [19,25].

The circadian response to light depends critically on the timing of exposure and is described by the Phase Response Curve (PRC) [17,26].

- Light exposure during the biological morning (shortly after the minimum core body temperature) produces a phase advance, shifting the biological clock to an earlier time and facilitating earlier awakening.
- Light exposure during the evening and early biological night induces a phase delay, whereby the circadian system interprets the stimulus as an extension of daytime, postponing physiological readiness for sleep [17,22].

Evening exposure to light, particularly that emitted by LED-based electronic devices, markedly suppresses melatonin synthesis [10,21,25]. This delays the Dim Light Melatonin Onset (DLMO), the physiological point at which melatonin concentrations begin to rise under dim-light conditions [24,27]. Consequently, the circadian phase is delayed (phase delay) [22,26,28].

Studies conducted in controlled laboratory settings demonstrate that this effect is substantially more pronounced in children and adolescents. Their circadian systems exhibit significantly greater sensitivity to light-induced melatonin suppression, responding to both lower light intensities and

shorter exposure durations compared to adults [24,25,27]. We can assume, that evening use of electronic technologies contributes to delayed sleep onset and chronic sleep restriction in young individuals [1,2,12,13,14,15,16]. Shortened or irregular sleep duration has been consistently correlated with adverse health outcomes, including metabolic dysregulation, weight gain, reduced cognitive performance, and frequently co-occurs with mood disorders and depression [3,4,5,6,7,29,30].

Developmental specificity: Childhood and adolescence

Analysis of the available evidence indicates that the visual and circadian systems of children and adolescents differ physiologically from those of adults, resulting in a markedly greater sensitivity to evening light exposure [8,17,22]. Anatomical differences in ocular structure play a pivotal role in this phenomenon. During childhood, the pupil diameter is naturally larger, allowing a greater amount of light to reach the retina. In addition, the crystalline lens is more transparent, particularly to short-wavelength (blue) light [19,20,21]. Consequently, retinal photoreceptors, especially melanopsin-containing intrinsically photosensitive retinal ganglion cells (ipRGCs), are more strongly stimulated by evening light exposure [17,19].

As a result, clinical observations indicate that children exhibit substantially greater light-induced melatonin suppression than adults when exposed to comparable evening light stimuli [18,27]. An experimental study has demonstrated that 1 hour of evening bright light exposure (~1000 lux) produces a marked reduction in salivary melatonin concentrations, revealing that the circadian system of preschool-age children is highly sensitive to light [31]. Specifically, preschool- and school-aged children can exhibit nearly complete melatonin suppression under these controlled lighting conditions, which typically produce only minimal neuroendocrine changes in adults. These findings indicate that the pediatric circadian system has a markedly lower lux threshold for sensitivity to artificial light [27,31].

During adolescence, these processes undergo further complex modification as a consequence of neurodevelopmental and hormonal maturation. From a biological perspective, one of the most important changes is the physiological, puberty-related delay of the circadian phase [1]. The endogenous biological clock shifts naturally toward a later schedule, as reflected by a delayed Dim Light Melatonin Onset (DLMO) [1,18]. Importantly, experimental protocols measuring melatonin suppression indicate that early and mid-puberty coincide with the period of greatest circadian sensitivity to evening light exposure [27]. Consequently, adolescents are biologically predisposed to light-induced phase delays, resulting in progressively later sleep onset and reinforcement of an evening chronotype [23,24,32].

Simultaneously, the homeostatic process regulating the accumulation of sleep pressure also changes during adolescence. Compared with younger children, adolescents accumulate sleep pressure more slowly, enabling them to remain awake for longer periods despite increasing fatigue [1]. However, this biological capacity for prolonged wakefulness becomes maladaptive when combined with evening exposure to artificial light, as the weakened homeostatic drive is insufficient to counterbalance the alerting effects induced by light-mediated circadian stimulation [25].

These biological predispositions toward delayed sleep timing interact synergistically with profound behavioral and social changes, creating what has been described in the literature as a "perfect storm" for sleep deprivation [1]. During this developmental period, adolescents gain increasing autonomy over their bedtime decisions [1,2]. This greater independence coincides with growing academic demands, frequently requiring late-night studying under artificial household lighting [2,4].

The most important modifiable behavioral factor is the widespread use of digital media immediately before bedtime [12,13,14]. The use of smartphones, tablets, and laptops in bed exposes adolescents to intense blue-enriched LED light emitted at a very close viewing distance [3,10,11,28]. This effect is further amplified by peer pressure and the desire for continuous online social interaction, commonly referred to as the Fear of Missing Out (FOMO), which encourages prolonged nighttime engagement with digital platforms [15,16].

These activities are accompanied by additional emotional and cognitive arousal that, together with light-induced melatonin suppression and biologically driven circadian phase delay, has been consistently linked to chronic sleep deprivation, marked delays in sleep timing, and progressive destabilization of circadian rhythms throughout the week [5,6,16,29,30].

Sources of evening light exposure

Sleep phase delays in children and adolescents are closely associated with the contemporary light environment, which differs markedly from the natural light-dark cycle during the evening hours [8,17]. Analysis of the sources of evening light exposure identifies three major, synergistically interacting factors: artificial household lighting, electronic device screens, and the interaction between visual stimuli and cognitive-emotional arousal.

The traditional home lighting environment has undergone a profound technological transformation [32]. The widespread replacement of conventional incandescent bulbs with energy-efficient fluorescent and light-emitting diode (LED) lighting has substantially altered the spectral composition of light reaching the human eye [19,22]. Modern indoor lighting with a high correlated color temperature is characterized by a pronounced emission peak within the short-

wavelength range (460–480 nm), corresponding to blue light [19]. Importantly, the physiological impact of this lighting is inextricably linked to illuminance levels, distance from the light source, and cumulative exposure time [17,25].

Retinal photoreceptors in children and adolescents have been shown in controlled exposure studies to exhibit significantly greater sensitivity to this wavelength range compared to adults, reacting more strongly to equivalent levels of photon flux [25,27]. Blue light directly activates melanopsin-containing intrinsically photosensitive retinal ganglion cells (ipRGCs), which, through the retinohypothalamic pathway, suppress pineal melatonin secretion [8,18,20,27]. Experimental studies have demonstrated that exposure to bright, blue-enriched evening lighting induces substantial phase shifts in both the transcriptional circadian clock and hormonal rhythms [23,24,25]. In preschool- and early school-aged children, experimental models simulating evening exposure to standard indoor lighting (often below 50 lux) have been shown to produce profound suppression of melatonin synthesis, thereby directly preventing sleep initiation at the physiologically appropriate time [18,27,31].

Electronic devices, including smartphones, tablets, personal computers, and televisions, have become the most significant and pervasive sources of evening light exposure among children and adolescents [12,13]. These devices emit intense blue-enriched light immediately before bedtime [11,16]. Whereas television screens are typically viewed from a distance of several meters, smartphones and tablets are held close to the eyes, dramatically increasing illuminance (measured in lux) at the corneal surface and the photon flux stimulating ipRGCs [14,15].

A growing body of evidence demonstrates that evening exposure to LED-emitting screens directly disrupts circadian physiology by reducing subjective sleepiness, delaying the nocturnal melatonin peak, and adversely affecting sleep architecture during subsequent sleep cycles [10,21,28,33].

Children and adolescents who use portable electronic devices while in bed exhibit significantly shorter sleep duration and more pronounced circadian phase delays [14,16,28]. Smartphone use immediately before bedtime prolongs sleep latency and alters evening sleepiness, although experimental findings on its direct impact on overnight declarative memory consolidation remain nuanced, with some studies showing no significant impairment under specific exposure conditions, while overall sleep restriction continues to negatively affect next-day cognitive performance [3,11,29,30]. The cumulative effects of prolonged screen time are considered a significant risk factor associated with the development of Delayed Sleep-Wake Phase Disorder (DSWPD) among adolescents [26,34].

The adverse effects of evening screen exposure are not limited to the direct biological influence of light on the retina. Rather, they involve a complex interaction between photic stimulation and

cognitive-emotional activation [1,2]. Digital content consumed through social media platforms, instant messaging applications, and video games requires continuous cognitive engagement and frequently elicits strong emotional responses, ranging from excitement to anxiety or frustration [13].

Such engagement activates the sympathetic nervous system, leading to increased secretion of cortisol and catecholamines, thereby directly counteracting the physiological relaxation required for sleep initiation. This phenomenon further reinforces behavioral bedtime procrastination, whereby adolescents intentionally delay turning off electronic devices in order to continue online social interactions [4,14]. The combination of blue light-induced melatonin suppression and heightened psychological arousal creates a substantial barrier to sleep initiation, ultimately contributing to chronic sleep deprivation [14,15]. Consequently, alongside shifting circadian preference toward a more pronounced evening chronotype, it shows a strong observational association with elevated rates of mood disorders, anxiety, and depression [6,7].

Consequences of sleep phase delay in young individuals

A shift in sleep timing toward an evening chronotype is a common phenomenon during adolescence, resulting from the combined influence of psychosocial factors and profound biological changes. In its most severe form, this process manifests clinically as Delayed Sleep-Wake Phase Disorder (DSWPD). In this age group, the pathophysiology of DSWPD is associated with a prolonged endogenous circadian period and a slower accumulation of homeostatic sleep pressure [1,26]. Furthermore, experimental trials confirm that the adolescent circadian system exhibits significantly greater sensitivity to melatonin suppression induced by short-wavelength evening light (blue light), meaning that even moderate screen luminance can markedly delay sleep initiation [19,20,24,25,27,31].

The diagnostic criteria for DSWPD in children and adolescents include a persistent delay in the major sleep episode lasting for at least three months, typically exceeding two hours relative to socially desired or required sleep schedules. Affected individuals experience a pronounced inability to initiate sleep at an earlier time while maintaining normal sleep architecture and duration when allowed to sleep according to their endogenous circadian preference without externally imposed wake-up times [26,35]. Diagnosis requires the exclusion of other sleep disorders and confirmation of the sleep pattern using actigraphy or sleep diaries over a period of at least two weeks [28,34]. The rapid advancement of digital technology and the widespread evening use of LED-emitting electronic devices [10,11,12,13,14,15,16,21] are in direct conflict with the externally imposed schedules governing the daily lives of young people. The discrepancy between the biological clock,

shifted by evening light exposure, and fixed school schedules requiring early awakening gives rise to the phenomenon known as social jetlag [2,8,16,22,32].

While adolescents are able to follow their naturally delayed chronotype on weekends and school holidays, they are compelled to substantially shorten their sleep duration on school days. This results in the cumulative accumulation of chronic sleep debt. Instead of the recommended 8 to 10 hours of sleep per night, many adolescents obtain fewer than 6 to 7 hours of sleep during the school week [1,14]. Such persistent sleep deprivation disrupts transcriptional rhythms in peripheral tissues, further aggravating internal circadian desynchronization [8,23].

The consequences of chronic sleep loss become most apparent in the domain of cognitive functioning. Sleep deprivation associated with evening exposure to artificial light, particularly from smartphones and computers, directly impairs sustained attention, working memory, and the consolidation of declarative memory [3,10,11,21]. In the school setting, this manifests as excessive daytime sleepiness, microsleep episodes, and impaired concentration [29]. Longitudinal studies show that social jetlag and sleep irregularity are strongly correlated with lower academic performance and increased absenteeism, although these relationships are often mediated by broader psychosocial and environmental factors [30].

The close relationship between the circadian system and the neurobiology of mood makes delayed sleep phase a significant risk factor for psychiatric disorders [6]. Reduced sleep duration and impaired sleep continuity are strongly associated with symptoms of anxiety, anhedonia, and clinical depression in adolescents [7]. Moreover, sleep deprivation compromises prefrontal cortex function, resulting in impaired emotional regulation, reduced inhibitory control, and markedly increased impulsivity. Consequently, adolescents become more vulnerable to risk-taking behaviors, including substance use and self-harm [1,6].

Chronic social jetlag also exerts profound adverse effects on metabolic homeostasis. Circadian disruption combined with insufficient sleep alters the secretion of appetite-regulating hormones, leading to decreased leptin levels and increased ghrelin concentrations [5]. Together with the increased tendency toward nighttime eating associated with evening light exposure, these hormonal changes are associated with an elevated risk of overweight and obesity. In the long term, chronic sleep deficiency has been linked to peripheral insulin resistance, reduced glucose tolerance, and elevated cortisol secretion, which correlate with the development of carbohydrate metabolism disorders, including type 2 diabetes mellitus at a younger age [5,8].

Intervention strategies and recommendations

In response to the growing prevalence of evening light exposure and the resulting sleep phase delays, intervention strategies have become increasingly important [8,22]. These approaches aim

to minimize the adverse effects of artificial lighting on the circadian system of children, adolescents, and adults, whose vulnerability to circadian disruption reflects the interaction of biological and psychosocial factors [1,24,25,27,31].

The cornerstone of non-pharmacological prevention is the implementation of strict light hygiene practices within the home environment. Given the well-established sensitivity of the human circadian system to short-wavelength blue light (approximately 460-480 nm) [17,19,20], the primary recommendation is to avoid the use of light-emitting electronic devices (the "no screens" principle) for at least one to two hours before the intended bedtime [12,14]. Blue-enriched light emitted by LED screens of smartphones, tablets, and computers has been shown to strongly suppress melatonin secretion while simultaneously increasing alertness [3,10,11,21,25].

When complete avoidance of portable electronic devices is not feasible, the use of blue-light filters or blue-blocking glasses is often considered as a potential compensatory strategy. Although certain studies have demonstrated that blue-light-blocking eyewear can help preserve evening melatonin secretion and mitigate circadian phase delay in adolescents, the overall evidence remains mixed. The heterogeneity of research findings and methodological differences across studies should be taken into account when evaluating their long-term clinical efficacy [36,37]. Equally important is the regulation of ambient household lighting through dimming indoor illumination and replacing high color-temperature light sources with warmer lighting, thereby reducing melatonin suppression before sleep [32].

Alongside limiting evening light exposure, intentional morning exposure to bright natural light represents another key intervention [16]. According to the Phase Response Curve (PRC), retinal light stimulation during the early biological morning induces a phase advance of the circadian rhythm [17]. This effect is particularly important during adolescence, when the physiological delay of the circadian phase makes early morning awakening more difficult [27]. Exposure to natural daylight immediately after waking produces a sustained advance in the timing of the minimum core body temperature and the onset of evening melatonin secretion, thereby facilitating earlier sleep onset and increasing total sleep duration [23].

The effectiveness of these technical interventions depends largely on behavioral and educational support. Parents play a central role in regulating screen-related behaviors by establishing clear household rules, such as prohibiting smartphones and other electronic devices in bedrooms during the night [13,15].

School-based educational programs promoting circadian health and sleep hygiene have been proposed to improve sleep behaviors in adolescents, with large-scale prospective frameworks such as the CLASS study ongoing to evaluate their long-term feasibility and health outcomes [4].

Peer-based and system-wide educational initiatives may mitigate the adverse consequences of chronic sleep deprivation, which has been consistently associated with poorer academic performance, metabolic disturbances [5], and an increased risk of anxiety and depressive disorders [6,7].

In patients with persistent and more severe circadian rhythm disturbances, such as Delayed Sleep-Wake Phase Disorder (DSWPD), behavioral interventions alone may prove insufficient [26,28,34]. In such cases, medical treatment may be considered, but it requires a highly cautious approach, particularly in the pediatric population, and must be conducted strictly under specialist supervision. Recommended therapeutic approaches include chronotherapy (the controlled and gradual adjustment of sleep timing), combined with bright light therapy or pharmacological treatment using exogenous melatonin. When utilizing melatonin in children and adolescents, special attention must be given to both the dosage (typically restricted to low doses) and the exact time of administration. Administration several hours before the individual's Dim Light Melatonin Onset (DLMO) acts as a powerful circadian synchronizer (zeitgeber), producing the desired phase advance. However, this intervention requires precise timing based on the patient's individual circadian profile; without proper specialist oversight, improper dosing or timing of both melatonin and chronotherapy can paradoxically exacerbate circadian misalignment [35] (Table I).

Table I. Chronobiological strategies and their impact on circadian rhythm regulation and sleep quality

Strategy	Main mechanism of action	Target chronobiological effect	Recommended time frame	Key references
Light hygiene	Protection of endogenous melatonin secretion through reduced stimulation of intrinsically photosensitive retinal ganglion cells (ipRGCs)	Prevention of circadian phase delay	1 to 2 hours before bedtime	[9,22]
Blue light filters	Physical attenuation of light in the ~460 to 480 nm wavelength range emitted by LED devices	Potential suppression of evening arousal and possible reduction in sleep onset latency	2 to 3 hours before bedtime, particularly during screen exposure	[36,37]
Morning sunlight exposure	Activation of the retinohypothalamic pathway leading to phase advancement of the suprachiasmatic nucleus (SCN)	Circadian phase advance	Within 30 to 60 minutes after waking	[17,23]
Parental control and sleep education	Reduction of psychological and sensory arousal before bedtime	Increased total sleep duration and improved sleep-wake rhythm regulation	Consistent home and school routines	[1,5]
Exogenous melatonin	Biological phase shifting through interaction with MT1 and MT2 melatonin receptors	Correction of persistent Delayed Sleep-Wake Phase Disorder (DSWPD)	Individualized according to clinical needs	[26,35]

Summary and conclusions

The evidence reviewed clearly demonstrates that children and adolescents exhibit a unique neurobiological susceptibility to evening exposure to artificial light. While short-wavelength blue light (460-480 nm) plays a central role due to ipRGC sensitivity, the magnitude of circadian disruption is ultimately determined by a dynamic interaction involving the light's intensity, duration, exact timing of exposure, and the individual's photic history throughout the day. This heightened vulnerability results from the combined influence of anatomical, hormonal, and psychosocial factors.

From an anatomical perspective, the larger pupil diameter and greater lens transparency observed in children allow a greater number of photons to reach the retina. This enhances the stimulation of intrinsically photosensitive retinal ganglion cells (ipRGCs), leading to profound suppression of pineal melatonin synthesis under light exposures (e.g., moderate ambient light or screen luminance) that typically produce only minimal neuroendocrine responses in adults. Evidence from controlled studies characterizes adolescence as a period of peak biological sensitivity to evening photic stimuli.

Adolescence is characterized by peak sensitivity of the circadian system to light stimuli. Combined with the slower accumulation of homeostatic sleep pressure during this developmental period, this promotes a natural delay in Dim Light Melatonin Onset (DLMO) and consequently delays sleep onset (phase delay).

An additional contributing factor is the so-called "perfect storm" phenomenon. Adolescents' increasing behavioral autonomy, widespread use of mobile electronic devices at close viewing distances immediately before bedtime, and the accompanying cognitive and emotional stimulation associated with Fear of Missing Out (FOMO) and social media use act synergistically with their biological susceptibility.

This persistent circadian phase delay contributes to the development of social jetlag and chronic sleep deprivation. As a result, it increases the risk of Delayed Sleep-Wake Phase Disorder (DSWPD), impairs cognitive functioning and academic performance, promotes metabolic dysregulation, including insulin resistance and obesity, and substantially increases vulnerability to mood disorders, anxiety, and depression.

Despite the growing body of literature published over the past two decades, significant research gaps and methodological limitations remain. Most existing evidence is derived from cross-sectional studies or short-term experimental investigations, which limit causal inferences. Longitudinal cohort studies following children from preschool age into adulthood, similar to protocols such as the CLASS study, are needed to accurately assess the cumulative long-term

effects of chronic social jetlag on brain plasticity, metabolic development, and cardiovascular health. Another important limitation of the current literature is the widespread reliance on self-reported sleep diaries and estimated screen time, which are prone to recall bias. Future studies should increasingly incorporate continuous, multisensor monitoring, combining actigraphy with 24-hour personal light dosimetry using wearable sensors positioned near the chest or eyes. Crucially, the available literature is characterized by considerable heterogeneity, leading to discrepancies in reported outcomes across different studies. These inconsistencies often stem from highly variable experimental conditions, including differences in light intensity, spectral composition, distance from the light source, duration of exposure, and the diverse age ranges of the studied cohorts. For instance, while some laboratory studies demonstrate robust melatonin suppression following moderate evening screen use, other real-world observational studies report more mixed or marginal effects. This discrepancy may be attributed to uncontrolled confounding factors, such as individuals' daytime photic history, baseline homeostatic sleep pressure, or interindividual genetic differences in circadian sensitivity.

Furthermore, future studies must more clearly disentangle the biological impact of evening light exposure from the behavioral and psychological effects of electronic device use. Currently, it remains a methodological challenge to separate the direct physiological consequences of light-induced melatonin suppression from the cognitive and emotional arousal driven by digital content. Implementing individual light hygiene measures, such as limiting screen exposure before bedtime, applying chronotherapy, administering melatonin treatment, or using blue-light filters despite heterogeneous evidence regarding their absolute efficacy, may not be sufficient to counteract the societal constraints imposed by fixed school schedules.

One of the most debated, yet potentially most impactful, public health strategies is the implementation of delayed school start times. In line with recommendations from medical and chronobiological societies, delaying the start of the school day better aligns educational schedules with adolescents' biological need for later wake times. Available evidence suggests that this intervention reduces sleep deprivation, decreases rates of tardiness and absenteeism, improves academic performance, and lowers the incidence of depressive episodes and motor vehicle accidents among adolescent drivers.

In addition, incorporating education on the biology of sleep and the effects of light exposure into standard school curricula could improve awareness among students, parents, and teachers. Widespread implementation of evidence-based educational and preventive programs may contribute to healthier sleep behaviors across the pediatric population.

Finally, greater attention should be devoted to standardizing lighting environments in educational and childcare settings. School and residential lighting systems should be designed in accordance with circadian principles by maximizing exposure to bright natural or dynamic light during the morning and daytime to strengthen circadian entrainment through the Phase Response Curve (PRC), while minimizing blue-light exposure during the evening hours. Such environmental modifications may represent an important component of comprehensive strategies aimed at protecting sleep health and promoting healthy circadian development in children and adolescents.

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Ethical considerations

As this study is a literature synthesis and did not involve direct human participants or animals, institutional review board approval was not required.

Conflict of interest

The authors declare no conflict of interest regarding the publication of this manuscript. None of the commercial systems mentioned have provided financial support, incentives, or funding for this review.

Use of AI tools statement

ChatGPT was used solely for the purpose of integrating and styling the content. The entire research process, including the literature review, analysis of scientific articles, and selection of key information regarding evening light exposure and sleep phase shifts in adolescence and childhood, was conducted independently by the authors.

Authors' contribution

Study design – K. Leś, T. Męcik-Kronenberg

Data collection – K. Leś, T. Męcik-Kronenberg

Manuscript preparation – K. Leś, M. Kronenberg, T. Męcik-Kronenberg

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