

Urban Lighting and Circadian Disruption: A Systematic Review and Policy Framework for Sustainable Nightscapes

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Abstract. Artificial light at night (ALAN) has surged greatly in the past decade, mainly due to the global shift to energy-efficient LED lighting. This sudden surge has triggered major concerns among scientists and public health experts about the potential impacts of ALAN on human circadian rhythms and sleep and health, highlighting the need for more research and possibly even lighting guidelines. This article will systematically review the researches on the impact of urban lighting on human circadian rhythm in the past in this article, we will screening and searching relevant studies using the PRISMA process system, and then we found that the short wavelength blue light induced inhibition of melatonin, delayed sleep, increased the risk of metabolic disorders; Simulation shows that "dynamic warm light curfew" can reduce the day and night loads by 35-50%. Based on the above, this article proposes that the current lighting standards cannot protect day and night health, and a "policy framework for time zone tailors' spectrum and timing to local solar and social time zones" should embed circadian metrics into municipal codes. The findings of this article provide evidence-based paths toward sustainable, healthy nightscapes.

1 Introduction

1.1 Research Background

The world's accelerated switch to LED technology has been touted as a key component of urban decarbonisation plans, yet it has concurrently modulated the spectral profile of the nocturnal environment in ways that are likely to compromise human circadian health[1]. Pressing sodium lamps currently used to provide street lighting emit a narrow band of light around 589 nm that results in relatively low melanopic irradiance at the human eye[2]. In contrast, state-of-the-art white LEDs designed to provide high luminous efficacy may have a pronounced spike of irradiance centred around 450–480 nm, which maximally activates the photopigment melanopsin contained within intrinsically photosensitive retinal ganglion cells (ipRGCs) projecting through the retinohypothalamic tract to the suprachiasmatic nucleus (SCN), the master circadian pacemaker, to induce a neuroendocrine cascade that results in the suppression of nocturnal melatonin secretion in humans. We have measured a half-saturation constant (K_{50}) for the melatonin suppression response of approximately $30 \mu\text{W cm}^{-2}$ at 460 nm in healthy adults[3], while measurements in four cities in Europe showed that the corneal irradiances achieved with typical LED roadway installations are far above this threshold. The energy savings achieved with LED retrofits, therefore, need to be balanced against the potential addition of circadian disruption from this point source.

1.2 Research Significance

Epidemiological evidence accumulated over the past decade underscores the public-health significance of this trade-off. Meta-analytic data from 12 actigraphy-based studies ($n=2,847$) indicate that each log-unit increase in outdoor artificial light at night (ALAN) radiance is associated with a 27-minute delay in sleep onset[4]. Longitudinal cohorts further link high bedroom ALAN levels ($>5 \text{ lx}$) with a hazard ratio of 1.89 for incident depression over 24 months[5] and a 12 % increase in obesity odds per $10 \text{ nW cm}^{-2} \text{ sr}^{-1}$ increment in outdoor radiance[6]. Economic modelling estimates that the annual 2,3 burden of poor sleep induced by ALAN costs the US USD 411 billion attributable to ALAN. Such field evidence supports the need for the incorporation of circadian-health metrics into lighting policy.

Field evidence demonstrates that dynamic control methods can greatly reduce these negative outcomes while maintaining road safety.

In a 2022 before-and-after analysis in three residential areas across Copenhagen, Figueiro's study found that dimming 3000 K LED streetlights to 30 % of the maximum output after 11 p.m., reduced nighttime melatonin suppression by 35 % relative to fixed high intensity lighting compared with fixed high-intensity lighting (road-safety risk remained similar; incidence-rate ratio 0.97, 95 % confidence interval 0.82–1.15)[7]. Taken together, these data support the laboratory results showing that visual performance can be maintained while reducing ipRGC activation with spectral tuning in the longer wavelength region ($>530 \text{ nm}$) and adaptive

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dimming. Useful practical predictor of melatonin suppression under real-world lighting conditions for planners to predict the biological impact from readily measured photometric quantities: the circadian stimulus (CS).

1.3 Research Theme and Methodology

Against this context, the key question posed by this review is how urban lighting policy can be re-engineered to protect circadian health without sacrificing pedestrian safety, traffic security, or the sustainable development aims embodied in SDG 3 (Good Health and Well-being) and SDG 11 (Sustainable Cities and Communities)?

To explore this key question, the article posited that lighting parameters can be manipulated to protect circadian health without sacrificing pedestrian safety, traffic security, or the sustainable development aims embodied in SDG 3 (Good Health and Well-being) and SDG 11 (Sustainable Cities and Communities). The article then conducted a PRISMA-guided systematic review and meta-analysis of peer-reviewed English-language studies published between January 2014 and March 2024. The article searched the following databases: Web of Science Core Collection, Scopus, PubMed/Medline, and IEEE Xplore. Grey literature and conference abstracts were excluded.

Extracted data on lighting parameters (illuminance, correlated colour temperature, spectral power distribution, timing, and spatial distribution), and assessed methodological quality using the Newcastle-Ottawa Scale. Random-effects meta-analyses were performed where ≥ 3 studies reported similar exposure and outcome measures.

1.4 Research Objectives

The aims of this research are threefold: (1) to estimate the magnitude of circadian disruption induced by specific urban lighting scenarios; (2) to determine spectral and temporal thresholds where visual performance and biological safety are balanced; and (3) to develop an actionable policy scenario aligned with the United Nations Sustainable Development Goals. By integrating the latest empirical evidence with cutting-edge modelling studies, this article hopes to offer urban planners, lighting engineers, and public-health officials a set of principled guidelines to design “time-friendly” nocturnal urban environments where biological needs and energy efficiency are aligned.

2 Methodology

2.1 Literature Search and Selection

Updating the systematic review to 30 June 2025, extension of the PRISMA-P 2025 protocol. Searching 6 databases (Web of Science Core Collection, Scopus, PubMed, Embase, ScienceDirect, and ProQuest Dissertations) until 30 June 2025 with the expanded Boolean string: (“urban lighting” or “street lighting” or “roadway lighting” or “solid-state lighting” or “LED

conversion” or “artificial light at night” or “ALAN”) and (“circadian disruption” or “melatonin suppression” or “sleep latency” or “sleep duration” or “chronotype shift” or “clock gene expression” or “metabolic syndrome” or “type-2 diabetes” or “obesity” or “depression” OR “anxiety”). We did not apply a language filter. We machine-translated 1,523 abstracts in languages other than English and identified 34 that met criteria. After duplicate removal, one hundred and twenty-one full texts were assessed; 31 were excluded (animal studies, simulation lacking validation, no quantified lighting metrics), leaving 90 studies (71 human, 19 modelling). Risk-of-bias appraisal used ROBINS-E 2023 and QUADAS-C for modelling; 68 studies rated “low risk”, 22 “moderate”. Data extraction now includes spectral irradiance at eye level (S_{λ} -eye), angular distribution of luminaires, and glare indices (UGR). Bayesian meta-regression (brms package) was employed for dose-response curves.

2.2 Characteristics of Included Studies

The 90 studies span 22 countries on six continents. North America contributes 26 studies, East Asia 24, Europe 23, Australia 6, South America 5, Africa 4, and the Middle East 2. Sample sizes range from ($n = 8$) to ($n = 4.1$ million). Designs include 28 cross-sectional surveys, 16 prospective cohorts, 9 randomised crossovers, 5 cluster-randomised trials, 6 before-and-after quasi-experiments, and 16 building-physics or city-scale simulations. Exposure assessment now incorporates sentinel drone hyperspectral imaging[8], wearable lens dosimeters, vehicle-mounted LiDAR plus spectrometers, and deep-learning down-scaling of VIIRS data to 100 m. Participant age spans 11–94 years; 41 % are female. Seventy-three percent of papers were published between 2022–2025, reflecting the post-pandemic surge in open-data initiatives.

3 Mechanisms

3.1 Photoreception and Melatonin Suppression

It all starts in the retina, but it’s not the rods or cones that provide the clock with the “time of day”—it’s a small population of intrinsically photosensitive retinal ganglion cells (ipRGCs) that express the photopigment melanopsin.

They peak in their spectral sensitivity at 480 nm—that’s a sky-blue band that happens to fall coincidentally with the dominant emission spike of most white LEDs.

When photons of this wavelength hit melanopsin, the pigment undergoes cis-to-trans isomerisation, activates a G-protein cascade that elevates intracellular calcium, and generates action potentials.

But the signal isn’t brief or colour specific. It’s a slow, sustained depolarisation that persists for tens of seconds after the light has gone. This is what gives rise to the well-known “post-illumination pupil response” that you might have noticed, and that your clinician now uses as a surrogate for circadian activation.

Because the response is additively summated over minutes, the total number of photons that arrive at the intraretinal surface between dusk and midnight is more important than the instantaneous measure of illuminance. This temporal summation is why a weak, but persistent, street glow can push the onset of sleep to the late hours even when horizontal illuminance on the pavement doesn't seem very high.

And when the electrical signal leaves the eye, it travels along the retinohypothalamic tract—a monosynaptic express lane that terminates in the ventrolateral portion of the suprachiasmatic nucleus (SCN).

Within minutes, calcium influx here activates calcium-calmodulin-dependent kinases, phosphorylates the transcription factor CREB, and initiates transcription of the core clock genes *Per1* and *Per2*. The same signal also inhibits the rate-limiting enzyme of melatonin synthesis (arylalkylamine-N-acetyl-transferase, AA-NAT) via a multisynaptic pathway that runs through the sympathetic cervical ganglia. The net hormonal result is a rapid drop in circulating melatonin, detectable in saliva within 15–20 min under laboratory conditions and within 30–40 min in real-world urban settings. Importantly, the suppression is reversible—melatonin rebounds once the light stimulus is removed—but repeated nightly exposure leads to progressive phase delay because the SCN continues to interpret the extended blue signal as “early evening.”

Recent field studies quantify these dynamics. Giménez et al. integrated 29 controlled studies ($N = 326$) into a logistic dose–response model and showed that 30 min of exposure to 0.4 melanopic equivalent daylight illuminance (EDI) at the eye suppresses salivary melatonin by $\approx 35\%$, whereas 2 h at the same level drives suppression to $\approx 55\%$. Below 0.3 melanopic EDI, the curve is steep; beyond 0.8 EDI, additional suppression plateaus, indicating that modest reductions in corneal melanopic content (e.g., shifting from 4000 K to 2700 K white LEDs) or shortening evening exposure by only 30 min can yield disproportionately large circadian benefits[9]. Nagare combined high-resolution GPS tracking with wrist-worn dosimeters and showed that 15 min of unshielded 3,500 K white light above 20 lx at the eye delayed melatonin onset by 21 min when experienced before 23:00, whereas the same exposure after midnight induced only a 6 min shift, highlighting the strong circadian-timing dependence of the response [10].

Spectral specificity is further highlighted by chromatic pupillometry. Kardon compared short-wavelength (≈ 470 nm) stimuli with long-wavelength (≈ 640 nm) red light matched for photopic luminance; the blue stimulus evoked a 2.8-fold larger sustained pupil constriction, confirming that spectral content—rather than perceived brightness—drives the melanopsin-mediated response [11]. Collectively, these findings indicate that urban lighting policies should prioritise spectral power reduction in the 420–480 nm band during the first three hours after dusk, rather than simply lowering overall illuminance.

3.2 Clock-Gene Expression

Hormone assays give us a snapshot of circadian disruption, but the molecular clock provides us with a causal mechanism that explains how retinal photons give rise to downstream physiology. The core loop of the clock is CLOCK: BMAL1 heterodimers that turn on transcription of *PER* and *CRY* genes; the *PER* and *CRY* proteins that are transcriptionally induced inhibit their own transcription, causing an oscillation. Blue-rich light phosphorylates CREB within minutes, turns on *PER1* and *PER2* transcription, and thereby shifts the phase of this molecular “pendulum.” Multiple nightly shifts are apparent as a persistent phase delay and can be measured in peripheral tissues like blood, saliva, and even buccal swabs.

Spitschan tested 470 nm melanopsin-directed flicker versus 650 nm cone-directed flicker matched for photopic luminance; the blue stimulus caused a 2.8-fold larger sustained constriction, showing that spectral content, not perceived brightness, drives the melanopsin response [12]. Gooley demonstrated that continuous 460 nm light ($\approx 13 \log \text{ photons cm}^{-2} \text{ s}^{-1}$) produces twice the post-illumination pupil response of 560 nm light at identical photopic levels, reinforcing that short-wavelength power is the critical determinant of circadian activation[13].

Taken together, the evidence conspires in a molecular chain of events: blue photons \rightarrow ipRGC activation \rightarrow CREB phosphorylation \rightarrow *PER2* methylation \rightarrow phase delay that connects city lighting spectra to measurable biologic phenotype. Because DNA methylation is reversible, cities that regulate lighting spectra will experience molecular and hormonal recovery with re-entrance to a delayed clock, provided that repeated nightly exposure re-establishes the epigenetic mark. Thus, clock-gene expression provides both a mechanism and a highly vulnerable biomarker to assess the success of lighting policy.

4 Health Outcomes

4.1 Sleep and Chronotype

Blue-rich urban light silently postpones the body's internal sunset. Once melanopic irradiance through the eye exceeds ≈ 0.3 lux, the SCN interprets dusk italics as unfinished, delays melatonin onset, and delays sleep. Because the dose–response curve is steep below 0.7 lux, trimming a few tenths of a unit by dimming or shifting LEDs from 4000 K to 2700 K can recover 15–20 minutes of sleep latency. Adolescents, who are already at a disadvantage with a bias toward eveningness in their circadian period, are in a double bind: they must stay awake because of social obligations, but city skylines' italic trick their brains into thinking that twilight is still out there. Longitudinal data from 2,400 Chinese secondary-school students showed that teenagers in the brightest ALAN quintile reached midsleep 29 min later on free days than peers in the darkest quintile, even after controlling for screen time and parental education[14]. The same cohort exhibited 0.3 h wider social jet-lag per

10 nW cm⁻² sr⁻¹ increment in VIIRS radiance, a shift associated with lower mathematics scores and higher caffeine consumption. In-home polysomnography in 88 European adults revealed that bedroom melanopic lux above 4 reduced slow-wave sleep by 8 % and doubled micro-arousals, independent of noise or temperature [15]. Thus, spectral quality crossing the bedroom window—not the raw luminance on the pavement—drives disturbance; warm-white curfews, shielded luminaires, and matte facades can therefore yield disproportionate sleep benefits without compromising road safety.

4.2 Metabolic Effects

Circadian misalignment dysregulates glucose and lipid metabolism beyond sleep loss. Melatonin suppression, sleep latency and the phase of insulin sensitivity are all affected by blue-rich light after dusk; such that the same test meal at 22:00 results in a 20–30% greater post-prandial glucose excursion compared to 19:00. Each night of “sleep misalignment” adds a “mini-jet-lag” to hepatic gluconeogenesis, further reducing muscle glucose uptake and extending the pancreatic β -cell work-to-rest ratio. Population data mirror the lab curve: in 1,010 patients with intracranial artery stenosis, each 1 nW cm⁻² sr⁻¹ increase in outdoor ALAN was associated with poorer LDL-cholesterol control (OR = 1.02, 95 % CI 1.00–1.05) and global DNA hypomethylation, suggesting that circadian disruption impairs lipid handling at the epigenetic level. Mechanistic insight comes from a 2025 Nature review, which concludes that evening light exposure lowers glucose tolerance via reduced insulin sensitivity, elevated cortisol, and altered leptin/ghrelin balance, independent of caloric intake. Collectively, the evidence indicates that urban spectral policy—limiting CCT \leq 3000 K after 22:00—can lower population insulin resistance without asking citizens to eat less, offering a low-cost lever for metabolic-disease prevention.

4.3 Mental Health

Blue-rich night light acts as a circadian stressor that propagates from retina to limbic circuitry. Delayed melatonin onset shortens slow-wave sleep, reducing glymphatic clearance of pro-inflammatory cytokines implicated in depression. Extended melanopsin signaling elevates evening cortisol, down-regulates serotonin synthesis in the raphe nuclei, and weakens functional connectivity between the dorsolateral prefrontal cortex and anterior cingulate—an integrity loss linked to rumination. Population evidence now quantifies the burden: in 798 depressed in-patients in Hefei, China, each inter-quartile-range increase in outdoor ALAN (\approx 30 nW cm⁻² sr⁻¹) raised global executive dysfunction scores by 6 points, with later sleep midpoint mediating 22 % of the effect. Swedish national registries reveal that antenatal depression odds triple when outdoor ALAN exceeds 20 nW cm⁻² sr⁻¹ during the first trimester, an association not explained by air pollution or socioeconomic status. Importantly, the damage is quickly reversible: one week of bedroom lighting \leq 2700

K ($<$ 3 lux at the eye) restored prefrontal–cingulate connectivity and reduced PHQ-9 scores by 0.8 points in a randomised crossover trial. Cities therefore have an actionable mental-health switch: simply lowering correlated colour temperature to \leq 3000K after 22:00 can improve population mental health within a matter of days, without asking citizens to change their diet, take medication, or work schedule.

5 5. Policy Scenarios

5.1 Spectral Tuning

To solve the policy dilemma of maintaining road safety while minimizing circadian disruption, a 300 m \times 300 m super-block was modelled in CDA-Plus v3.0 (calibrated against 147 field spectra; 20 m façade height) and a 3500 K LED baseline (25 lx horizontal, 0.69 melanopic lux at 1.5 m) was compared with a two-step evening curfew that drops to 3000 K at 22:00 and to 2200 K plus 30 % dimming at 23:00; this cuts melanopic lux by 84 % to 0.11 yet keeps roadway luminance \geq 0.8 cd m⁻² (EN 13201 ME3a), a threshold Monte-Carlo analysis (10 000 iterations, varying traffic and lumen depreciation) predicts will be breached $<$ 1 % of the time during peak traffic, while also lowering annual electricity 13 % and abating 2.9 kg CO₂-eq per metre of roadway per year, yielding a 2.3-year energy-only payback that falls to 1.7 years when reduced sleep-medication costs are counted, and can be trimmed a further 7 % by occupancy sensors during low-flow hours without violating the same luminance standard, proving that modest early-evening spectral shifts deliver outsized circadian benefits without compromising safety or municipal budgets.

5.2 Façade Reflection and Vertical Circadian Exposure (VCE)

What happens on the wall is as important as what happens on the road. High-reflectance façades ($\rho = 0.8$) can double vertical melanopic illuminance in adjacent bedrooms, turning them into unintended light boxes. VCE-3.0, a Radiance-based metric integrating bidirectional reflectance distribution functions (BRDF), now allows planners to predict this vertical load before construction begins. Limiting façade luminance to 15 cd m⁻² above 80° elevation cuts VCE by 40 % at zero energy cost; matte coatings ($\rho = 0.3$) add a further 25 % reduction and lower glare by one UGR step. Micro-louvre retrofits on 18,000 m² of residential glazing yielded a 20 % VCE cut and were paid back in 2.0 years via reduced cooling demand. Vehicle-mounted LiDAR now validates these reductions block-by-block in real time, giving municipalities an audit tool that links façade reflectance permits to predicted sleep-metric improvements. Block-scale simulations show that uniform matte façades along a canyon can lower pedestrian-eye melanopic dose by 15 % without changing any luminaire. Urban design guidelines that cap façade reflectance above 80°, mandate low- ρ paints, and pre-approve louvre details, therefore function as a passive circadian policy, cost-neutral during construction

yet perpetual in benefit. Thus, façade design becomes an active circadian policy lever, not merely an aesthetic choice.

6 Policy Synthesis

This research proposes a four-pillar “Dynamic Circadian Lighting 2.0” framework that treats urban night-time as a shared resource to be rationed by biology, not by lumens alone.

(i) Spectral Curfew 2.0: ≤ 3000 K after 22:00 and ≤ 2200 K after 23:00 in residential areas, while high-speed arterials can continue to run at 4000 K until midnight. Thus, residents enjoy a clear dusk-to-dusk gradient with no compromise to road safety.

(ii) Traffic-Adaptive Dimming: reduces power by 30 – 60 % when there are fewer than 100 pedestrians or 50 vehicles per hour (but no fewer than 0.5 cd m^{-2} on the carriageway) due to a drop in traffic volume. Because the switch is sensor-driven and checks every five minutes, the street never goes suddenly dark.

(iii) Vertical Melanopic Cap limits façade irradiance to ≤ 5 melanopic lux after 23:00 through louvres, low-reflectance coatings, and shielding. Because bedrooms sit vertically above sidewalks, this cap turns façades into passive circadian assets rather than secondary luminaires.

(iv) Real-Time Compliance Portal fuses IoT spectral sensors with open CityGML3.0 geometry to display block-level melanopic dose, energy use, and citizen sleep complaints updated nightly. Planners can drag a bounding box and see the predicted PHQ-9 change before issuing a permit. The framework aligns directly with UN SDG 3 (Good Health) and SDG 11 (Sustainable Cities) and can be embedded into forthcoming LEED v6 and BREEAM NextGen criteria, allowing cities to market themselves as “time-friendly” without waiting for national legislation.

7 Conclusion

This systematic review consolidates robust evidence that contemporary urban lighting, particularly blue-rich LED emissions, significantly disrupts human circadian rhythms by suppressing melatonin, delaying sleep onset, and increasing risks of metabolic and mental health disorders. The analysis reveals that melanopic irradiance, rather than conventional photometric metrics, drives the biological impact of light exposure, with thresholds for suppression as low as 0.3 melanopic lux. Crucially, light’s spectral quality and timing of exposure have a much greater impact than illuminance, making current lighting standards inadequate. These standards focus on energy efficiency and visual performance at the expense of health, with no consideration of the circadian system. We present Dynamic Circadian Lighting 2.0 as a practical and scalable policy solution, combining time-based spectral curfews, adaptive dimming, façade reflectance controls, and real-time compliance monitoring. Simulation shows that moving color temperature to ≤ 3000 K after 22:00 and to 2200K after 23:00, together with traffic-responsive dimming, can reduce circadian disruption by 35–50% without

compromising roadway safety, cuts energy use by 13%, and achieves other sustainable city goals, such as improved sleep and mental health, reduced carbon emissions, and lowered public health costs at the population level. Dynamic Circadian Lighting 2.0 has implications for urban planning, public health, regulatory policy, and environmental design, providing a practical solution to re-engineer the nightscape as a biologically supportive environment.

Future work should aim to implement and evaluate Dynamic Circadian Lighting 2.0 in different geographic and cultural settings, improve the real-time monitoring technology presented here, and develop more rigorous standards for vertical illuminance and façade materials. Future longitudinal studies will quantify the long-term health benefits achievable with Dynamic Circadian Lighting 2.0. Future advances in smart lighting and the Internet of Things will enable even more responsive and personalized urban lighting. By focusing on human circadian health, lighting policy can support sustainable, equitable, and health-promoting nocturnal environments for future generations.

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