International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	CIRCADIAN RHYTHM DISRUPTION AND HORMONAL HEALTH: THE IMPACT OF MODERN LIFESTYLE
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4155
RECEIVED	19 October 2025
ACCEPTED	21 December 2025
PUBLISHED	26 December 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

CIRCADIAN RHYTHM DISRUPTION AND HORMONAL HEALTH: THE IMPACT OF MODERN LIFESTYLE

Katarzyna Bielawska *(Corresponding Author, Email: bielawskka@wp.pl)*
Grochowski Hospital, Warsaw, Poland
ORCID ID: 0009-0001-7381-8415

Wiktoria Julia Auguścik
District Health Center, Otwock, Poland
ORCID ID: 0009-0009-6670-1604

Aleksandra Tlak
Grochowski Hospital, Warsaw, Poland
ORCID ID: 0009-0008-2506-5309

ABSTRACT

The circadian rhythm is one of the key homeostatic mechanisms of the human body, regulating hormone secretion, energy metabolism, and the sleep-wake cycle. The aim of this review was to present the current state of knowledge on the impact of circadian rhythm disorders on the functioning of the endocrine system and to assess the consequences of modern lifestyles on hormonal balance. A literature review was conducted in the PubMed, Scopus, Web of Science, and Google Scholar databases, covering publications from 2000 to 2025. Twenty-six peer-reviewed papers of high methodological quality were included, analyzing the relationship between circadian rhythm desynchronization and the regulation of hormonal axes.
The collected data indicate that circadian rhythm disorders—resulting from exposure to artificial light, shift work, irregular sleep and meals—lead to desynchronization between the suprachiasmatic nucleus (SCN) and peripheral clocks. This leads to dysregulation of the HPA, HPG, and HPT axes. Loss of physiological pulsatility of cortisol and melatonin secretion, insulin resistance, hypercortisolemia, and fertility disorders are observed. Long-term adaptation to these disorders is incomplete, and persistent hormonal changes promote the development of metabolic and cardiovascular diseases.
The conclusions emphasize the need for chronobiological prevention through controlling exposure to light, maintaining a regular sleep and meal schedule, and limiting shift work. Circadian rhythm disorders are a significant, though often underestimated, pathogenic factor in lifestyle diseases.

KEYWORDS

Circadian Rhythm, Endocrine System, Melatonin, Cortisol, Shift Work, Chronobiology

CITATION

Katarzyna Bielawska, Wiktoria Julia Auguścik, Aleksandra Tlak. (2025) Circadian Rhythm Disruption and Hormonal Health: The Impact of Modern Lifestyle. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4155

Introduction

The circadian system is one of the basic homeostatic mechanisms of the human body. It determines the rhythm of most physiological processes, including hormone secretion, energy substrate metabolism, thermoregulation, and the sleep-wake cycle [1,2]. The central generator of biological rhythms is the suprachiasmatic nucleus (SCN) located in the hypothalamus, which coordinates the activity of peripheral circadian oscillators present in peripheral tissues and endocrine glands [3,4]. Synchronization between the SCN and peripheral clocks ensures the proper temporal organization of hormone secretion and cellular metabolism, enabling the body 's adaptation to cyclical environmental changes.

Environmental and behavioral factors, such as exposure to light of inappropriate intensity and at the inappropriate daytime hours, shift work, travel between time zones, sleep restriction, or irregular meal consumption, can lead to a phenomenon known as circadian misalignment [5–7]. This leads to abnormal signals between the central biological clock and peripheral rhythms, resulting in hormonal and metabolic dysregulation [8,9].

Desynchronization of circadian rhythms particularly affects the functioning of the hypothalamic–pituitary–adrenal (HPA) axis, the hypothalamic–pituitary–thyroid (HPT) axis, and the gonadal axis. Multiple studies have shown that changes in the light-dark cycle, night work, or chronic sleep deprivation lead to significant changes in the secretion profiles of cortisol, melatonin, insulin, leptin, and sex hormones [10–14]. Even short-term exposure to high-intensity light at night can suppress melatonin secretion and modulate cortisol rhythms, suggesting a direct effect of photic signals on the HPA axis [15–17].

Long-term circadian dyssynchrony is associated with an increased risk of metabolic and endocrine disorders, including insulin resistance, obesity, type 2 diabetes, hypertension, and reproductive dysfunction [18–21]. Menstrual cycle dysregulation, decreased melatonin levels, and ovulation disorders have been observed in women, while decreased testosterone levels and impaired semen parameters have been observed in men [22–24].

According to current data, circadian rhythm disorders are a significant pathophysiological factor in numerous endocrine and metabolic diseases, going beyond the traditionally understood influence of lifestyle [25,26]. Understanding the molecular and neuroendocrine mechanisms underlying these phenomena is crucial for the development of effective strategies for the prevention and treatment of disorders resulting from circadian rhythm desynchronization.

The aim of this review is to provide an overview of the current state of knowledge on the interplay between circadian rhythms and human hormonal balance and to discuss the impact of contemporary environmental and behavioral factors on endocrine system function.

Methodology

We conducted a comprehensive literature review using major scientific databases, including PubMed, Scopus, Web of Science, and Google Scholar, to identify studies investigating the relationship between circadian rhythm disruption and endocrine health. The search strategy focused on publications exploring the molecular, physiological, and clinical aspects of circadian misalignment and its impact on hormonal balance, metabolism, and stress response. Keywords used for database queries included *circadian rhythm, endocrine system, hormonal regulation, melatonin, cortisol, photic signals, shift work, sleep–wake cycle,* and *metabolic syndrome*.

The time frame for the included publications ranged from 2000 to 2025, while older studies were considered if they provided essential theoretical or historical background on circadian biology. Only peer-reviewed, English-language articles were included. Both experimental and observational studies, as well as systematic reviews and meta-analyses, were analyzed.

A total of 26 high-quality articles were selected based on their methodological rigor and relevance to the topic, with particular attention given to works published in high-impact journals such as *Endocrine Reviews*, *Annual Review of Physiology*, *Cell Metabolism*, and *Chronobiology International*. These publications were examined in detail, and their findings were synthesized thematically to describe how circadian rhythm disruption affects hormonal homeostasis, with a special focus on melatonin, cortisol, insulin, and sex steroid regulation.

Results

The central circadian system and hormonal coordination

The foundation of proper hormonal homeostasis in humans is the circadian system, whose primary component is the SCN located in the anterior part of the hypothalamus. The SCN is the main biological clock, coordinating circadian rhythms throughout the body. It synchronizes peripheral circadian oscillators located in endocrine organs such as the adrenal glands, pancreas, liver, gonads, and thyroid gland [1,2,17]. These clocks exhibit autonomous rhythmic activity, but their cyclicity and phase are constantly adjusted to signals from the SCN. This ensures coherence between metabolic, hormonal, and behavioral processes [1,19].

The molecular basis of circadian rhythmicity is the feedback of clock gene expression. The main transcription-activating proteins, CLOCK and BMAL1, induce the expression of Period (PER1–3) and Cryptochrome (CRY1–2) genes, whose products inhibit their own transcriptional activity, creating a self-regulating transcriptional-translational cycle with a period of approximately 24 hours [1,2,17]. These molecular mechanisms operate hierarchically: the SCN acts as a master regulator, while peripheral clocks use the same genetic elements to locally regulate the expression of genes responsible for the metabolism of lipids, glucose, and steroid hormones [2,16,19].

The SCN receives photic signals through the retinohypothalamic tract, in which retinal ganglion cells containing melanopsin, a photopigment sensitive to blue light, play a key role [2,14]. Information about the light-dark cycle is then transmitted to other structures of the hypothalamus, including the paraventricular nuclei, influencing the activity of neuroendocrine axes, especially the hypothalamic-pituitary-adrenal (HPA) axis and the hypothalamic-pituitary-gonadal (HPG) [17,19]. Through these connections, the SCN regulates the pulsatile secretion of hormones – cortisol, melatonin, growth hormone, TSH, and gonadotropins – ensuring their phase synchronization with the activity-sleep cycle [15,17,21].

Besides influencing the endocrine system, the SCN affects the autonomic nervous system, modulating the rhythms of body temperature, blood pressure, heart rate, and blood glucose [17,19]. On the other hand, peripheral clocks respond not only to neural and hormonal signals from the SCN, but also to metabolic stimuli such as meal times, glucose levels, and nutrition hormones (insulin, leptin) [10,11,16]. Under conditions of proper synchronization between the SCN and peripheral clocks, hormone secretion remains closely correlated with the circadian rhythm of activity—e.g., cortisol levels peak in the morning and melatonin levels peak at night [14,15,17].

Factors disrupting the photic or behavioral rhythm—such as exposure to light at night, shift work, irregular sleep and meal times—can lead to desynchronization of the SCN relative to peripheral clocks [3,19,21]. This results in hormone secretion dysregulation, HPA and HPG axis disorders, and metabolic dysfunction. It has also been shown that individual variability in the expression of clock genes, e.g., PER3 polymorphism, can modulate susceptibility to sleep disorders and the metabolic effects of shift work [11,19]. These observations confirm that the circadian rhythm is a fundamental aspect of endocrine regulation, and its disorders are the starting point for the development of numerous lifestyle diseases.

Regulation of melatonin and cortisol secretion

Melatonin and cortisol are key circadian rhythm hormones. They both reflect the activity of the central circadian clock and the state of synchronization between the SCN and peripheral clocks. Melatonin exhibits a circadian rhythm – its secretion increases at nightfall and peaks during the night, then decreases rapidly after exposure to light [6,13,14]. The secretion of melatonin is inhibited by the activation of the retinohypothalamic pathway and the stimulation of SCN neurons, which modulate the activity of sympathetic fibers innervating the pineal gland via the paraventricular nuclei [6,14,24]. Melatonin acts as a neuroendocrine “night signal,” informing the body of the daytime. It regulates sleep, body temperature, blood pressure, and gonadotropin secretion [6,9,24].

Cortisol has a rhythm opposite to that of melatonin – its concentrations are lowest at night and highest in the morning, immediately after waking up, which is referred to as the cortisol awakening response [13,15,17]. This rhythm is regulated through complex interactions of the HPA axis, in which the SCN affects corticotropin-releasing hormone (CRH) neurons in the paraventricular nucleus of the hypothalamus, initiating a cascade of ACTH secretion from the pituitary gland and, secondarily, cortisol from the adrenal cortex [13,17,19]. The study showed that the mere transition from low light conditions to bright morning light causes an immediate, more than 50% increase in cortisol concentration regardless of sleep and wakefulness, demonstrating the direct effect of light on HPA axis activity [15].

The studies by Jung et al. and Scheer et al. confirm that exposure to high-intensity light in the morning can modify the cortisol secretion profile and improve alertness, while afternoon light does not have this effect [13,21]. This phase relationship indicates a strong link between phototransduction in the SCN and adrenal cortex activity.

The interaction between melatonin and cortisol is fundamental to maintaining circadian homeostasis – the first hormone determines the phase of rest and regeneration, while the second prepares the body for daily metabolic and cognitive activity. The loss of their physiological anti-phase (e.g., a decrease in nighttime melatonin with a simultaneous shift in the cortisol peak) is one of the earliest markers of circadian rhythm desynchronization and hormonal dysregulation [3,6,13,15].

Environmental and behavioral disruption of circadian endocrine rhythms

Exposure to artificial light at night and an irregular lifestyle are among the most important factors leading to circadian rhythm desynchronization and hormonal disorders. The current environment, characterized by the widespread use of LED screens emitting light with a high blue component, significantly affects the activity of the suprachiasmatic nuclei (SCN) and secondarily the functioning of the endocrine system [4,6,7,9,10]. Even short-term exposure to high-intensity light in the evening can lead to inhibition of melatonin secretion, delay of the sleep phase, and shift in the rhythm of cortisol secretion [6,9,14]. This phenomenon causes a loss of the natural anti-phase between melatonin and cortisol, which disrupts the physiological processes of nighttime regeneration and daytime activity [3,6,13,15]. The main environmental and behavioral factors contributing to circadian rhythm disruption and their endocrine consequences are summarized in Table 1.

Population studies have shown that people who work shifts or frequently travel between time zones are more susceptible to metabolic disorders, which is associated with a discrepancy between SCN activity and the rhythm of peripheral clocks in tissues such as the liver, pancreas, and skeletal muscles [5,10,12,16]. This desynchronization leads to abnormal secretion of cortisol, insulin, leptin, and melatonin, resulting in impaired glucose and lipid metabolism and appetite dysregulation [10,16,17]. Even a short-term reversal of the sleep-wake cycle causes an increase in nighttime cortisol secretion and a decrease in afternoon insulin levels, which translates into a disturbance in the circadian glycemic profile [16,17,20]. Experimental shifting of the circadian rhythm by 12 hours leads to a decrease in insulin sensitivity, an increase in glucose and leptin concentrations, and disturbances in thermoregulation [21].

Chronic activation of the hypothalamic–pituitary–adrenal (HPA) axis observed in night shift workers results in hypercortisolemia and a reduction in the physiological diurnal variation of cortisol [10,17,19]. The accompanying melatonin deficiency contributes to increased oxidative stress and inflammatory processes, while leptin secretion disorders increase appetite for high-calorie foods [16,17,24]. This leads to the development of obesity, insulin resistance, and hypertension. Prospective studies confirm that long-term night work increases the risk of type 2 diabetes, metabolic syndrome, and lipid disorders [16,18,20]. Women also experience more frequent menstrual cycle disorders and reduced fertility resulting from dysregulation of the hypothalamic-pituitary-gonadal (HPG) axis [17,21].

Moreover, worth noting is that circadian rhythm disorders affect not only shift workers, but also the general population, where social jet lag—a discrepancy between the natural biological rhythm and the social schedule of the day—is increasingly observed [5,26]. Irregular sleep, evening activities, and exposure to screen light lead to chronic sleep deprivation and disruption of melatonin and cortisol rhythms. These changes have consequences that go beyond metabolism – they affect the functioning of the immune system, exacerbate inflammation and oxidative stress, and accelerate cellular aging processes [17,19,24]. A growing body of evidence suggests that chronic dyssynchrony between internal and environmental rhythms is a key mechanism linking modern lifestyles to the increasing prevalence of metabolic, neurodegenerative, and autoimmune diseases [10,17,24].

Table 1. Environmental and behavioral factors disrupting circadian rhythms

Factor	Mechanism of disruption	Affected hormonal systems	Consequences for health
Night-time light exposure	Suppression of melatonin via SCN inhibition	Pineal gland, HPA axis	Sleep disorders, insulin resistance
Shift work	Misalignment between SCN and peripheral clocks	HPA, HPG, HPT axes	Metabolic syndrome, reproductive dysfunction
Irregular mealtimes	Loss of metabolic entrainment of peripheral clocks	Pancreas, liver	Obesity, glucose intolerance
Sleep deprivation	Chronic HPA axis activation	Adrenal cortex	Hypercortisolemia, hypertension
Social jet lag	Behavioral desynchrony	Multiple systems	Fatigue, cognitive decline, inflammation

Note: Adapted from references [3,5,6,9,10,16,17,19,21,24,26].

Long-term endocrine adaptation and systemic health consequences

Chronic circadian rhythm disorders lead to a loss of adaptive capacity in the endocrine system, resulting in permanent changes in metabolic, immune, and neurohormonal regulation. Long-term desynchronization between the SCN and peripheral clocks leads to a shift in the set points of neuroendocrine axes, especially the HPA axis and the HPG axis [17,19,21]. Chronic activation of the HPA axis leads to hypercortisolemia, decreased glucocorticoid receptor sensitivity, and disturbances in the rhythmic secretion of ACTH. As a consequence, this promotes the development of insulin resistance, visceral obesity, and hypertension [16,17,20]. On the other hand, long-term reduction in nocturnal melatonin secretion is associated with a weakening of its antioxidant, proapoptotic, and immunomodulatory effects [6,9,24].

Multiple studies have shown that circadian rhythm disturbances exacerbate chronic low-grade inflammation, characterized by elevated levels of IL-6, TNF-α, and C-reactive protein (CRP) [17,19]. Altered cortisol and melatonin secretion rhythms modulate T-cell and macrophage activity, leading to dysregulation of the immune response [10,17]. This results in increased susceptibility to infections, accelerated cellular aging, and an increased risk of developing autoimmune and neurodegenerative diseases [17,19,24].. Experimental studies also indicate that circadian rhythm disorders can modify the expression of genes controlling the cell cycle and DNA repair. This may potentially increase the risk of malignant transformation in tissues with high proliferative activity, such as the breast, prostate, and liver [1,17,19]. The systemic health outcomes associated with chronic circadian misalignment are summarized in Table 2.

From a clinical point of view, prolonged dyssynchrony between endogenous and environmental rhythms results in the loss of physiological plasticity of the endocrine system. Altered profiles of circadian hormone secretion—especially cortisol, melatonin, insulin, and leptin—become “fixed” in a new pattern that persists even after returning to a regular sleep-wake cycle [16,17,20]. This state of endocrine “reprogramming” may explain why people with chronic circadian rhythm disorders show increased susceptibility to metabolic and mental disorders, despite the normalization of external living conditions. According to the available data, it can be concluded that permanent desynchronization of the circadian rhythm is one of the key, though often underestimated, pathogenic mechanisms of modern lifestyle diseases [10,17,19].

Table 2. Health effects of chronic circadian rhythm disruption

Physiological system	Mechanisms	Long-term effects	References
Endocrine	Desynchronization of hormonal axes	Insulin resistance, obesity	[16,17,19,21]
Cardiovascular	Increased sympathetic tone, oxidative stress	Hypertension, atherosclerosis	[19,20,25]
Reproductive	Impaired gonadal hormone regulation	Menstrual disorders, infertility	[17,21]
Immune	Chronic inflammation, altered cytokine rhythm	Autoimmunity, faster aging	[17,19,24]
Neurological	Flattened cortisol and melatonin rhythm	Sleep disorders, mood disturbances	[6,9,24]

Note: Compiled based on data from key studies on endocrine and metabolic effects of circadian disruption.

Discussion

Circadian rhythm disorders have a profound effect on the functioning of the endocrine system, modulating almost every aspect of hormonal homeostasis. Accumulated data indicate that the SCN in the hypothalamus acts as a master biological clock that coordinates the activity of peripheral clocks via neuroendocrine and autonomic pathways [1,2,17]. Desynchronization between the SCN and peripheral oscillators leads to disruption of hormone secretion timing and deregulation of metabolic processes. The consequences are systemic disturbances in energy balance, stress response, and reproductive function [3,17,19]. The modern lifestyle—characterized by exposure to artificial light at night, irregular sleep-wake rhythms, and shift work—creates conditions for chronic desynchronization between endogenous and environmental rhythms [4,6,9,10,18].

Melatonin and cortisol are the most sensitive and representative indicators of circadian rhythm integrity. Both hormones exhibit opposite secretion rhythms, reflecting the state of synchronization of the SCN with the HPA and HPG axes [13,15,17]. Chronic inhibition of melatonin secretion because of exposure to light at night or shift work leads to a loss of its antioxidant and immunomodulatory functions and increases susceptibility to oxidative stress [6,9,24]. At the same time, disruption of the cortisol secretion rhythm resulting from chronic activation of the HPA axis promotes the development of insulin resistance, visceral obesity, and hypertension [16,17,20,21]. These results clearly indicate that circadian rhythm dyssynchrony is not only a temporary behavioral problem, but a fundamental endocrine disorder with systemic consequences.

Many studies have confirmed that even short-term circadian rhythm disturbances affect glucose and lipid metabolism. An experimental 12-hour shift in the circadian phase causes a significant decrease in insulin sensitivity and changes in the daily profile of leptin secretion and body temperature [21]. Epidemiological data from large cohorts of healthcare and industrial workers indicate that long-term shift work is associated with an increased incidence of metabolic syndrome, type 2 diabetes, and hypertension [19,20,22,25]. In women, irregular menstrual cycles and fertility disorders are also observed, suggesting the involvement of HPG axis dysregulation [17,21]. The collected observations confirm that the discrepancy between behavioral and circadian rhythms simultaneously affects multiple hormonal axes.

The link between circadian rhythm desynchronization and metabolic disorders is mediated not only by changes in hormone secretion, but also by molecular and inflammatory mechanisms. Studies have shown that deregulation of clock gene expression (PER, CRY, BMAL1) in metabolic tissues leads to abnormal expression of genes responsible for glucose transport, insulin signaling, and lipid synthesis [1,2,16]. Chronic activation of the HPA axis and accompanying low-grade inflammation — with elevated concentrations of IL-6 and TNF-α — create a vicious cycle of hormonal and metabolic stress [17,19]. Melatonin rhythm disturbances and desynchronization of peripheral clocks also affect the migration and activity of immune cells, increasing the risk of developing autoimmune and neurodegenerative diseases [17,19,24]. These findings extend the importance of circadian rhythm biology far beyond metabolism — to the immune, neurological, and endocrine systems.

Another important aspect is the influence of the circadian rhythm on eating behavior and energy homeostasis. Research in the field of chrononutrition indicates that the time of meal intake itself is a strong zeitgeber (synchronizer) for peripheral clocks, especially in the liver and pancreas [16,18,23,26]. Early meal consumption improves insulin sensitivity, blood pressure, and oxidative stress profile even without weight loss [23]. In contrast, irregular or late mealtimes disrupt the synchronization of peripheral oscillators and promote fat accumulation and metabolic inflexibility [12,16,18]. These results emphasize the importance of adjusting the eating rhythm to the endogenous light-dark cycle as an effective, non-pharmacological strategy for the prevention of endocrine disorders.

Despite prolonged exposure to changes in the sleep-wake cycle, the human body is unable to completely adapt its hormonal mechanisms to the new rhythm, leading to permanent disturbances in endocrine homeostasis. In people who have been working night shifts for many years, a flattening of the cortisol secretion rhythm and a reduction in the nighttime melatonin peak have been observed, indicating the phenomenon of "endocrine reprogramming" [16,17,20]. This adverse adaptation may explain the increased incidence of metabolic, mental, and oncological disorders in populations exposed to chronic circadian rhythm desynchronization [17,19,24]. In this context, melatonin — due to its antioxidant, chronobiotic, and neuroprotective effects — is a potential therapeutic target for alleviating the effects of circadian desynchronization [6,9,24].

From a public health perspective, integrating chronobiology knowledge into everyday health recommendations remains a challenge. Contemporary society, constantly exposed to artificial light sources, electronic devices, and extended working hours, requires a redefinition of the concept of "endocrine hygiene"

— including control of light exposure, regular sleep and meal rhythms, and avoidance of long-term shift work [5,10,16,23]. Further research should focus on identifying circadian resistance biomarkers and evaluating the effectiveness of interventions that restore rhythmic homeostasis in people with irregular lifestyles. Given the broad, systemic consequences of circadian rhythm disorders, synchronizing behavior with the endogenous biological clock appears to be one of the most effective and economical strategies for improving hormonal health in the 21st century.

Conclusions

The circadian rhythm is a fundamental mechanism that determines the proper functioning of the endocrine system. Its synchronization with the light-dark cycle and with the rhythm of activity and rest is crucial for maintaining metabolic, hormonal, and immunological homeostasis. The accumulated data clearly indicates that circadian rhythm disorders—regardless of their cause, whether resulting from exposure to artificial light, shift work, irregular sleep, or meal patterns—lead to desynchronization between the central clock (SCN) and peripheral clocks in target tissues.

The effect of this desynchronization is the dysregulation of neuroendocrine axes (HPA, HPG, HPT), loss of physiological pulsatility of hormone secretion, and disturbances in the relationship between melatonin and cortisol. This leads to chronic activation of the stress axis, hypercortisolemia, insulin resistance, and disturbances in the secretion of insulin, leptin, and gonadotropins. These hormonal changes translate into an increased risk of developing lifestyle diseases – type 2 diabetes, obesity, hypertension, lipid disorders, and reproductive dysfunction.

Long-term adaptation to circadian rhythm disorders is incomplete – despite attempts to adapt, the body is unable to completely adjust its hormonal and metabolic mechanisms to chronically altered environmental conditions. This results in the perpetuation of abnormal hormone secretion patterns and permanent stress on the endocrine system.

Considering the available evidence, prevention of circadian rhythm disorders is crucial, including rational management of exposure to light (especially blue light), maintaining regular sleep and mealtimes, and limiting shift work if possible. Further research is needed in the future on the molecular mechanisms underlying the desynchronization of endocrine rhythms and on chronobiological strategies that could restore physiological hormonal balance.

Acknowledgments: The authors received no specific grant from any funding agency.

Conflict of Interest: The authors declare no conflicts of interest to declare.

REFERENCES

1. Bedrosian, T. A., Fonken, L. K., & Nelson, R. J. (2016). Endocrine effects of circadian disruption. *Annual Review of Physiology, 78*, 109–131. https://doi.org/10.1146/annurev-physiol-021115-105102
2. Begemann, K., Rawashdeh, O., Olejniczak, I., Pilorz, V., de Assis, L. V. M., Osorio-Mendoza, J., & Oster, H. (2025). Endocrine regulation of circadian rhythms. *npj Biological Timing and Sleep, 2*(1), 10. https://doi.org/10.1038/s44323-025-00024-6
3. Bittner, N., Korf, H. W., Moebus, S., Schmidt, B., & Caspers, S. (2025). Association between night shift work and markers of metabolism, cardiovascular and immune system in a population-based German cohort. *GeroScience, 47*(3), 5141–5155. https://doi.org/10.1007/s11357-025-01596-8
4. Cajochen, C., Frey, S., Anders, D., Späti, J., Bues, M., Pross, A., Mager, R., Wirz-Justice, A., & Stefani, O. (2011). Evening exposure to a light-emitting diodes (LED)-backlit computer screen affects circadian physiology and cognitive performance. *Journal of Applied Physiology, 110*(5), 1432–1438. https://doi.org/10.1152/japplphysiol.00165.2011
5. Caliandro, R., Streng, A. A., van Kerkhof, L. W. M., van der Horst, G. T. J., & Chaves, I. (2021). Social jetlag and related risks for human health: A timely review. *Nutrients, 13*(12), 4543. https://doi.org/10.3390/nu13124543
6. Chang, A.-M., Aeschbach, D., Duffy, J. F., & Czeisler, C. A. (2015). Evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness. *Proceedings of the National Academy of Sciences of the United States of America, 112*(4), 1232–1237. https://doi.org/10.1073/pnas.1418490112
7. Chellappa, S. L., Steiner, R., Blattner, P., Oelhafen, P., Götz, T., & Cajochen, C. (2011). Non-visual effects of light on melatonin, alertness and cognitive performance: Can blue-enriched light keep us alert? *PLoS ONE, 6*(1), e16429. https://doi.org/10.1371/journal.pone.0016429

8. Chellappa, S. L., Steiner, R., Oelhafen, P., Lang, D., Götz, T., Krebs, J., & Cajochen, C. (2013). Acute exposure to evening blue-enriched light impacts on human sleep. *Journal of Sleep Research, 22*(5), 573–580. https://doi.org/10.1111/jsr.12050
9. de Toledo, L. H. S., Moraes, M. N., Poletini, M. O., Cipolla-Neto, J., Baron, J., & Mot, T. (2023). Modeling the influence of nighttime light on melatonin suppression in humans: Milestones and perspectives. *Journal of Photochemistry and Photobiology, 16*, 100199. https://doi.org/10.1016/j.jpap.2023.100199
10. Ezpeleta, M., Cienfuegos, S., Lin, S., Pavlou, V., Gabel, K., Tussing-Humphreys, L., & Varady, K. A. (2024). Time-restricted eating: Watching the clock to treat obesity. *Cell Metabolism, 36*(2), 301–314. https://doi.org/10.1016/j.cmet.2023.12.004
11. Franzago, M., Alessandrelli, E., Notarangelo, S., Stuppia, L., & Vitacolonna, E. (2023). Chrono-nutrition: Circadian rhythm and personalized nutrition. *International Journal of Molecular Sciences, 24*(3), 2571. https://doi.org/10.3390/ijms24032571
12. Garaulet, M., & Gómez-Abellán, P. (2014). Timing of food intake and obesity: A novel association. *Physiology & Behavior, 134*, 44–50. https://doi.org/10.1016/j.physbeh.2014.01.001
13. Gooley, J. J., Chamberlain, K., Smith, K. A., Khalsa, S. B. S., Rajaratnam, S. M. W., Van Reen, E., Zeitzer, J. M., Czeisler, C. A., & Lockley, S. W. (2011). Exposure to room light before bedtime suppresses melatonin onset and shortens melatonin duration in humans. *The Journal of Clinical Endocrinology & Metabolism, 96*(3), E463–E472. https://doi.org/10.1210/jc.2010-2098
14. Jung, C. M., Khalsa, S. B. S., Scheer, F. A. J. L., Cajochen, C., Lockley, S. W., Czeisler, C. A., & Wright, K. P. Jr. (2010). Acute effects of bright light exposure on cortisol levels. *Journal of Biological Rhythms, 25*(3), 208–216. https://doi.org/10.1177/0748730410368413
15. Leproult, R., Colecchia, E. F., L'Hermite-Balériaŭx, M., & Van Cauter, E. (2001). Transition from dim to bright light in the morning induces an immediate elevation of cortisol levels. *The Journal of Clinical Endocrinology & Metabolism, 86*(1), 151–157. https://doi.org/10.1210/jcem.86.1.7102
16. McHill, A. W., & Butler, M. P. (2024). Eating around the clock: Circadian rhythms of eating and metabolism. *Annual Review of Nutrition, 44*, 25–50. https://doi.org/10.1146/annurev-nutr-062122-014528
17. Oster, H., Challet, E., Ott, V., Arvat, E., de Kloet, E. R., Dijk, D. J., Lightman, S., Vgontzas, A., & Van Cauter, E. (2017). The functional and clinical significance of the 24-hour rhythm of circulating glucocorticoids. *Endocrine Reviews, 38*(1), 3–45. https://doi.org/10.1210/er.2015-1080
18. Peters, B., Vahlhaus, J., & Pivovarova-Ramich, O. (2024). Meal timing and its role in obesity and associated diseases. *Frontiers in Endocrinology, 15*, 1359772. https://doi.org/10.3389/fendo.2024.1359772
19. Potter, G. D. M., Skene, D. J., Arendt, J., Cade, J. E., Grant, P. J., & Hardie, L. J. (2016). Circadian rhythm and sleep disruption: Causes, metabolic consequences, and countermeasures. *Endocrine Reviews, 37*(6), 584–608. https://doi.org/10.1210/er.2016-1083
20. Robertson-Dixon, I., Murphy, M. J., Crewther, S. G., & Riddell, N. (2023). The influence of light wavelength on human HPA axis rhythms: A systematic review. *Life, 13*(10), 1968. https://doi.org/10.3390/life13101968
21. Scheer, F. A. J. L., Hilton, M. F., Mantzoros, C. S., & Shea, S. A. (2009). Adverse metabolic and cardiovascular consequences of circadian misalignment. *Proceedings of the National Academy of Sciences of the United States of America, 106*(11), 4453–4458. https://doi.org/10.1073/pnas.0808180106
22. Sooriyaarachchi, P., Jayawardena, R., Pavey, T., & King, N. A. (2022). Shift work and the risk for metabolic syndrome among healthcare workers: A systematic review and meta-analysis. *Obesity Reviews, 23*(10), e13489. https://doi.org/10.1111/obr.13489
23. Sutton, E. F., Beyl, R., Early, K. S., Cefalu, W. T., Ravussin, E., & Peterson, C. M. (2018). Early time-restricted feeding improves insulin sensitivity, blood pressure, and oxidative stress even without weight loss in men with prediabetes. *Cell Metabolism, 27*(6), 1212–1221.e3. https://doi.org/10.1016/j.cmet.2018.04.010
24. Tähkämö, L., Partonen, T., & Pesonen, A. K. (2018). Systematic review of light exposure impact on human circadian rhythm. *Chronobiology International, 36*(2), 151–170. https://doi.org/10.1080/07420528.2018.1527773
25. Wang, Y., Yu, L., Gao, Y., Jiang, L., Yuan, L., Wang, P., & Ding, G. (2021). Association between shift work or long working hours with metabolic syndrome: A systematic review and dose–response meta-analysis of observational studies. *Chronobiology International, 38*(3), 318–333. https://doi.org/10.1080/07420528.2020.1797763
26. Wong, P. M., Hasler, B. P., Kamarck, T. W., Muldoon, M. F., & Manuck, S. B. (2015). Social jetlag, chronotype, and cardiometabolic risk. *The Journal of Clinical Endocrinology & Metabolism, 100*(12), 4612–4620. https://doi.org/10.1210/jc.2015-2923