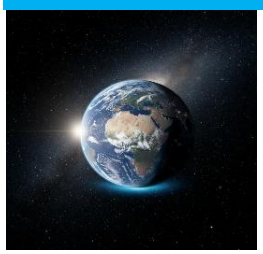
Quality in Sport. 2026;51:68635. eISSN 2450-3118.

https://doi.org/10.12775/QS.2026.51.68635



Quality in Sport. eISSN 2450-3118

Journal Home Page
https://apcz.umk.pl/QS/index

STASZKO, Natalia, BAŁA, Kamila, BISKUP, Alicja, SMAGOWSKA, Julia, ZBRONIEC, Jakub and BUKOWSKA, Małgorzata. Mechanisms linking blue light exposure, circadian misalignment and metabolic dysregulation in adolescents. Quality in Sport. 2026;51:68635. eISSN 2450-3118. https://doi.org/10.12775/QS.2026.51.68635

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 27.01.2026. Revised: 12.02.2026. Accepted: 12.02.2026. Published: 20.02.2026.

Mechanisms linking blue light exposure, circadian misalignment and metabolic dysregulation in adolescents

Authors:

Natalia Staszko

Wrocław Medical University; Wrocław, Poland

e-mail: natalia.staszko@student.umw.edu.pl

ORCID https://orcid.org/0009-0005-8335-5257

Kamila Bała

Wrocław Medical University; Wrocław, Poland

e-mail: kamila.bala@wp.pl

ORCID https://orcid.org/0009-0008-2621-7677

Alicja Biskup
Uniwersyteckie Centrum Stomatologii Śląskiego Uniwersytetu Medycznego w Katowicach sp. z o.o., Bytom
e-mail: alicja.b2104@gmail.com
ORCID https://orcid.org/0009-0001-2228-1478

Julia Smagowska
Uniwersyteckie Centrum Stomatologii Śląskiego Uniwersytetu Medycznego w Katowicach sp. z o.o., Bytom
e-mail: juliasmagowska1@gmail.com
ORCID https://orcid.org/0009-0003-4275-0846

Jakub Zbroniec
Wrocław Medical University; Wrocław, Poland
e-mail: jakub.zbroniec@student.umw.edu.pl
ORCID: https://orcid.org/0009-0000-2580-7626

Małgorzata Bukowska
Wrocław Medical University; Wrocław, Poland
e-mail: malgorzata.bukowska@student.umw.edu.pl
ORCID https://orcid.org/0009-0006-2117-3762

Abstract

Sleep disturbances and circadian rhythm disruption in children and adolescents represent a growing public health concern and are increasingly linked to the development of metabolic disorders. Adolescence is characterized by a physiological delay in chronotype which, when combined with social pressures and increasing evening exposure to blue light, promotes

desynchronization of the biological clock and the phenomenon of social jetlag. The aim of this paper was to discuss the mechanisms through which circadian rhythm disturbances and sleep deprivation affect glucose metabolism and the risk of insulin resistance in adolescents. Particular attention is given to the role of melatonin and its receptors, activation of the hypothalamic-pituitary-adrenal axis, chronic low-grade inflammation, dysregulation of clock gene expression, alterations in the gut-brain axis, reduced adiponectin levels, and decreased non-exercise activity thermogenesis (NEAT). The significance of chrononutrition and peripheral clock misalignment in the pathogenesis of metabolic jetlag is also addressed. The available evidence indicates that sleep and circadian rhythm disturbances, exacerbated by exposure to blue light, constitute an important and potentially modifiable risk factor for the development of insulin resistance, overweight and obesity during childhood and adolescence.

Keywords: circadian rhythm, blue light, sleep deprivation, circadian misalignment, insulin resistance

Introduction

The circadian rhythm is an endogenous biological cycle lasting approximately 24 hours that regulates most physiological and behavioral processes of the organism, including the sleep-wake cycle, body temperature, hormone secretion and metabolism. The central biological clock is located in the suprachiasmatic nucleus (SCN) of the hypothalamus and its proper functioning requires continuous synchronization with the external environment [1, 2]. Environmental cues that set the phase of the biological clock are referred to as zeitgebers [3], the most powerful of which is light, acting through the retinohypothalamic pathway [3]. Light signals reaching the SCN regulate the timing of melatonin secretion in the pineal gland, thereby synchronizing the sleep-wake cycle with the day-night cycle [3].

Light with a short wavelength in the blue spectrum (approximately 460-480 nm) is particularly important for the circadian system, as it most strongly suppresses nocturnal melatonin secretion [4-6]. Experimental studies have demonstrated that, at the same light intensity, blue light induces stronger suppression of melatonin secretion and greater phase shifts of the circadian rhythm than light of longer wavelengths [1, 6]. At the retinal level, a key role in this process is played by intrinsically photosensitive retinal ganglion cells (ipRGCs) containing melanopsin, which primarily mediate non-visual functions, including circadian rhythm regulation and melatonin secretion [1, 4].

Modern electronic devices such as smartphones, tablets, and laptops, emit substantial amounts of light within this wavelength range. Studies have shown that even several hours of evening exposure to blue light leads to a delay in circadian phase, a reduction in melatonin levels and a shift toward later sleep onset [1, 7]. Under conditions typical of electronic device use, as little as 2-3 hours of evening exposure to screen light can result in a significant delay in melatonin secretion and a shift of the circadian rhythm toward a later phase [4, 7]. The sensitivity of the circadian system to blue light also depends on prior exposure to daytime light - insufficient light exposure during the day may increase vulnerability to evening screen light and amplify its circadian-disrupting effects [6]. At the same time, recent years have seen a marked increase in screen time among children and adolescents. Most studies confirm an association between the use of electronic devices (smartphones, tablets, laptops), particularly in the evening, and later bedtimes as well as a reduction in total sleep duration [8]. Importantly, high levels of screen exposure are also often associated with a limitation of other health-promoting behaviors, such as physical activity and regular sleep hygiene practices [(9)].

Adolescence represents a particularly sensitive period from a chronobiological perspective. During puberty, a physiological delay of the circadian rhythm occurs, with a shift of melatonin secretion to later evening hours, which promotes later sleep onset and wake times [10]. This phenomenon is described as a ,,delayed adolescent chronotype" and is associated with a higher prevalence of disorders from the delayed sleep-wake phase disorder (DSWPD) spectrum among adolescents [10, 11]. Studies indicate that, in adolescents, the same dose of blue light induces stronger melatonin suppression and a greater phase shift of the circadian rhythm than in adults, further exacerbating the physiological circadian delay observed during pubertal development [6, 12, 13]. At the same time, modern school systems impose early start times, leading to a mismatch between adolescents' physiological sleep rhythms and actual daily schedules. This

discrepancy is referred to as ,,social jetlag," defined as the difference between sleep and wake times on school days versus free days. It is estimated that approximately half of adolescents experience clinically significant social jetlag, with differences of several hours between weekday and weekend sleep timing [14]. Evening exposure to blue light further intensifies circadian delay and exacerbates social jetlag. Evidence indicates that a later chronotype and greater social jetlag are associated with an unfavorable metabolic profile and weight gain [11, 15].

The COVID-19 pandemic further aggravated this problem. The introduction of remote learning, restrictions on out-of-home activities, and the shift of much of social life to the digital environment led to a significant increase in screen time, often extending into late night hours, resulting in reduced sleep duration and poorer sleep quality among adolescents [17].

Aim of the work

Circadian rhythm disturbances in adolescents have consequences that extend beyond sleep itself. An increasing body of evidence links these disturbances to adverse metabolic changes, including insulin resistance, obesity and metabolic syndrome [10]. In the context of growing exposure to blue light, understanding the mechanisms linking circadian rhythm disruption with metabolism becomes particularly important. The aim of this paper is to discuss these relationships, with special emphasis on the role of blue light in modulating circadian rhythms in adolescents.

Methods

The literature search strategy was conducted using the PubMed database based on a combination of keywords: circadian rhythm, blue light, sleep deprivation, circadian misalignment, insulin resistance. Additionally, references from selected publications were reviewed to identify related studies. After analyzing titles and abstracts, incomplete articles and those not directly related to the impact of circadian rhythm disturbances on metabolism in adolescents were excluded. The final analysis included 42 publications that met the inclusion criteria.

Literature review results

The impact of sleep and circadian rhythm disturbances on glucose homeostasis and metabolism

Desynchronization of the circadian rhythm resulting from evening exposure to blue light, irregular sleep schedules and so-called social jet lag leads in adolescents to a reduction in total sleep duration, deterioration of sleep quality, increased sleep fragmentation, and a decreased proportion of deep and rapid eye movement (REM) sleep [18]. Experimental models of circadian misalignment indicate that a shift of the biological rhythm relative to the day-night cycle, in itself, even when sleep duration is preserved, increases daytime sleepiness and impairs the restorative function of sleep [19, 20]. In adolescents, who physiologically exhibit a delayed chronotype, blue light further shifts melatonin secretion to later hours, promoting even later sleep onset and chronic sleep deprivation on school days [1, 12]. Numerous randomized controlled trials (RCTs) conducted both in adult populations [21, 22] and in children and adolescents [23] have demonstrated that experimental sleep restriction, even by only a few hours per day, leads to impaired insulin sensitivity, reflected by an increase in HOMA-IR (homeostatic model assessment for insulin resistance), a decrease in the Matsuda index or worsened results of the euglycemic clamp [21, 22, 23] and also promotes weight gain and obesity [24, 25]. Conversely, sleep extension in RCTs has been associated with improvement in these parameters [26]. As demonstrated in the study by Dutil et al., even modest but systematic sleep extension (+1 hour per night for one week) may reverse insulin resistance in high-risk adolescents [23].

Sleep and circadian rhythm disturbances lead to the development of insulin resistance through several coexisting mechanisms. Sleep restriction acts as a chronic stressor, resulting in activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system. This leads to increased levels of cortisol and catecholamines, enhanced hepatic gluconeogenesis and increased lipolysis in adipose tissue, resulting in greater release of free fatty acids (FFAs) [19]. Elevated FFA levels impair insulin signaling in skeletal muscle and the liver, thereby promoting the development of insulin resistance [19]. Concurrently, sleep deprivation and circadian rhythm desynchronization promote the development of chronic low-grade inflammation. These processes lead to increased concentrations of pro-inflammatory cytokines (IL-6, TNF-α) and C-reactive protein (CRP) [25, 27]. These cytokines disrupt insulin signaling, among others, through serine phosphorylation of IRS-1 (insulin receptor substrate 1) and activation of the JNK

(c-Jun N-terminal kinases)/NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells) pathways in adipose tissue, skeletal muscle and the liver. Study results indicate that circadian rhythm shifts exacerbate increases in inflammatory markers to a greater extent than sleep restriction alone [25, 27]. Sleep restriction also impairs the hormonal regulation of appetite, resulting in decreased leptin levels (the satiety hormone) and increased ghrelin levels (the hunger hormone), which translates into increased appetite, particularly for high-calorie foods rich in fat and simple sugars [28]. These changes promote a positive energy balance and increase the risk of obesity and insulin resistance [18, 27]. In recent years, the concept of chrononutrition has emerged, proposing that metabolic health depends not only on what we eat, but also on when we eat. The central clock in the SCN is primarily synchronized by light, whereas peripheral clocks in metabolic organs (the liver, pancreas, adipose tissue, and skeletal muscle) are largely synchronized by the timing of food intake [29]. Available studies show that consuming most daily calories in the morning and early part of the day, when insulin sensitivity and glucose tolerance are physiologically higher, is associated with a more favorable metabolic profile. In contrast, late eating and nighttime snacking, intensified by prolonged wakefulness due to evening blue light exposure, lead to so-called metabolic jet lag, a condition in which metabolic clocks are forced to process nutrients outside the physiologically optimal „window" of activity [29, 30]. This results in impaired glucose tolerance and higher postprandial insulinemia [29, 30]. Studies in pediatric populations have shown that a late distribution of energy intake correlates with a higher risk of insulin resistance (higher HOMA-IR) and an unfavorable lipid profile [31].

Melatonin has traditionally been associated with the regulation of sleep and circadian rhythms, however, an increasing body of evidence indicates that it also plays key metabolic and cytoprotective roles [32]. Melatonin receptors MT1 and MT2 are located not only in the central nervous system but also in numerous peripheral tissues, including the pancreas, liver, heart, adipose tissue and vascular walls [32, 33]. Through MT1 and MT2 receptors expressed on pancreatic β-cells, melatonin modulates the circadian pattern of insulin secretion. At night, it exerts an inhibitory effect on insulin release, which is consistent with the physiological reduction of food intake during darkness and constitutes a protective mechanism against nocturnal hypoglycemia [33]. Disturbances in the melatonin rhythm may lead to an inappropriate alignment of peak insulin secretion with meal timing [33]. Evening exposure to blue light shortens and flattens the nocturnal melatonin profile, resulting in desynchronization

of the pancreatic clock from the central SCN, impaired postprandial insulin responses and promotion of insulin resistance [33, 34]. Experimental studies also suggest that melatonin influences the expression of clock genes in pancreatic islets and the liver and, through its antioxidant and anti-inflammatory properties, may protect β-cells from damage in the course of diabetes [32].

At the molecular level, the circadian rhythm is generated by the cyclic expression of clock genes CLOCK, BMAL1, PER1-3, and CRY1-2 in SCN and in peripheral tissues [34]. Sleep deprivation and chronic circadian desynchronization lead to disruption of clock gene expression both in the SCN and in peripheral tissues, resulting in dysregulation of the transcription of numerous downstream genes involved in glucose and lipid metabolism as well as inflammatory processes [20, 35]. Because many enzymes critical for carbohydrate metabolism (e.g., hepatic kinases and glucose transporters) are under circadian control, alterations in clock gene expression may further exacerbate sleep-loss-induced insulin resistance [34].

The literature also highlights the involvement of the gut-brain axis as a potential intermediary mechanism linking sleep disturbances with the development of insulin resistance. Animal models have shown that chronic sleep fragmentation leads to alterations in gut microbiota composition, increased intestinal barrier permeability, endotoxemia and adipose tissue inflammation, thereby promoting insulin resistance [36]. In human studies, short-term sleep restriction has likewise been associated with changes in the gut microbiota profile and deterioration of insulin sensitivity indices [36].

Another potential mechanism linking sleep disturbances with insulin resistance involves adiponectin [37]. Adiponectin is an adipokine produced mainly by adipocytes, characterized by strong anti-inflammatory and insulin-sensitizing effects. Low adiponectin levels are consistently associated with insulin resistance, type 2 diabetes and metabolic syndrome in both cross-sectional and prospective studies [37]. Pediatric studies have demonstrated that sleep restriction and circadian rhythm disturbances reduce adiponectin levels and lead to increased insulin concentrations and higher HOMA-IR values [38]. Data from adult populations confirm that sleep restriction can lower adiponectin levels independently of body weight, suggesting that sleep is an independent modulator of this adipokine [39]. Mechanistically, reduced adiponectin levels following sleep restriction and circadian desynchronization may promote insulin resistance through:

- decreased activation of AMPK (5'AMP-activated protein kinase) and PPAR-α (peroxisome

proliferator-activated receptor alpha) in skeletal muscle and the liver, leading to reduced fatty acid oxidation, intracellular lipid accumulation and impaired insulin signaling [40]

- weaker inhibition of hepatic gluconeogenesis, resulting in increased hepatic glucose production and elevated fasting glycemia [40]
- loss of the anti-inflammatory effects of adiponectin, which enhances low-grade inflammation in adipose tissue and the endothelium and further worsens insulin sensitivity [40].

Sleep deficiency in children and adolescents leads to increased daytime sleepiness, fatigue, and reduced spontaneous physical activity, resulting in a decrease in non-exercise activity thermogenesis (NEAT), which encompasses all activity not related to sleep, eating or planned exercise [41]. Observational and experimental studies have shown that individuals with short sleep duration exhibit lower levels of spontaneous daytime activity and spend more time in sedentary behaviors, promoting a positive energy balance even without changes in caloric intake [42]. In adolescents, sleep deficiency is additionally associated with increased screen time and further reductions in NEAT. Decreased spontaneous physical activity promotes weight gain, increased visceral adipose tissue accumulation and secondary exacerbation of insulin resistance [42].

Sleep disturbances and circadian rhythm desynchronization, exacerbated by evening exposure to blue light, affect carbohydrate metabolism through multiple pathways: activation of the stress axis, chronic inflammation, disruption of hormonal appetite regulation, dysregulation of the pancreatic clock and clock genes, modification of the gut microbiota, reduction in adiponectin levels and decreased spontaneous physical activity (NEAT). The combined action of these mechanisms promotes the development of insulin resistance, overweight, and obesity in children and adolescents (Figure 1).

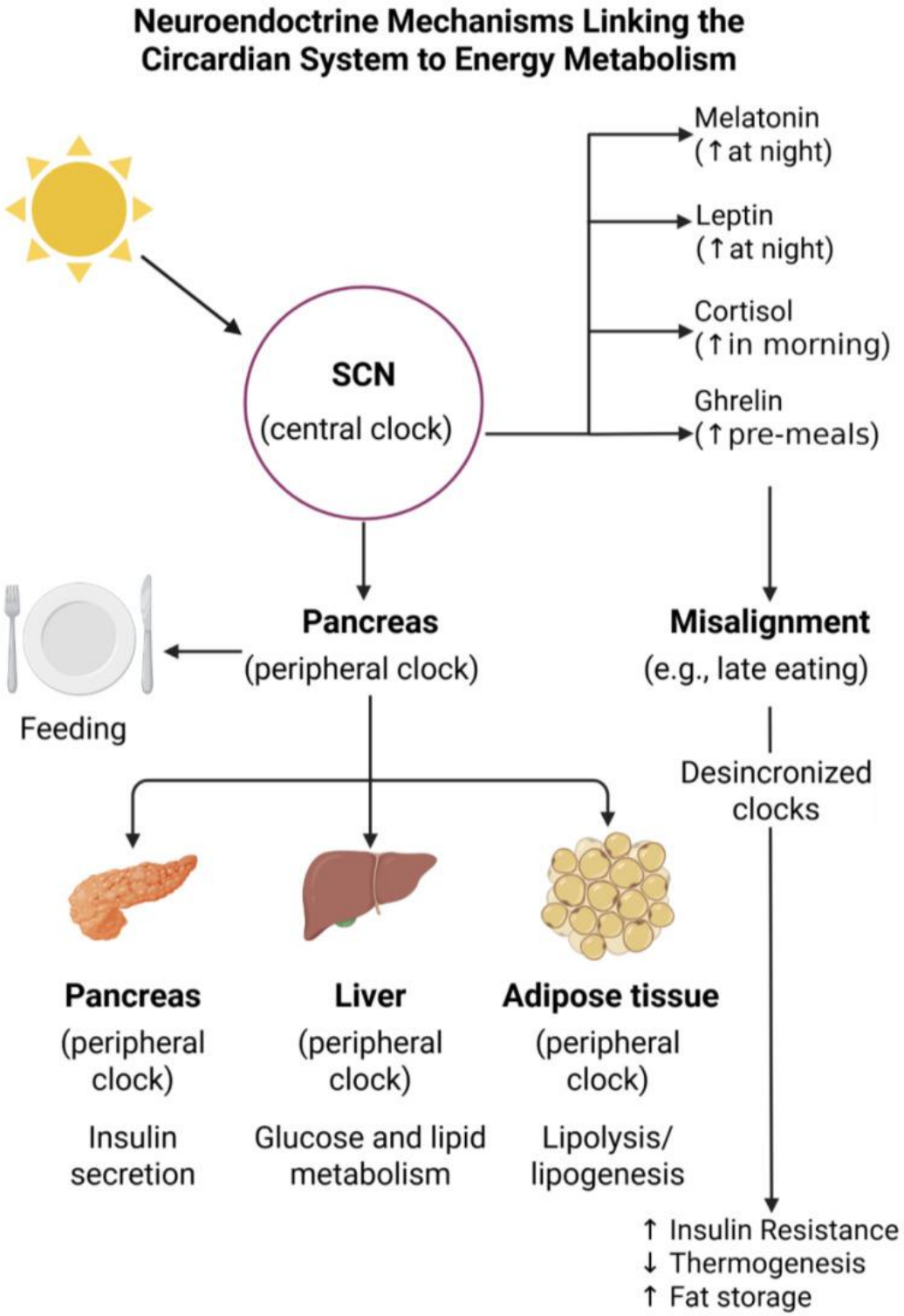


Figure 1.

Illustration of the bidirectional relationship between the circadian system and metabolic regulation. It emphasizes how feeding behavior and light exposure interact with the master clock in the SCN and peripheral clocks in metabolic tissues [32].

Conclusions

Sleep disturbances and circadian rhythm disruption in adolescents exert a significant, multidirectional impact on carbohydrate metabolism and overall metabolic health. The physiological delay of the adolescent chronotype, exacerbated by evening exposure to blue light and misalignment between the biological clock and social schedules, promotes chronic circadian desynchronization. This, in turn, leads to activation of the stress axis, low-grade chronic inflammation, disturbances in the hormonal regulation of appetite, dysregulation of clock gene expression, impaired synchronization of the pancreatic clock, alterations in the gut microbiota, and reduced adiponectin levels. In addition, sleep deprivation results in decreased spontaneous physical activity, favoring a positive energy balance. The combined effects of these mechanisms increase the risk of insulin resistance, overweight, and obesity in children and adolescents. Available evidence suggests that interventions aimed at improving circadian synchronization, including limiting evening exposure to blue light, optimizing sleep duration and timing, and incorporating principles of chrononutrition, may represent an important component of preventive and therapeutic strategies in this age group.

Disclosure

Author's Contribution

Conceptualization: Natalia Staszko, Kamila Bała, Alicja Biskup, Julia Smagowska, Jakub Zbroniec, Małgorzata Bukowska

Formal analysis: Natalia Staszko, Kamila Bała, Alicja Biskup, Julia Smagowska, Jakub Zbroniec, Małgorzata Bukowska

Investigation: Natalia Staszko, Kamila Bała, Alicja Biskup, Julia Smagowska, Jakub Zbroniec, Małgorzata Bukowska

Writing rough preparation: Natalia Staszko, Kamila Bała, Alicja Biskup, Julia Smagowska, Jakub Zbroniec, Małgorzata Bukowska

Writing review and editing: Natalia Staszko, Kamila Bała, Alicja Biskup, Julia Smagowska, Jakub Zbroniec, Małgorzata Bukowska

Supervision: Natalia Staszko, Kamila Bała, Alicja Biskup, Julia Smagowska, Jakub Zbroniec, Małgorzata Bukowska

All authors have read and agreed with the published version of the manuscript.

Funding Statement:

The Study Did Not Receive Special Funding.

Institutional Review Board Statement:

Not Applicable.

Informed Consent Statement:

Not Applicable.

Data Availability Statement:

Not Applicable.

Conflict Of Interest:

The authors declare no conflict of interest.

References:

1. Wahl S, Engelhardt M, Schaupp P, Lappe C, Ivanov IV. The inner clock-Blue light sets the human rhythm. J Biophotonics. 2019 Dec;12(12):e201900102.

2. de Toledo LHS, Moraes MN, Poletini M de O, Neto JC, Baron J, Mota T. Modeling the influence of nighttime light on melatonin suppression in humans: Milestones and perspectives. J Photochem Photobiol. 2023 Aug;16:100199.

3. Bautista J, Ojeda-Mosquera S, Ordóñez-Lozada D, López-Cortés A. Peripheral clocks and systemic zeitgeber interactions: from molecular mechanisms to circadian precision medicine. Front Endocrinol (Lausanne). 2025 May 29;16:1606242.

4. Alam M, Abbas K, Sharf Y, Khan S. Impacts of blue light exposure from electronic devices on circadian rhythm and sleep disruption in adolescent and young adult students. Chronobiol Med. 2024 Mar 31;6(1):10–4.

5. Do MTH, Yau K-W. Intrinsically photosensitive retinal ganglion cells. Physiol Rev. 2010 Oct;90(4):1547–81.

6. Nagare R, Rea MS, Plitnick B, Figueiro MG. Nocturnal melatonin suppression by adolescents and adults for different levels, spectra, and durations of light exposure. J Biol Rhythms. 2019 Apr;34(2):178–94.

7. Chang A-M, Aeschbach D, Duffy JF, Czeisler CA. Evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness. Proc Natl Acad Sci USA. 2015 Jan 27;112(4):1232–7.

8. Hale L, Kirschen GW, LeBourgeois MK, Gradisar M, Garrison MM, Montgomery-Downs H, et al. Youth Screen Media Habits and Sleep: Sleep-Friendly Screen Behavior Recommendations for Clinicians, Educators, and Parents. Child Adolesc Psychiatr Clin N Am. 2018 Apr;27(2):229–45.

9. Zablotsky B, Ng AE, Black LI, Haile G, Bose J, Jones JR, et al. Associations between screen time use and health outcomes among US teenagers. Prev Chronic Dis. 2025 Jul 10;22:E38.

10. Lang C, Richardson C, Micic G, Gradisar M. Understanding Sleep-Wake Behavior in Late Chronotype Adolescents: The Role of Circadian Phase, Sleep Timing, and Sleep Propensity. Front Psychiatry. 2022 Mar 11;13:785079.

11. Cespedes Feliciano EM, Rifas-Shiman SL, Quante M, Redline S, Oken E, Taveras EM. Chronotype, social jet lag, and cardiometabolic risk factors in early adolescence. JAMA Pediatr. 2019 Nov 1;173(11):1049–57.

12. Ricketts EJ, Joyce DS, Rissman AJ, Burgess HJ, Colwell CS, Lack LC, et al. Electric lighting, adolescent sleep and circadian outcomes, and recommendations for improving light health. Sleep Med Rev. 2022 Aug 12;64:101667.

13. Höhn C, Hahn MA, Gruber G, Pletzer B, Cajochen C, Hoedlmoser K. Effects of evening smartphone use on sleep and declarative memory consolidation in male adolescents and young adults. Brain Commun. 2024 May 17;6(3):fcae173.

14. Yang FN, Picchioni D, Duyn JH. Effects of sleep-corrected social jetlag on measures of mental health, cognitive ability, and brain functional connectivity in early adolescence. Sleep. 2023 Dec 11;46(12).

15. Jankovic N, Schmitting S, Krüger B, Nöthlings U, Buyken A, Alexy U. Changes in chronotype and social jetlag during adolescence and their association with concurrent changes

in BMI-SDS and body composition, in the DONALD Study. Eur J Clin Nutr. 2022 May;76(5):765–71.

16. Sun S, Yang Y, Yu F, He Y, Luo C, Zhang M, et al. Social jetlag and depressive symptoms among young people: a systematic review and meta-analysis. BMC Psychiatry. 2025 Jul 1;25(1):664.

17. Kiss O, Nagata JM, de Zambotti M, Dick AS, Marshall AT, Sowell ER, et al. Effects of the COVID-19 pandemic on screen time and sleep in early adolescents. Health Psychol. 2023 Dec;42(12):894–903.

18. Dutil C, Chaput JP. Inadequate sleep as a contributor to type 2 diabetes in children and adolescents. Nutr Diabetes. 2017 May 8;7(5):e266.

19. Leproult R, Holmbäck U, Van Cauter E. Circadian misalignment augments markers of insulin resistance and inflammation, independently of sleep loss. Diabetes. 2014 Jun;63(6):1860–9.

20. Potter GDM, Skene DJ, Arendt J, Cade JE, Grant PJ, Hardie LJ. Circadian rhythm and sleep disruption: causes, metabolic consequences, and countermeasures. Endocr Rev. 2016 Dec;37(6):584–608.

21. Zuraikat FM, Laferrère B, Cheng B, Scaccia SE, Cui Z, Aggarwal B, et al. Chronic insufficient sleep in women impairs insulin sensitivity independent of adiposity changes: results of a randomized trial. Diabetes Care. 2024 Jan 1;47(1):117–25.

22. Sondrup N, Termannsen A-D, Eriksen JN, Hjorth MF, Færch K, Klingenberg L, et al. Effects of sleep manipulation on markers of insulin sensitivity: A systematic review and meta-analysis of randomized controlled trials. Sleep Med Rev. 2022 Apr;62:101594.

23. Dutil C, Podinic I, Featherstone RB, Eaton A, Sadler CM, Goldfield GS, et al. Sleep and insulin sensitivity in adolescents at risk of type 2 diabetes: the Sleep Manipulation in Adolescents at Risk of Type 2 Diabetes randomized crossover study. Sleep. 2024 May 10;47(5).

24. Deng X, He M, He D, Zhu Y, Zhang Z, Niu W. Sleep duration and obesity in children and adolescents: evidence from an updated and dose-response meta-analysis. Sleep Med. 2021 Feb;78:169–81.

25. Chaput J-P, McHill AW, Cox RC, Broussard JL, Dutil C, da Costa BGG, et al. The role of insufficient sleep and circadian misalignment in obesity. Nat Rev Endocrinol. 2023 Feb;19(2):82–97.

26. Xu Y, Hua J, Wang J, Shen Y. Sleep duration is associated with metabolic syndrome in adolescents and children: a systematic review and meta-analysis. J Clin Sleep Med. 2023 Oct 1;19(10):1835–43.

27. Akhlaghi M, Kohanmoo A. Sleep deprivation in development of obesity, effects on appetite regulation, energy metabolism, and dietary choices. Nutr Res Rev. 2025 Jun;38(1):4–24.

28. Zhang N, Gao X, Li D, Xu L, Zhou G, Xu M, et al. Sleep deprivation-induced anxiety-like behaviors are associated with alterations in the gut microbiota and metabolites. Microbiol Spectr. 2024 Apr 2;12(4):e0143723.

29. Reytor-González C, Simancas-Racines D, Román-Galeano NM, Annunziata G, Galasso M, Zambrano-Villacres R, et al. Chrononutrition and energy balance: how meal timing and circadian rhythms shape weight regulation and metabolic health. Nutrients. 2025 Jun 27;17(13).

30. Vahlhaus J, Peters B, Hornemann S, Ost A-C, Kruse M, Busjahn A, et al. Later eating timing in relation to an individual internal clock is associated with lower insulin sensitivity and affected by genetic factors. EBioMedicine. 2025 Jun;116:105737.

31. Intemann T, Bogl LH, Hunsberger M, Lauria F, De Henauw S, Molnár D, et al. A Late Meal Timing Pattern Is Associated with Insulin Resistance in European Children and Adolescents. Pediatr Diabetes. 2024 Mar 1;2024:6623357.

32. Ahmad SB, Ali A, Bilal M, Rashid SM, Wani AB, Bhat RR, et al. Melatonin and health: insights of melatonin action, biological functions, and associated disorders. Cell Mol Neurobiol. 2023 Aug;43(6):2437–58.

33. Xia A-Y, Zhu H, Zhao Z-J, Liu H-Y, Wang P-H, Ji L-D, et al. Molecular mechanisms of the melatonin receptor pathway linking circadian rhythm to type 2 diabetes mellitus. Nutrients. 2023 Mar 15;15(6).

34. Hunter AL, Bechtold DA. The metabolic significance of peripheral tissue clocks. Commun Biol. 2025 Mar 26;8(1):497.

35. Morrison M, Halson SL, Weakley J, Hawley JA. Sleep, circadian biology and skeletal muscle interactions: Implications for metabolic health. Sleep Med Rev. 2022 Dec;66:101700.

36. Supasitdikul T, Mazariegos JRR, Nhat NN, Tung Y-T, Yang D-F, Lee L-J, et al. Sleep Deprivation Alters Gut Microbiome Diversity and Taxonomy: A Systematic Review and Meta-Analysis of Human and Rodent Studies. J Sleep Res. 2025 Jun 25;e70125.

37. Wang Y, Meng R-W, Kunutsor SK, Chowdhury R, Yuan J-M, Koh W-P, et al. Plasma adiponectin levels and type 2 diabetes risk: a nested case-control study in a Chinese population and an updated meta-analysis. Sci Rep. 2018 Jan 10;8(1):406.

38. Li L, Fu J, Yu XT, Li G, Xu L, Yin J, et al. Sleep Duration and Cardiometabolic Risk Among Chinese School-aged Children: Do Adipokines Play a Mediating Role? Sleep. 2017 May 1;40(5).

39. van Egmond LT, Meth EMS, Engström J, Ilemosoglou M, Keller JA, Vogel H, et al. Effects of acute sleep loss on leptin, ghrelin, and adiponectin in adults with healthy weight and obesity: A laboratory study. Obesity (Silver Spring). 2023 Mar;31(3):635–41.

40. Abou-Samra M, Selvais CM, Dubuisson N, Brichard SM. Adiponectin and its mimics on skeletal muscle: insulin sensitizers, fat burners, exercise mimickers, muscling pills … or everything together? Int J Mol Sci. 2020 Apr 9;21(7).

41. Van Dyk TR, Krietsch KN, Saelens BE, Whitacre C, McAlister S, Beebe DW. Inducing more sleep on school nights reduces sedentary behavior without affecting physical activity in short-sleeping adolescents. Sleep Med. 2018 Jul;47:7–10.

42. Chaput J-P, Dutil C. Lack of sleep as a contributor to obesity in adolescents: impacts on eating and activity behaviors. Int J Behav Nutr Phys Act. 2016 Sep 26;13(1):103.