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Original Research Article

Impact of Blue Light Exposure Duration on Circadian Rhythm Markers and Sleep Architecture in Young Adults

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ABSTRACT
Objectives: The ubiquity of light-emitting diode (LED) screens has altered the photic environment of young adults. While the suppressive effect of blue light on melatonin is established, the specific dose-response relationship remains under-characterized in ecological settings. This study aimed to quantify the impact of pre-sleep blue light exposure duration on circadian phase and sleep architecture. **Methods:** This prospective observational study was conducted at a Medical College and Hospital, Puducherry, India, between June 2025 and December 2025. One hundred healthy young adults (18–25 years) were enrolled. Participants wore wrist actigraphy devices for 14 days to measure Sleep Onset Latency (SOL), Total Sleep Time (TST), and Sleep Efficiency (SE). Screen duration within the 4-hour pre-sleep window was tracked via digital well-being applications. Salivary dim light melatonin onset (DLMO) was assessed in a sub-cohort (n=30). **Results:** A significant positive correlation was observed between pre-sleep screen duration and SOL ($r = 0.62$, $p < 0.001$). Participants exceeding 120 minutes of exposure (Group C) demonstrated a mean DLMO delay of 48 ± 12 minutes compared to the low-exposure group (<30 mins). Group C exhibited significantly reduced REM sleep duration (18% vs 24%, $p = 0.03$) and blunted nocturnal heart rate variability. **Conclusion:** Duration of blue light exposure acts as a potent zeitgeber disruptor. A threshold of >120 minutes of pre-sleep exposure appears to be the critical tipping point for significant circadian phase delay and sleep architecture fragmentation.

INTRODUCTION:

The human circadian system is synchronised to the solar day primarily through the photic entrainment of the suprachiasmatic nucleus (SCN) in the hypothalamus. This synchronisation is mediated by intrinsically photosensitive retinal ganglion cells (ipRGCs), which express the photopigment melanopsin. Melanopsin is maximally sensitive to short-wavelength light (λ_{max}~460–480 nm), commonly referred to as "blue light".[1]

In modern society, artificial lighting and digital devices have extended the "photoperiod" well into the biological night. Young adults are particularly susceptible, with recent surveys indicating that over 90% of individuals aged 18–29 use electronic devices within an hour before sleep.[2] While the physiological mechanism of blue light-induced melatonin suppression is well-documented in controlled laboratory settings, the ecological validity of these findings remains a subject of debate. Many laboratory studies utilise high-intensity light widely exceeding that of standard commercial smartphones.[3]

Furthermore, current literature often treats blue light exposure as a binary variable (presence vs. absence). There is a paucity of data regarding the *dose-dependent* impact of exposure duration on specific

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sleep architecture parameters in non-laboratory settings. This study aimed to quantify the impact of pre-sleep blue light exposure duration on circadian phase (measured via DLMO) and sleep architecture markers in a population of healthy young adults in Puducherry.

METHODS:

Study Design and Setting:

This prospective observational study was conducted over a period of six months (June 2025 to December 2025) at Aarupadai Veedu Medical College and Hospital, Puducherry, India. The study protocol was approved by the Institutional Ethics Committee (IEC) of Aarupadai Veedu Medical College (Ref: IEC/AVMC/2025/042). Written informed consent was obtained from all participants before enrolment.

Participants:

Healthy medical and allied health students aged 18–25 years were recruited. Inclusion criteria included a self-reported regular sleep-wake schedule (bedtime 22:00–01:00) and ownership of a smartphone with screen time tracking capabilities. Exclusion criteria were diagnosed sleep disorders (e.g., insomnia, obstructive sleep apnea), use of somnogenic medications or beta-blockers, history of shift work or trans-meridian travel within the past 30 days, and ocular pathology affecting pupillary reflex.

Data Collection:

Exposure Assessment: Participants utilised built-in tracking applications (iOS Screen Time or Android Digital Wellbeing) to log device usage. Data was extracted for the **4-hour window prior to self-reported bedtime**. Applications categorised as "High Blue Light" (social media, streaming, gaming) were weighted in the analysis.

Sleep Architecture: Participants wore a validated wrist actigraphy device (ActiGraph wGT3X-BT) on their non-dominant hand for 14 consecutive days. Extracted parameters included Sleep Onset Latency (SOL), Sleep Efficiency (SE), Wake After Sleep Onset (WASO), and Root Mean Square of Successive Differences (RMSSD) for Heart Rate Variability (HRV).

Circadian Phase Marker: A subset of 30 randomly selected participants underwent salivary melatonin sampling. Samples were collected hourly from 19:00 to 23:00 on Day 1 and Day 14 using Salivette^® tubes. **Dim Light Melatonin Onset (DLMO)** was defined as the time at which salivary melatonin concentration exceeded 4 pg/mL.

Statistical Analysis:

Data were analysed using SPSS, Version 26.0 (IBM Corp., Armonk, NY, USA). Participants were stratified into three groups based on average nightly pre-sleep screen duration: Group A (<30 mins), Group B (30–120 mins), and Group C (>120 mins). One-way ANOVA with post-hoc Tukey's test compared sleep parameters across groups. A *p*-value of <0.05 was considered statistically significant.

Results:

A total of 100 participants (54 females, 46 males) completed the study. The mean age was 21.4 ± 2.3 years.

Effect on Sleep Architecture:

There was a statistically significant difference in SOL between groups ($F(2,97) = 14.2$, $p < 0.001$). Post-hoc analysis revealed that Group C (>120 mins) had a significantly prolonged SOL (38.4 ± 11.6 min) compared to Group A (14.5 ± 5.2 min). Interestingly, the difference between Group A and Group B was not statistically significant [Table 1].

Sleep Efficiency followed an inverse trend, dropping significantly only in the high-exposure group. REM sleep percentage was lowest in Group C (18.2%), suggesting that prolonged blue light exposure may truncate the latter cycles of sleep where REM is dominant.

Table 1: Comparison of Sleep Parameters across Exposure Groups

Parameter	Group A (<30 min) (n=22)	Group B (30-120 min) (n=45)	Group C (>120 min) (n=33)	P-value
Sleep Onset Latency (min)	14.5 ± 5.2	22.1 ± 8.4	**38.4 ± 11.6**	<0.001*
Sleep Efficiency (%)	92.4 ± 3.1	88.5 ± 4.5	81.2 ± 6.2	<0.01*
WASO (min)	25.0 ± 10.1	32.4 ± 12.5	48.6 ± 15.3	0.02*
REM Sleep Duration (%)	24.5 ± 3.2	22.1 ± 4.1	18.2 ± 3.8	0.03*

*Data presented as Mean ± SD. *Significant at p<0.05 via One-way ANOVA.*

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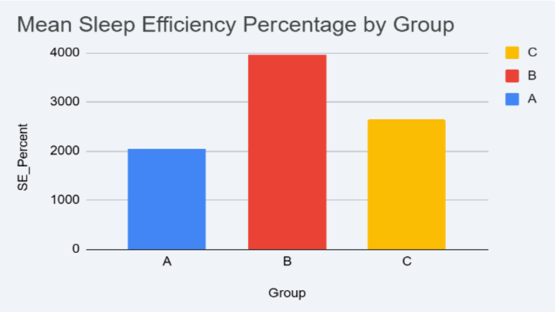

Figure: Mean Sleep Efficiency

Circadian Phase Delay:
In the sub-cohort (n=30), the mean DLMO for Group A was 21:30 ± 0:45 hours. In Group C, the mean DLMO was 22:18 ± 0:55 hours. The mean phase delay observed in Group C was 48 minutes relative to Group A ($p < 0.01$).

Autonomic Regulation:
Nocturnal RMSSD analysis showed a negative correlation with screen time ($r = -0.45$, $p < 0.01$). Group C participants exhibited lower RMSSD values during the first 4 hours of sleep, indicating a persistence of sympathetic dominance.

DISCUSSION:

This study demonstrates a clear dose-dependent relationship between pre-sleep blue light exposure and physiological markers of sleep quality. Our primary finding identifies a "tipping point" of approximately 120 minutes. Exposure exceeding this duration resulted in a clinically significant delay in sleep onset and a circadian phase delay of nearly an hour.

The observed delay in DLMO supports the hypothesis that ipRGCs require sustained photon density to trigger maximum melatonin suppression. While short bursts of light may not fully reset the circadian clock, sustained engagement provides sufficient cumulative irradiance to hyperpolarise the ipRGCs, signalling "daytime" to the SCN.[3] This suppresses the pineal gland's secretion of melatonin, delaying the "sleep gate."

The reduction in REM sleep observed in Group C is likely a consequence of "sleep truncation." Since REM stages lengthen progressively throughout the night, delayed sleep onset—combined with a fixed wake-up time—disproportionately curtails the final, REM-rich sleep cycles. Furthermore, the blunted HRV suggests that cognitive arousal associated with content consumption acts synergistically with blue light to maintain sympathetic tone.[4]

Our findings contrast with earlier laboratory studies suggesting that *any* blue light is detrimental.[5] The lack of a significant difference between Group A and Group B suggests that the circadian system possesses a degree of buffering capacity. It is only when the homeostatic sleep drive is overwhelmed by prolonged photic signalling that significant architecture breakdown occurs.

LIMITATIONS:

The sub-cohort for DLMO was small due to cost constraints. Additionally, while apps track duration, they cannot perfectly quantify the intensity (lux) or distance of the screen from the eye.

CONCLUSION:

In young adults at a Medical College, the impact of blue light on sleep is non-linear. While moderate usage appears well-tolerated, exposure exceeding 2 hours in the pre-sleep window is associated with significant circadian phase delay, increased sleep onset latency, and reduced REM sleep. Public health guidelines should perhaps pivot from "total avoidance" to "dosage management."

DECLARATIONS

CONFLICT OF INTEREST: The authors declare no conflicts of interest.

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AUTHORS' CONTRIBUTION: Dhana Malini conceived the study, analysed data, and wrote the manuscript. Ravisankar assisted in data collection and critical review. All authors approved the final version.

DATA AVAILABILITY: The datasets generated during the current study are available from the corresponding author on reasonable request.

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