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Review

Evaluating circadian light hygiene: Methodology and health implications

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Abstract: Background — A growing body of research demonstrates that a substantial daily range of light exposure, characterized by ample daylight followed by darkness during sleep, is essential for human well-being. This encompasses crucial aspects like sleep quality, mood regulation, and cardiovascular and metabolic health.

Objective — This study characterizes Circadian Light Hygiene (CLH) as an essential factor in maintaining health, well-being, and longevity in modern society. CLH involves adjusting the 24-hour light exposure dynamic range to support the natural sleep-wake cycle and circadian rhythms. Three major challenges to CLH negatively impacting human health are: 1) light pollution (light at night, or LAN), characterized by excessive evening and nighttime artificial light; 2) insufficient natural daylight; and 3) irregular light exposure patterns. These interacting challenges necessitate a systematic approach to measurement and analysis.

Material and Methods — A systematic review of peer-reviewed literature published through October 30, 2024, examined the methodologies and health effects of circadian and seasonal aspects of light exposure.

Conclusion — This review elucidates fundamental principles of circadian light hygiene, synthesizing existing literature and our research to assess the benefits of adequate daylight, the risks of light at night, and adverse outcomes stemming from diminished light exposure range, mistimed light exposure, and irregular patterns. Novel indices for quantifying and optimizing circadian light hygiene are introduced.

Keywords: light, circadian rhythms, light hygiene, light at night, human-centric lighting, cognition, health, well-being, mood, metabolism.

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*We dedicate this work to the memory of our esteemed
colleague, Konstantin Danilenko*

Introduction

Light is crucial for aligning the circadian clock with environmental conditions and facilitating adaptation to natural seasonal changes [1, 2]. The 24-hour light-dark cycle is fundamental for synchronizing biological clocks with the environment, maintaining properly aligned circadian phases and sufficient circadian amplitude in physiological processes [3, 4]. Circadian amplitude is considered a universal marker of health status [5-7], a finding confirmed by recent large-scale longitudinal studies [8-10]. Throughout human evolution, the natural light-dark cycle has been the primary driver of biological clock and circadian rhythm evolution. However, in present-day society, artificial light

has profoundly altered human activity patterns and significantly disrupted the natural light environment. This has led to prolonged periods of activity misaligned with the natural day-night cycle. The rapid increase in nighttime illumination, coupled with decreased natural daylight exposure, particularly among urban office workers, has markedly reduced the day-night light exposure dynamic range, leading to numerous negative health consequences [1, 2, 11-16]. These negative health outcomes are more pronounced at high latitudes, where seasonal variations in photoperiodism are pronounced [7, 17], and in countries that observe daylight saving time [18, 19]. Research demonstrates that light intensity, regularity, and spectral composition are all critical for health. Light exposure intricately regulates sleep-wake cycles, activity levels, dietary habits, body temperature, energy metabolism, and hormone balance.

Pre-industrial societies at high latitudes commonly exhibited biphasic sleep patterns [20], unlike those in equatorial regions [21]. The presence of terms for “first sleep” and “second sleep” in many languages [20] suggests this was a widespread cultural practice, dividing nighttime sleep into two phases with an intervening wake period. Recent physiological studies suggest that modern humans retain the capacity for biphasic sleep under short photoperiods [22]. However, widespread artificial lighting in homes and workplaces has shifted human sleep patterns to a predominantly monophasic mode. Excessive Artificial Light at Night (ALAN), or light pollution, prevalent in modern industrial societies, has further altered sleep habits. Moreover, it has been linked to increased risks of malignant neoplasms [13, 23-27],

neuroinflammation, and earlier onset of neurodegenerative diseases [28, 29]. While total sleep duration in urban residents is not significantly different from that in pre-industrial societies [21], ALAN may contribute to shorter sleep duration in some individuals [30], suggesting it is a major contributor to the rise of “diseases of civilization” in modern society.

This comprehensive review aims to establish fundamental principles of circadian light hygiene, integrating existing literature and our research to elucidate the benefits of adequate daylight and the detrimental effects of nighttime light exposure, reduced light exposure dynamic range, delayed light exposure phases, and irregular light exposure patterns. Furthermore, we present novel indices for quantifying and improving CLH management.

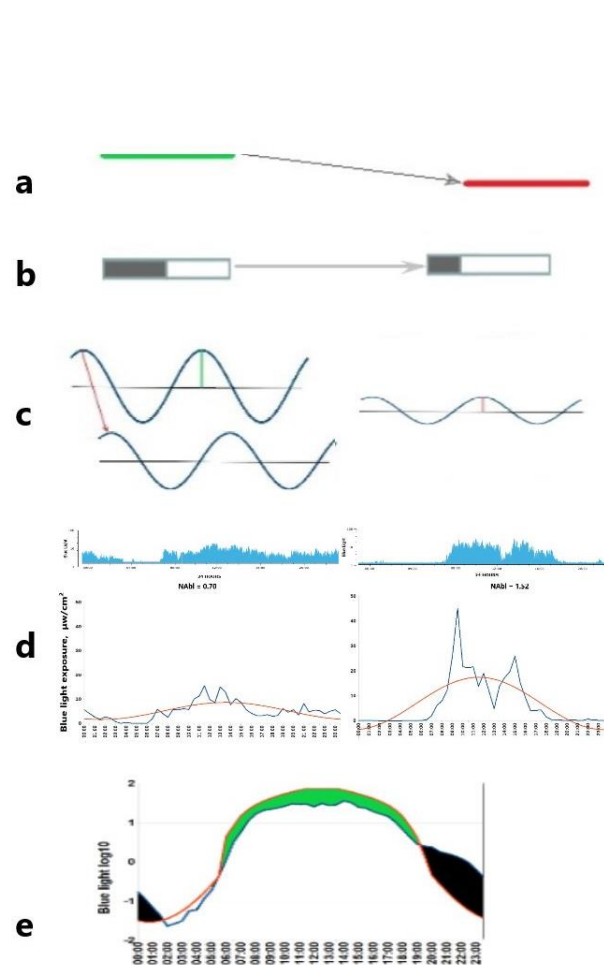
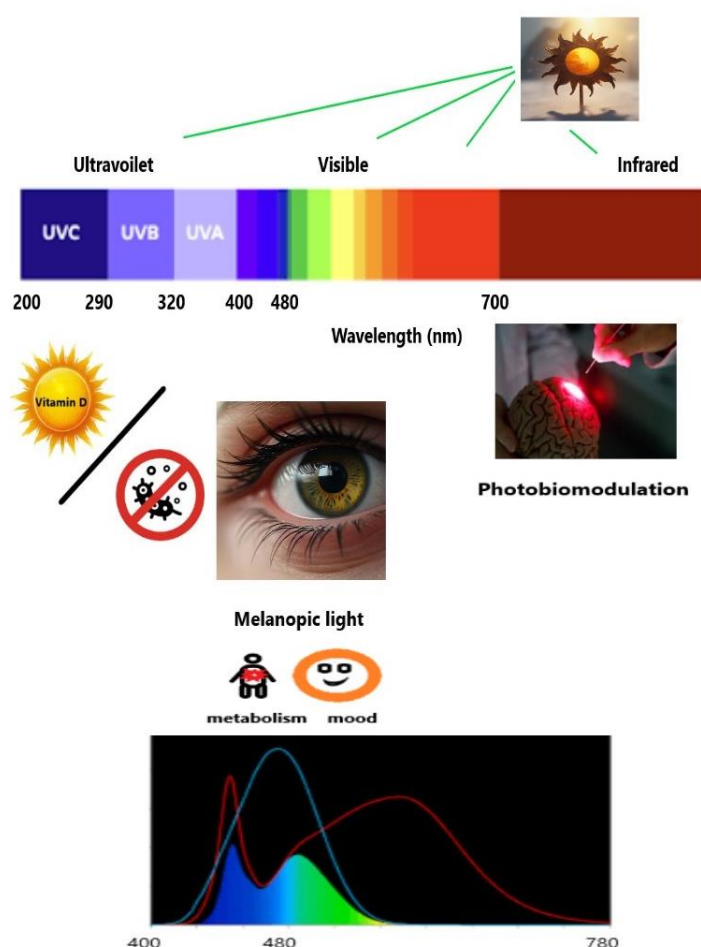


Figure 1. Quantitative Assessment of Circadian Light Hygiene (CLH).

Left: Different ranges of light exhibit distinct visual and non-visual physiological effects, which can vary based on intensity, timing, and gating (retina vs. skin). Generally, “cooler”, shorter-wavelength light is advantageous for humans in the morning and at midday, whereas it should be avoided later in the day. Conversely, “warmer”, longer-wavelength light can be beneficial in the evening. For example, ultraviolet light stimulates vitamin D production in the skin and has an antimicrobial effect. Blue melanopic light is best quantified using melanopic-equivalent daylight illuminance (melanopic EDI), an international standard metric reflecting the effect of light on intrinsically photosensitive retinal ganglion cells, which peaks around 480 nm and is closely associated with melatonin production and circadian clock entrainment. Infrared light may promote tissue healing and reduce inflammation by enhancing cellular metabolism and circulation.

Right: Methodologies for measuring and evaluating CLH. a) Average values (24-hour mean, daytime, nighttime). b) Variations in the duration of light exposure (photoperiodic/seasonal, lifestyle, shift work, etc.). c) Phase delay (left) and amplitude reduction (right) as major indicators of suboptimal CLH. d) Amplitude-to-mean ratio as a normalized index of circadian light hygiene (left: low; right: optimal). e) Cumulative indices within time epochs based on log-transformed data for blue light exposure (daylight deficiency index indicated in green; nocturnal excess index indicated in black) [109, 110].

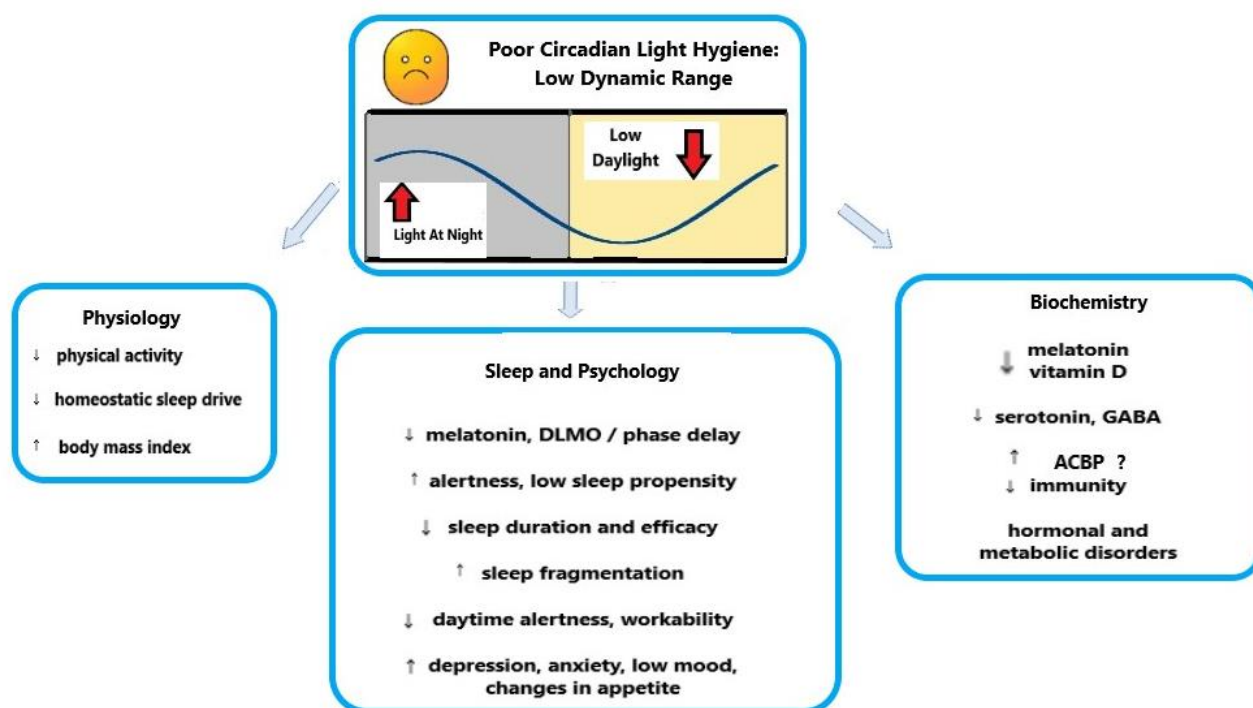


Figure 2. Health Risks Associated with Impaired Circadian Light Hygiene.

DLMO, Dim Light Melatonin Onset; GABA, Gamma-Aminobutyric Acid; ACBP, Acyl-Coenzyme-A-binding protein.

Circadian Light Hygiene: Definition

Circadian light hygiene (CLH) has become increasingly important in modern society due to its recognized role in maintaining health. CLH involves adjusting the 24-hour dynamic range of light exposure to support the natural sleep-wake cycle and circadian rhythms. Three key challenges hindering optimal CLH and negatively impacting human health are: 1) excessive exposure to artificial light at night (ALAN); 2) insufficient natural daylight; and 3) irregular light exposure patterns. These interacting factors necessitate a systematic approach to measurement and analysis.

Daylight vs. Nightlight: What Makes the Difference?

The impact of light on health is intrinsically linked to biological clocks and is strongly modulated by the circadian phase, varying from early morning to late evening [2]. A thorough understanding of phase response curves (PRCs) is essential to appreciate the diverse biological effects of light exposure at different times of day. The physical characteristics of light, such as intensity and spectrum, exert multifaceted influences on the body's internal biological clock, driving the phases of circadian rhythms [31, 32]. Ultraviolet light, particularly ultraviolet B (UVB, wavelength 280–315 nm), stimulates vitamin D production in the skin and exhibits antimicrobial properties. UVB irradiation can also activate vitamin D-independent pathways that mediate the effects of light. For instance, UVB activates p53 transcription, which induces ghrelin expression in mouse skin adipocytes; however, conditional p53 knockout abolishes this response and the associated food-seeking behavior in males. In females, estrogen disrupts the p53-chromatin interaction at the ghrelin promoter, inhibiting both ghrelin expression and food-seeking behavior following UVB

exposure [33]. Moreover, moderate UV exposure increases blood urocanic acid (UCA), which crosses the blood-brain barrier and is subsequently converted to glutamate via an intraneuronal metabolic pathway, enhancing synaptic release and improving behaviors related to learning and memory, thus highlighting a novel mechanism for sunlight-induced neurobehavioral changes [34]. Notably, opsins in skin cells play multiple roles, although not all are fully understood, in wound healing, pigmentation, hair growth, and skin aging [35].

Blue melanopic light effects are best quantified using melanopic Equivalent Daylight Illuminance (melanopic EDI), an internationally standardized metric reflecting the impact of light on intrinsically photosensitive retinal ganglion cells (ipRGCs). Melanopic EDI peaks around 480 nm and is closely associated with melatonin production and the entrainment of the circadian clock [2]. Infrared light may promote tissue healing and reduce inflammation by enhancing cellular metabolism and circulation [36], [Figure 1. Figure 1 \(left\)](#) illustrates the differences between visual and non-visual physiological and psychological effects of light with low versus high frequencies. [Figure 1 \(right\)](#) also provides schematics of approaches and measures that can be applied to assess optimal versus suboptimal CLH. Further details are provided in footnotes to [Figure 1](#). Morning light exposure facilitates earlier awakening and heightened alertness, effectively advancing the internal biological clock (circadian phase). Conversely, evening light exposure delays the circadian phase, promoting later sleep and wake times. Midday daylight exposure has a relatively minor effect on the circadian clock, but still enhances alertness and cognitive performance. Furthermore, natural light containing ultraviolet (UV) radiation supports vitamin D synthesis, potentially contributing to the synchronization of

biological clocks [37]. After midday, the benefits of natural daylight diminish, while the detrimental effects of artificial light at night (ALAN) become more pronounced [1, 2, 13, 38]. The timing of this transition can be influenced by genetic factors associated with the biological clock, which partially determine individual chronotypes and habitual sleep patterns. Additionally, the quantity and timing of sunlight exposure, which fluctuate throughout the year, are critical factors. The combination of daylight exposure and regular physical activity may exert a more substantial effect on the phase and amplitude of the circadian clock [1, 39]. Understanding PRCs is crucial for regulating sleep and wake times, especially for individuals experiencing jet lag or working shifts. Managing the duration of light exposure can help individuals align their sleep schedules with their lifestyles. The timing of exposure and the physical characteristics of light are critical determinants of sleep quality and overall health [1, 40-42]. In summary, morning and daytime light exposure benefits circadian health by enhancing rhythm synchronization and amplitude [32], while nighttime light exposure is associated with circadian misalignment and impaired health.

Benefits of Daylight

A significant portion of the urban population experiences chronic daylight deficiency, which has complex implications for sleep, mental health, and overall well-being [1, 2, 40, 41]. The positive effects of daylight on mental health are well-established, as light influences mood and cognitive function, primarily via melanopsin-containing retinal ganglion cells. These cells connect to brain regions involved in mood regulation and circadian rhythms [43, 44]. Consequently, elevated Beck Depression Inventory scores are associated with both insufficient daylight exposure and a later chronotype, reflecting the impact of insufficient natural light and excessive evening light on circadian phase delay [45, 46]. Additionally, the loss of retinal ganglion cells in glaucoma has been linked to increased depression scores [47]. Daylight exposure may increase sleep duration, potentially by facilitating earlier sleep onset [1, 31, 32], and can be used to manage seasonal affective disorder [48]. Exposure to sufficient natural, dynamic, or artificial blue light improves alertness, concentration, and cognitive function [49-51], sleep, and mood [52]. This could benefit strategies aimed at enhancing learning abilities and counteracting low motivation in evening-type students [53]. Furthermore, promoting increased exposure to early daylight may be a useful strategy for adjusting chronotype, as eveningness is associated with higher morbidity and mortality [54].

Light is also crucial in regulating metabolic processes, such as glucose homeostasis and thermogenesis. Recent studies suggest that light influences metabolism through brain circuits and the direct stimulation of metabolic tissues [55]. Disruptions in light exposure, such as those encountered during night shifts, can result in metabolic disorders. For example, morning exposure to bright light has been shown to decrease body fat and appetite in overweight women [56], while daytime illumination above 500 lux is associated with reduced body weight, regardless of sleep duration [42]. Individual metabolic responses to light vary significantly, influenced by genetic factors, gender, and age [57-59]. Light sensitivity can differ markedly among individuals, with genetic factors contributing to these variations [60]. Furthermore, the loss of retinal ganglion cells is associated with disruptions in lipid metabolism [61].

Daylight is essential for adequate vitamin D levels, which directly and indirectly regulate sleep and metabolism [63, 63]. Vitamin D receptors are present in brain regions related to sleep regulation, influencing both neurotransmitter synthesis and melatonin production [64]. Adequate vitamin D concentrations can improve mood and mitigate depressive symptoms, thus contributing to improved sleep quality [64]. A large-scale study in the USA ($n=25,534$) demonstrated that vitamin D concentrations are correlated with excessive sleep duration, particularly when combined with inadequate sunlight exposure [65]. Similar findings were reported in Brazil, where daylight deficiency and low vitamin D levels were associated with reduced sleep quality [66]. Our recent study suggests that increased exposure to blue and ultraviolet B daylight, especially during office hours, may have had a protective effect against severe acute respiratory syndrome-related coronavirus 2, SARS-CoV-2 infection [67].

Hazards of Light at Night

The impact of evening and nighttime light exposure on sleep is significantly influenced by light intensity, spectral composition, duration, and regularity [1, 2, 68]. A meta-analysis of seven studies involving 577,932 participants demonstrated that exposure to nighttime light is associated with a dose-dependent increase in sleep problems. Notably, indoor light sources have a more detrimental effect than outdoor light, with more pronounced sleep disturbances observed at light levels exceeding 5.8 $\text{NW/cm}^2/\text{cp}$ [69]. Increased nighttime exposure to short-wavelength light is associated with a higher incidence of sleep disorders, primarily due to the suppression of melatonin production. Even at low overall light levels, groups with greater exposure showed a higher prevalence of sleep disorders and decreased melatonin levels [70]. Artificial light at night (ALAN) is associated with delayed wake times, reduced sleep efficiency, and sleep fragmentation, though total sleep duration is not consistently affected [71]. However, ALAN can also delay the circadian phase in young adults, as indicated by dim light melatonin onset (DLMO) [72].

Sleep disruption has significant adverse effects on metabolism, increasing risks of metabolic disorders, obesity, diabetes, and cardiovascular disease [15, 16, 38, 40, 73-77]. An eight-year prospective study with 84,790 participants demonstrated that higher nighttime illuminance is associated with an increased risk of developing diabetes, particularly in individuals exhibiting reduced amplitude or irregular phase in their circadian rhythms under controlled light exposure conditions [15, 16]. Studies of seasonal variations in Arctic residents indicate that seasonal sleep phase alterations are associated with shifts in the melatonin phase, mirroring seasonal photoperiodic variations [38]. A reduced amplitude and delayed phase of light exposure in Arctic residents have been linked to negative metabolic changes in triglyceride and high-density lipoprotein levels, as evidenced by both cross-sectional and longitudinal analyses [38]. Some individuals show sensitivity to dim blue light, which can affect circadian health, suggesting that even minimal light exposure can have an impact [60]. The relationship between light exposure and metabolic health can be effectively modeled using a sinusoidal function mirroring the phase response curves (PRCs) of melatonin and lipid metabolism [78-80]. Specific genetic polymorphisms, such as the melatonin receptor 1 beta variant MTNR1B rs10830963, are associated with individual variations in response to glucose

tolerance tests (GTT) depending on the quantity and duration of daylight [81], with a more pronounced metabolic response to low-intensity evening blue light, often with alterations in the circadian pattern of body temperature [38]. Notably, a temperature threshold, which triggers adaptive thermogenesis in brown adipose tissue, is necessary for blue light-mediated modulation of glucose tolerance tests (GTT) in humans [82]. Furthermore, melatonin receptors share a common evolutionary ancestry with opsins, the primary light-sensitive chromophores in living organisms [83]. Light at night (LAN) has been shown to increase blood pressure irrespective of activity levels [84]. Nighttime light exposure disrupts sleep initiation and can start a vicious cycle of increased light exposure on subsequent nights, leading to further deviations in sleep timing [85]. Furthermore, LAN has been identified as a risk factor for various cancers [15, 23-27]. The adverse effects of LAN on sleep, circadian rhythms, hormonal balance, and metabolic health are largely mediated by the suppression of melatonin [86-88]. Reduced nighttime melatonin levels diminish the body's signals that promote sleep initiation, delaying sleep onset. Irregular light exposure disrupts the synchronization of the internal biological clock with the external environment, contributing to circadian disruption. Exposure to blue light, particularly from screens, enhances alertness and cognitive activity, counteracting the natural sleep drive. Consequently, exposure to light at night (LAN) results in frequent awakenings, reduced sleep quality, and sleep fragmentation [71, 85], as well as impaired mood [89, 90]. Chronic sleep disturbances and deprivation have adverse effects on both physical and mental health, contributing to mood disorders such as depression and anxiety, as well as cognitive impairments, including memory problems and reduced cognitive function.

Circadian disruption is closely associated with alterations in appetite, meal preferences, meal regularity, and the timing of food consumption [91-93]. Modified eating patterns can influence the circadian clock, as meal timing and composition can act as feedback signals to the internal biological clock. Appetite-regulating hormones such as leptin [94], ghrelin [95], and the recently described Acyl Coenzyme A binding protein (ACBP) [96] are all interconnected with circadian mechanisms and are expected to be affected by circadian disruptions resulting from poor circadian light hygiene (CLH). In addition to melatonin, neurotransmitters such as serotonin [97] and gamma-aminobutyric acid (GABA) [98] are modulated by light-dark cycles and influence circadian amplitude.

A recently discovered mechanism demonstrates the direct impact of light on glucose metabolism [82]. This pathway transmits light signals from intrinsically photosensitive retinal ganglion cells (ipRGCs) to vasopressin neurons in the hypothalamic supraoptic nucleus (SON), subsequently reaching the paraventricular nucleus and GABAergic neurons in the nucleus of the solitary tract, and ultimately affecting brown adipose tissue (BAT), without involving the brain's primary circadian pacemaker (SCN). In humans, this mechanism is dependent on thermogenesis, which is triggered by a lower ambient temperature [82]. Furthermore, while independent of the SCN, this mechanism in living organisms is still modulated by overt circadian rhythms of body temperature, peripheral rhythms within tissues and organs, and feedback loops within regulatory circuits, including dynamic shifts in appetite and eating patterns.

The consumption of foods rich in melatonin is associated with an earlier chronotype, reduced social jetlag, improved sleep quality, lower levels of depression, and reduced central adiposity in adolescents and young adults [100]. However, the precise biochemical mechanisms by which circadian light-dark cycles (CLH) influence appetite and metabolic alterations remain unclear, thus the question of whether daylight deficiency, light at night, or circadian phase shifts are the primary drivers of metabolic changes remains unanswered.

Regularity of Light Exposure

Consistent light exposure is also crucial for maintaining optimal circadian rhythms. Irregular light hygiene, from the perspective of circadian biological clocks, has been associated with decreased mood [44, 101] and irregular sleep patterns [101]. These irregular sleep-wake patterns, in turn, are linked to an increased risk of mortality [102]. One plausible explanation for this phenomenon is that inconsistent light signals may diminish the amplitude of circadian rhythms, thereby reducing the stability of the circadian system and increasing susceptibility to circadian disruption.

Overall, inadequate CLH is associated with a spectrum of adverse health outcomes, as illustrated in [Figure 2](#).

When interpreting data on the daily dynamics of light exposure, which are closely related to melatonin and physical activity, it is crucial to emphasize that the phase position and amplitude of these indicators are more informative and clinically relevant than average levels or comparisons to conditional "normal values" at any given time.

Circadian Light Hygiene: Measurement, Modeling, and Assessment

Health challenges arise from excessive evening or nocturnal light exposure, insufficient daylight, or a combination of these factors, potentially exacerbating each other's effects ([Table 1](#)). In addition to direct non-circadian effects [55, 82], a reduced circadian amplitude resulting from diminished daytime light and nighttime melatonin signaling, along with phase misalignment or instability caused by circadian disruption [103, 104], can act as mediating factors. Therefore, the most relevant indicators for assessing CLH are the amplitude and stability of the circadian phase, as a stable phase is intrinsically linked to the maintenance of a large amplitude. Health risks associated with inadequate CLH can be objectively measured and quantified using wearable devices [105, 106]. Analyzing dynamic patterns of light exposure presents analytical challenges, including the need for sensors with sufficient sensitivity to accurately capture a broad spectrum of light, from infrared to ultraviolet [106, 107], [Figure 1](#). Of particular importance is the measurement of melanopic sensitivity, often assessed through the melanopic equivalent daylight illuminance (EDI), since ipRGCs containing melanopsin are critical for transmitting photic signals to the brain's master clock, and are most responsive to blue light within the melanopic range [2, 108]. When quantifying the dynamic range of CLH, traditional methods – like comparing day and night exposure or assessing maximal and minimal illumination – may not provide a complete understanding of the actual risk. Instead, a quantitative approach emphasizing transitions between light and darkness may yield more effective insights [38, 109, 110].

Table 1. Circadian-Light-Hygiene (CLH) Associated Diseases by Mechanism and Co-Factors

Factor of compromised CLH	Health Hazards Mediator	Related Diseases	References
Light At Night (LAN)	Affected Sleep	Low sleep efficacy Insomnia	Hu, et al., 2023 [30] Xu, et al., 2023 [69] Mitsui, et al., 2022 [70]
	Altered lipid metabolism and/or appetite Excessive evening light	High Body Weight Metabolic Syndrome Diabetes Obesity Food addiction Cardiometabolic mortality	Danilenko, et al., 2013 [56] McFadden, et al., 2014 [73] Park, et al., 2019 [74] Fleury, et al., 2020 [40] Lai, et al., 2020 [75] Zhang, et al., 2023 [76] Kim, et al., 2023 [77] Gubin, et al., 2024 [38] Windred, et al., 2024 [15, 16]
	Elevated stress hormones	Endocrine diseases	Kim, et al., 2023; [77] Gubin, et al., 2024 [38]
	Eveningness Impaired mood	Anxiety Depression	Bedrosian & Nelson, 2017 [43] Walker, et al., 2020b [89] Paksarian, et al., 2020 [90]
	Impaired glucose Ambient temperature/gamma amino-butyric acid, GABA dependent glucose response	Type 2 Diabetes	Kim, et al., 2023 [77] Meng, et al., 2023 [82]
	Neurodegeneration Neuroinflammation	Alzheimer diseases Parkinson disease	Mazzoleni, et al., 2023 [28] Voigt, et al., 2024 [29]
	Impaired antioxidant defenses and cell cycle regulation	Cancer (e.g., breast cancer, prostate cancer)	Walker, et al., 2020a; [26] Anisimov, et al., 2004; [23] Stevens, et al., 2014 [25] Vinogradova, et al., 2010; [24] Touitou, et al., 2017 [13] Luo, et al., 2023 [27]
	Elevated blood pressure	Hypertension	Gubin, et al., 2017 [84] Kim, et al., 2023 [77]
	Disruption of circadian rhythm and clock-associated brain functions	Seasonal Affective Disorder (SAD) Cardiometabolic mortality	Melrose, 2015 [48] Windred, et al., 2024 [15, 16]
	Vitamin D deficiency	Disrupted sleep	Choi, et al., 2020 [65] de Menezes-Júnior, et al., 2023 [66]
Daytime/Sunlight Deficiency (DTLD)	Altered lipid/carbohydrate metabolism Lack of early light	High Body Weight Metabolic Syndrome	Reid, et al., 2014 [42] Harmsen, et al., 2022 [41] Ishihara, et al., 2022 [14] Weinert & Gubin, 2022 [39]
	Impaired immunity (mediation by physical activity or vitamin D?)		
	Altered metabolism due to lack of light reception	Glaucoma	Gubin, et al., 2022 [61]
	Impaired mood	Depression	Burns, 2021 [52] Gubin, et al., 2023 [47]
Circadian Disruption (LAN+DTLD misalignment)	Complex circadian disruption (misalignment): Reduced circadian amplitude	Multiple somatic and physiological diseases, Impaired cognitive function	Gubin, et al., 2024 [122] Harmsen, et al., 2022 [41] Windred, et al., 2024 [15, 16]
	Circadian phase shifted (delayed)	Sleep disorders	Burns, et al., 2021, 2023 [52, 58]
	Affected rhythms of neurotransmitters (melatonin, serotonin, GABA), Acyl Coenzyme A binding protein, ACBP (?) Eveningness	Chronic Fatigue Syndrome	Bedrosian & Nelson, 2017 [43] Granados-Fuentes, et al., 2024 [99] Dijk, et al., 2012 [3] Knutson & von Schantz, 2018 [54]

Similar to physical activity, non-parametric measures [111] can be applied to estimate light exposure in different spectral ranges: M10 – the average value of the 10-hour maximum; M10 Onset – the start of the M10 period; L5 – the average value of the 5-hour minimum; L5 Onset – the start of the L5 period. Furthermore, Relative Amplitude (RA=M10-L5/M10+L5) estimates the 24-hour dynamic range of exposure. In addition, Intradaily Variability (IV) and Interdaily Stability (IS) can be calculated [111, 112]. IV estimates the fragmentation of light exposure throughout the 24 hours, whereas IS approximates the degree of day-to-day consistency in light exposure.

Alternatively, cosinor modeling [113] can be applied, similar to its common use in analyzing circadian rhythms in physiological variables This approach is a parametric one.

$$y(t) = M + \sum_{i=1}^k A_i \cos(2\pi t/\tau_i + \phi_i), i=1, \dots, k$$

where τ_i are the periods; $\tau_1=24$ hours here. It typically accounts for most of the predictable variance, and its parameters (A1, ϕ_1) serve as biomarkers of Amplitude and Phase. The measured values (data) are denoted by y, t relates to time, and ϕ_i

are the acrophases (measures of the times when each fitted cosine component in the model reaches its maximum); MESOR (Midline Estimating Statistics of Rhythm) is the rhythm-adjusted average.

Since 24-hour average values of light exposure vary greatly across seasons, particularly at high latitudes [114], normalizing amplitude estimates can be important to reduce the influence of differences in mean light exposure, while emphasizing the dynamic range of exposure across the 24 hours. The normalized amplitude of light exposure, approximated as $NA=A/M$, may be one of the simplest indicators of CLH. NA may also outperform its proxy indicator, RA, as it tends to be more normally distributed. In contrast, RA can often reach saturation, equaling 1 when L5 is 0, leading to a highly uneven distribution in the data. For similar reasons, it is advisable to transform the data prior to analysis to bring their distribution closer to normality. A log transformation (after adding 1 to the data to avoid taking the logarithm of zero) or a square-root transformation typically helps meet the assumption of normality underlying model fitting and tests of equality among groups or conditions.

The fundamental principle that differentiates the beneficial from the harmful effects of light exposure on health, particularly its timing, remains not fully elucidated. Additionally, several critical questions remain unanswered, such as whether a sinusoidal or non-sinusoidal model should be used, and what types of misalignment primarily contribute to adverse outcomes: those related to activity/sleep patterns, melatonin phase, meal timing, or any other. Nevertheless, addressing light deficits in the morning and daytime versus excess exposure in the evening and night should be guided by recommendations for optimal illumination ranges based on the time of day [2], primarily focusing on the melanopic range within the blue light spectrum. Considering these recommendations over the entire 24-hour cycle, novel indices were proposed [38, 109, 110], which surpassed other measures in predicting metabolic health risks, even during the Arctic spring equinox, a relatively favorable period in terms of CLH conditions [38]. Given that physical activity and light exposure may act in tandem, the advocacy for facilitating circadian alignment and robust, large-amplitude rhythms has been proposed [39]. Deviations from the optimal phase relationship between light and activity, where the light phase precedes the activity phase by a particular lag, have also been linked to impaired metabolic health [115].

Melatonin as a Marker of Circadian Clock Phase and Light Hygiene

Precise estimation of the melatonin acrophase or dim light melatonin onset (DLMO) is paramount for evaluating the physiological impacts of light. These metrics serve as a precise measure of circadian rhythm phase and are regarded as the gold standard for assessing the body's response to light exposure [116]. Understanding the timing of melatonin production enables researchers to evaluate how light influences sleep patterns, hormonal regulation, and overall health, thereby facilitating improved management of light environments to promote optimal well-being. While DLMO is more frequently employed [117] owing to its reduced time and resource demands for sampling, the acrophase may be more suitable in field research when self-collected data is anticipated and consistent sampling quality across participants cannot be assured. Dim light melatonin onset and

offset are susceptible to data gaps, especially near the evening "inflation" and morning "deflation" phases, respectively, where melatonin exhibits rapid, hockey-stick-like changes [118]. The acrophase exhibits a strong correlation with DLMO and the phase of light exposure across seasons [38]. It is independent of subjective measures, which are necessary when determining basal levels with significant inter-individual variability, and is less prone to data gaps [113]. Notably, individual melatonin sensitivity to light shows approximately a 50-fold variation [60] and may be influenced by genes beyond core clock gene polymorphisms, including melatonin receptor genes [38].

Optimizing Circadian Light Hygiene/Human-Centric Lighting

Recommendations include bright light exposure in the morning and midday, followed by a gradual transition to dim lighting with "warm" tones in the evening to reduce blue light exposure. Creating a dark, cool, and quiet bedroom environment can enhance sleep quality and facilitate natural sleep onset. When screen use is unavoidable, utilizing blue light filters on devices or wearing blue light-blocking glasses is recommended. In specific circumstances, particularly for older adults or individuals with age-related conditions, low-dose melatonin supplementation may be warranted [119-123] and should be tailored by a chronobiology specialist to meet individual needs [104]. Modulating circadian light exposure during the day and melatonin release at night can enhance circadian rhythmicity by improving the amplitude, phase alignment, and stability of overt circadian signals. However, research specifically addressing light or melatonin therapies tailored to an individual's circadian phase or chronotype remains limited. Accordingly, personalized studies are necessary to determine how CLH should be modulated based on each individual's circadian phase [104, 123]. A recent phase 2 clinical trial demonstrated that targeted daylight therapy enhanced deep sleep quality in individuals with mild-to-moderate Parkinson's disease. Notably, no significant differences were observed between controlled daylight therapy and melanopsin-boosting light therapy [124].

Optimizing CLH is paramount for preventing circadian disruption, promoting biological clock health, and modulating circadian rhythm phases, sleep patterns, and sleep quality. Effective CLH strategies encompass regular exposure to natural daylight for at least 90 minutes daily, outdoor physical activity, and the use of optimal artificial light sources with appropriate intensity and color temperature at home and in the workplace [125]. In regions experiencing insufficient natural light due to seasonal variations, the implementation of "smart light" systems, such as biodynamic, anthropocentric, or human-centric lighting (HCL), becomes particularly vital [126-128]. Future approaches should consider individualized light modes and work schedules based on age, genetic risk factors, chronotype, and light sensitivity to achieve optimal health and economic outcomes.

Conclusions

This review provides compelling evidence underscoring the paramount importance of CLH for promoting overall health, well-being, and longevity in contemporary society. We emphasize the critical role of a sufficient dynamic range of light exposure, characterized by ample natural or appropriately modeled artificial daylight during the day and the avoidance of artificial light at

night. We have elucidated the rationale behind its essential role in maintaining healthy sleep patterns, mood stability, and cardiometabolic function. Furthermore, we have identified three primary challenges to optimal CLH: light pollution, insufficient daylight, and irregular light exposure patterns. To address these challenges, we propose a systematic approach for measuring and analyzing light exposure, essential for developing effective health strategies. We have also introduced novel indices to quantify and evaluate optimal versus suboptimal CLH. Ultimately, we aim to heighten public health awareness, as well as ongoing and future initiatives focused on maintaining healthier environments to promote mind-body health and longevity.

Conflict of Interest

The authors declare no conflict of interest.

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