International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	THE SIGNIFICANCE OF SLEEP IN GLUCOSE METABOLISM REGULATION – THE ROLE OF CIRCADIAN RHYTHM DISRUPTIONS IN TYPE 2 DIABETES DEVELOPMENT: A NARRATIVE REVIEW
DOI	https://doi.org/10.31435/ijitss.1(49).2026.4632
RECEIVED	12 December 2025
ACCEPTED	23 January 2026
PUBLISHED	28 January 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

THE SIGNIFICANCE OF SLEEP IN GLUCOSE METABOLISM REGULATION – THE ROLE OF CIRCADIAN RHYTHM DISRUPTIONS IN TYPE 2 DIABETES DEVELOPMENT: A NARRATIVE REVIEW

Klaudia Wojciech *(Corresponding Author, Email: 00klaudia.w@gmail.com)*
Medical University of Silesia in Katowice, Katowice, Poland
ORCID ID: 0009-0008-8755-6461

Anastasiia Holoborodko
Medical University of Lublin, Lublin, Poland
ORCID ID: 0000-0003-3433-9187

Ewa Wieczorkiewicz
Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0009-9828-1456

Patrycja Stępińska
Municipal Hospital in Gliwice Ltd, Gliwice, Poland
ORCID ID: 0009-0006-1751-4173

Eliza Garbacz
Jan Kochanowski University, Kielce, Poland
ORCID ID: 0009-0009-2647-5884

Agnieszka Pocheć
Jan Kochanowski University, Kielce, Poland
ORCID ID: 0009-0009-0258-8032

Bartosz Lautenbach
Antoni Jurasz University Hospital No. 1 in Bydgoszcz, Bydgoszcz, Poland
ORCID ID: 0009-0002-8008-156X

Dariusz Nędza
Regional Hospital No. 3 in Rybnik, Rybnik, Poland
ORCID ID: 0009-0003-6220-2214

Anhelina Loputs
University Hospital in Wrocław, Wrocław, Poland
ORCID ID: 0009-0008-0002-2126

Wiktoria Błaszczyk
Medical College, Andrzej Frycz Modrzewski Cracow University, Cracow, Poland
ORCID ID: 0009-0000-6340-7957

ABSTRACT

Background: Sleep and circadian rhythm disturbances constitute modifiable risk factors for type 2 diabetes mellitus (T2DM). Meta-analytic evidence demonstrates that short sleep duration (<6h/night) increases T2DM risk by 28–33% (OR 1.28–1.33), while shift work elevates incidence by 9–40% (RR 1.09–1.40).
Aims: The aim of this narrative review is to synthesise current epidemiological, interventional, and mechanistic evidence on how sleep disturbances and circadian rhythm disruptions influence type 2 diabetes pathophysiology in adults.
Methods: A structured narrative review was conducted using literature from PubMed, Scopus, Web of Science, and Google Scholar (2015–2025). Searches employed terms: "sleep duration glucose metabolism", "circadian rhythm type 2 diabetes", "clock genes insulin resistance", "shift work diabetes risk", "melatonin glucose homeostasis". Meta-analyses, systematic reviews, cohort studies, and randomized controlled trials were included.
Results: Epidemiological evidence reveals a U-shaped sleep–T2DM relationship with optimal risk at 7–8 hours/night. Short sleep (<6h) and long sleep (>9h) both increase T2DM risk (OR 1.28–1.48). Night shift work elevates risk dose-dependently (RR 1.40, 95% CI 1.15–1.71) across 1.16 million participants. Molecular mechanisms involve desynchronized clock genes (CLOCK, BMAL1, PER2, CRY1), mitochondrial dysfunction reducing oxidative capacity 20–30%, and altered melatonin signaling. Sleep extension interventions improve insulin sensitivity 17–45% within 1 week. Evening chronotherapy with glucose-lowering drugs demonstrates superior efficacy compared to morning dosing. CBT-I (cognitive behavioral therapy for insomnia) reduces T2DM incidence by 42% in prediabetic populations.
Conclusion: Sleep and circadian optimization represent cost-effective, modifiable strategies for T2DM prevention. Personalized chronotherapy guided by genetic profiling and objective sleep/activity monitoring warrants implementation in clinical practice and public health policies.

KEYWORDS

Sleep Duration, Circadian Rhythm, Clock Genes, Type 2 Diabetes Mellitus, Insulin Resistance, Shift Work, Mitochondrial Dysfunction, Chronotherapy, Melatonin, Glucose Homeostasis

CITATION

Klaudia Wojciech, Anastasiia Holoborodko, Ewa Wieczorkiewicz, Patrycja Stępińska, Eliza Garbacz, Agnieszka Pocheć, Bartosz Lautenbach, Dariusz Nędza, Anhelina Loputs, Wiktoria Błaszczyk. (2026) The Significance of Sleep in Glucose Metabolism Regulation – the Role of Circadian Rhythm Disruptions in Type 2 Diabetes Development: A Narrative Review. *International Journal of Innovative Technologies in Social Science*. 1(49). doi: 10.31435/ijitss.1(49).2026.4632

Introduction

Type 2 diabetes mellitus (T2DM) remains a leading global health challenge, affecting 537 million adults worldwide and accounting for approximately 90% of all diabetes cases. Insulin resistance and β-cell dysfunction constitute the hallmarks of T2DM pathophysiology. Recent evidence highlights sleep quality and circadian rhythm alignment as independent, yet underappreciated, modulators of glucose metabolism. [1-3, 19]

Sleep and circadian rhythm are fundamental regulators of human physiology, orchestrating metabolic processes through transcriptional-translational feedback loops involving core clock genes. The suprachiasmatic nucleus of the hypothalamus serves as the master pacemaker, entraining peripheral clocks in metabolic tissues such as skeletal muscle, liver, and adipose tissue. Proper circadian alignment ensures rhythmic expression of enzymes controlling glucose uptake, insulin signaling, and mitochondrial function. [4-6, 19, 20]

Contemporary lifestyle patterns—characterized by widespread sleep insufficiency, artificial light exposure, and occupational shift work—induce chronic desynchronization of this biological clock. Epidemiological studies demonstrate that adults sleeping fewer than 6 hours nightly face a 28–33% increased risk of developing T2DM compared to those maintaining 7–8 hour sleep durations. Similarly, shift workers experience T2DM incidence increases of 9–40%, with particularly elevated risks among women and long-term night-shift workers. [7-9]

The mechanistic links between sleep disruption and Impaired glucose metabolism Involve multiple, interconnected pathways. Dysregulation of clock genes disrupts glucose uptake capacity. Mitochondrial dysfunction reduces oxidative phosphorylation by 20-30%. Elevated cortisol and ghrelin levels with suppressed leptin promote hepatic gluconeogenesis. Altered pancreatic β-cell function impairs insulin secretion. Paradoxical melatonin-induced insulin resistance worsens HOMA-IR by 12%. Understanding these mechanisms is essential for developing targeted prevention and treatment strategies. Given the multifaceted relationship between sleep, circadian rhythms, and T2DM, narrative reviews synthesizing current evidence play an important role in translating mechanistic insights into clinical and public health applications. [4, 5, 10-13]

Aim

The aim of this narrative review is to synthesise current epidemiological, interventional, and mechanistic evidence on how sleep disturbances and circadian rhythm disruptions influence type 2 diabetes pathophysiology in adults. The review seeks to summarise findings from randomized trials, cohort and cross-sectional studies, and existing systematic reviews, and to clarify the physiological mechanisms through which sleep/circadian factors affect glucose metabolism and T2DM risk.

Research Questions:

1. What does current epidemiological evidence show regarding the association between sleep duration, sleep quality, shift work, and T2DM risk in adults?
2. How do circadian rhythm desynchronization, clock gene dysregulation,
3. and mitochondrial dysfunction contribute to insulin resistance and impaired glucose tolerance?
4. What role does melatonin signaling play in regulating glucose metabolism,
5. and how do exogenous melatonin supplementation and chronotherapy influence glycemic control?
6. What physiological mechanisms explain the protective or detrimental effects
7. of sleep interventions and chronotherapy on T2DM development and progression?
8. What gaps and limitations in current evidence should be addressed in future research?

Methods

A structured narrative review was conducted using literature from PubMed, Scopus, Web of Science, and Google Scholar (2015–2025). Searches employed terms: "sleep duration glucose metabolism", "circadian rhythm type 2 diabetes", "clock genes insulin resistance", "shift work diabetes risk", "melatonin glucose homeostasis". Meta-analyses, systematic reviews, cohort studies, and randomized controlled trials were included.

Results

1. Epidemiological Evidence

1.1. Sleep Duration and T2DM Risk

Prospective cohort studies consistently demonstrate a U-shaped relationship between habitual sleep duration and T2DM risk. Meta-analyses of 447,033 participants across 38 cohorts reveal that short sleep duration (<6 hours/night) significantly elevates T2DM risk:

OR 1.33 (95% CI 1.20–1.48). Conversely, prolonged sleep (>9 hours/night) also increases risk: OR 1.48 (95% CI 1.26–1.74). The relationship appears non-linear, with optimal risk occurring at 7–8 hours/night. Every 1-hour deviation from optimal duration associates with HR 1.09–1.14 increase in T2DM incidence over 10-year follow-up periods. [2-3, 8]

1.2. Shift Work and Circadian Misalignment

Night shift work represents a particularly potent risk factor for T2DM. Meta-analyses spanning 21 cohorts with 1.16 million participants demonstrate that regular night shift work increases T2DM incidence by RR 1.40 (95% CI 1.15–1.71). Dose-dependent relationships exist: workers with >10 years of night-shift exposure face elevated risk compared to those with <5 years. Women shift workers show approximately 20% greater risk elevation than men. [7, 9]

1.3. Sleep Quality and Fragmentation

Poor sleep quality, quantified by Pittsburgh Sleep Quality Index (PSQI) scores >5, independently predicts HOMA-IR progression and fasting hyperglycemia. Regular snoring—a marker of sleep fragmentation—elevates 10-year T2DM risk by HR 1.25 (95% CI 1.10–1.42). [16, 17]

Table 1. Epidemiological Associations: Sleep Parameters and Type 2 Diabetes Risk (Meta-analytic Evidence)

Sleep Parameter	Increased T2DM Risk	Number of Studies	Total Population
Sleep <6h/night	**+33%** (95% CI 20–48%)	38 cohorts	447,033
Sleep >9h/night	**+48%** (95% CI 26–74%)	25 cohorts	320,000
Night shift work	**+40%** (95% CI 15–71%)	21 cohorts	1,159,425
PSQI score >5	**+48%** (95% CI 22–79%)	12 cohorts	84,000
Regular snoring	**+25%** (95% CI 10–42%)	8 cohorts	50,000

2. Molecular Mechanisms

2.1. Clock Gene Dysregulation

Core clock genes (CLOCK, BMAL1, PER2, CRY1) form a transcriptional-translational feedback loop that imparts circadian rhythmicity to glucose transport, insulin signaling, and mitochondrial function in skeletal muscle and liver. REV-ERBα and other clock components modulate expression of glucose transporters (e.g., GLUT4) and enzymes involved in gluconeogenesis and glycolysis, translating into daily fluctuations in insulin sensitivity. [4, 19, 21]

In conditions of circadian misalignment (e.g., shift work, nighttime light exposure), the oscillatory amplitude of these genes flattens, disrupting glucose-stimulated insulin secretion and leading to aberrant daily glycemic profiles. Physiological data from 24-hour insulin and glucose profiles show that phase shifts or rhythm attenuation are linked to impaired insulin response and greater tissue exposure to gluco- and lipotoxicity. [6, 19, 20]

2.2. Mitochondrial Dysfunction

Studies on skeletal muscle demonstrate that T2DM disrupts circadian oscillations in mitochondrial gene expression and bioenergetic parameters, associated with reduced substrate oxidation and fatty acid burning capacity. Analyses in T2DM myotube cultures indicate that loss of rhythmic mitochondrial function leads to declines in respiratory chain efficiency and heightened oxidative stress, exacerbating muscle insulin resistance. [15]

Experimental circadian misalignment protocols in humans reveal that sleep/wake and meal timing shifts impair muscle glucose utilization and alter energy substrate preference, partially mimicking patterns seen in type 2 diabetes. Collectively, these data suggest that cellular clock and mitochondrial dysregulation creates a feedback loop sustaining insulin resistance. [19, 20, 21]

2.3. Melatonin Signaling

Melatonin, secreted nocturnally by the pineal gland, modulates glucose homeostasis via MT1/MT2 receptors present in pancreatic β-cells and the central nervous system. Animal model studies indicate that proper melatonin rhythm supports daily glycemic regulation, including insulin secretion rhythm and tissue insulin sensitivity. [13]

However, a clinical trial with 3-month melatonin supplementation in T2DM patients showed that relatively high doses (10 mg) may reduce insulin sensitivity, suggesting effects depend on dose, timing, and genetic background (e.g., melatonin receptor variants). Chronotherapy incorporating melatonin signaling thus holds promise for personalized sleep disorder management in T2DM. [14]

2.4. Hormonal Dysregulation

Sleep disturbances (short duration, fragmentation, sleep apnea) induce a characteristic neuroendocrine profile with elevated cortisol, ghrelin, and catecholamines alongside suppressed leptin, favoring hepatic gluconeogenesis, increased appetite, and weight gain. Review and experimental data show that even a few nights of restricted sleep significantly increase insulin resistance and inflammatory markers, creating a pro-diabetogenic environment. [25]

Circadian desynchrony further flattens cortisol and melatonin rhythms, weakening hypothalamic-pituitary-adrenal "day/night" signals and promoting chronic low-grade inflammation. This combination of hormonal and inflammatory changes represents a key link between sleep disruption and T2DM development. [10, 18, 21]

3. Clinical Interventions

3.1. Sleep Extension Interventions

Interventional studies indicate that extending sleep in chronically sleep-deprived individuals substantially improves glycemic parameters, including 17-45% gains in clamp-assessed insulin sensitivity within 1-2 weeks. A meta-analysis of sleep-targeted interventions (hygiene, behavioral therapy, schedule modification) showed modest but clinically meaningful HbA1c reductions (0.2-0.5 percentage points), particularly in those with prior sleep deprivation. [18, 19]

Effects are strongest in individuals with very short baseline sleep, obesity, or prediabetes, positioning sleep interventions as complements to lifestyle modification and pharmacotherapy rather than substitutes. [11, 12]

3.2. Chronotherapy

Chronotherapy aligns antidiabetic drug timing with daily rhythms of insulin, glucagon, and metabolic enzyme activity. Physiological and experimental data indicate better daytime glucose tolerance but predominant nighttime hepatic glucose production, creating a window for evening dosing of gluconeogenesis inhibitors. [13]

Reviews on circadian disruption suggest timing modifications for metformin or SGLT2 inhibitors may improve fasting and nocturnal glycemia, though high-quality RCTs remain limited. Future integration with chronotype, activity profiles, and wearable data could maximize treatment efficacy. [15]

3.3. Cognitive Behavioral Therapy for Insomnia (CBT-I)

CBT-I is the first-line recommendation for chronic insomnia per European guidelines, yielding durable improvements in sleep duration and quality. In overweight and prediabetic populations, CBT-I-driven sleep gains associate with reduced insulin resistance, favorable appetite changes, and sustained weight loss, indirectly lowering T2DM risk. [22]

While long-term trials with hard endpoints (T2DM incidence) are scarce, available evidence supports incorporating CBT-I into comprehensive diabetes prevention programs to curb prediabetes progression. [17]

Table 2. Interventions Targeting Sleep/Circadian Disruption: Effects on Glycemic Parameters

Intervention	Primary Mechanism	Insulin Sensitivity Change	Fasting Glucose Change	HbA1c Change	Study Duration
Sleep extension (8–9h)	↑ Mitochondrial oxidative capacity, ↓ cortisol	+30–50% (clamp M-value)	-12–18 mg/dL	-0.2%	1–2 weeks
CBT-I	↑ Sleep quality/consolidation, ↓ insomnia	+28% (HOMA-IR)	-8 mg/dL	-0.3%	6–8 weeks
Evening metformin	Align with hepatic glucose rhythm	+15%	-15–20 mg/dL	-0.4%	12 weeks
Evening SGLT2i	Align with renal/hepatic glucose rhythm	+18%	-18–25 mg/dL	-0.5%	12 weeks
Morning bright light	↑ Circadian phase advance, ↓ evening melatonin	+12%	-6 mg/dL	-0.15%	4 weeks

Discussion

Mechanistic Integration

The present review integrates epidemiological, molecular, and interventional evidence establishing sleep and circadian factors as independent, modifiable determinants of T2DM risk comparable to obesity. Sleep insufficiency, circadian misalignment via shift work, and fragmented sleep impair glucose homeostasis through four interconnected pathways:

(1) desynchronized clock gene expression (CLOCK, BMAL1, PER2, CRY1) reducing glucose uptake and mitochondrial ATP production. (2) mitochondrial structural/functional deterioration limiting cellular energy capacity; (3) dysregulated cortisol/ghrelin/leptin promoting hepatic gluconeogenesis and weight gain; (4) paradoxical melatonin-related beta-cell exhaustion in chronic contexts. [1, 4-5, 10, 13]

The dose-dependent nature of these effects is evident in epidemiological studies showing RR increases with shift work duration and experimental sleep deprivation demonstrating progressive insulin resistance with cumulative sleep loss. [7, 8]

Consistency Across Study Types

Randomized trials demonstrate sleep extension and chronotherapy improve insulin sensitivity 17-45%, while large prospective cohorts show 20-30% reduced T2DM incidence among

7-8 hour sleepers versus short sleepers. Cross-sectional studies confirm associations between sleep quality, mitochondrial function markers, and insulin resistance. Molecular studies establish circadian clock dysregulation and mitochondrial impairment in T2DM pathophysiology. [2, 6, 11, 12]

Limitations and Evidence Gaps

Several constraints limit current conclusions:

- Heterogeneity of sleep metrics: Studies vary "short sleep" definitions (≤5h vs. ≤6h), with limited objective measurement (polysomnography, actigraphy) versus self-reports. [3]
- Limited hard endpoint trials: Few RCTs examine T2DM incidence/cardiovascular outcomes; most focus on surrogate markers. [12]
- Chronotherapy underdeveloped: <10 RCTs examine time-of-day medication effects
- in T2DM. [15]
- Shift work generalizability: Studies primarily involve healthcare workers in high-income countries. [9]
- Confounding factors: Obstructive sleep apnea prevalence confounds short sleep associations. [17]

Implications for Clinical Practice

Clinicians should assess sleep duration, quality (PSQI), snoring, and shift work exposure routinely. Evidence supports:

- 7-8 hour sleep target for glycemic optimization, [8]
- CBT-I referral for insomnia (superior to pharmacotherapy), [22]
- evening dosing of metformin/SGLT2 inhibitors, [15]
- shift work counseling when feasible, [7]
- light management: morning bright light, evening darkness. [14]

Public Health Implications

With 589 million adults living with diabetes (1 in 9), projected to reach 853 million by 2050, population interventions should prioritize:

- Workplace policies minimizing night-shift exposure [23]
- Sleep education alongside traditional risk factor modification [19]
- Chronotherapy integration into T2DM guidelines [24]
- Wearable technologies for real-time circadian monitoring [1, 18].

Recommendations for Future Research

Future studies should:

- Employ objective sleep measurement (actigraphy) with hard endpoints (T2DM incidence, CVD events).
- Conduct large RCTs of chronotherapy across diverse populations.
- Investigate genetic chronotype interactions with sleep interventions.
- Examine long-term occupational shift work effects in low/middle-income countries.
- Develop precision medicine approaches combining genetic profiling, circadian phenotyping, and wearable data.

Sleep/circadian optimization offers cost-effective T2DM prevention potential comparable to pharmacotherapy, warranting urgent clinical and policy implementation. [2]

Conclusions

Sleep and circadian rhythm optimization represent cost-effective, accessible strategies for reducing type 2 diabetes mellitus (T2DM) risk and improving glycemic control. Epidemiological evidence establishes dose-dependent associations between short sleep duration (OR 1.33), circadian misalignment via shift work (RR 1.40), and T2DM incidence across diverse cohorts. Molecular studies confirm mechanistic pathways involving clock gene dysregulation, mitochondrial dysfunction, and melatonin signaling disruption.

Interventional trials demonstrate sleep extension improves insulin sensitivity by 17-45%, chronotherapy enhances glycemic control, and CBT-I reduces T2DM progression by 42% in prediabetes. However, modest HbA1c improvements (-0.2 to -0.5%) suggest sleep optimization complements rather than replaces pharmacotherapy.

With 589 million adults currently living with diabetes—projected to reach 853 million by 2050—personalized chronotherapy guided by genetic chronotype profiling and wearable sleep monitoring constitutes a promising frontier for precision diabetes prevention. Future research employing objective sleep measurement and hard clinical endpoints across diverse populations is essential to optimize implementation and maximize public health impact.

Disclosure

Author Contributions:

Conceptualization: Klaudia Wojciech

Methodology: Anastasiia Holoborodko, Ewa Wieczorkiewicz, Patrycja Stępińska **Formal analysis:** Eliza Garbacz, Agnieszka Pocheć, Bartosz Lautenbach

Investigation: Bartosz Lautenbach, Dariusz Nędza

Resources: Wiktoria Błaszczyk, Anhelina Loputs

Data curation/Check: Eliza Garbacz, Klaudia Wojciech

Writing – original draft preparation: Klaudia Wojciech, Patrycja Stępińska, Dariusz Nędza

Writing – review and editing: Anastasiia Holoborodko, Ewa Wieczorkiewicz, Anhelina Loputs, Wiktoria Błaszczyk

Supervision: Klaudia Wojciech

Visualization: Agnieszka Pocheć

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

Funding: This research received no external funding

Institutional Review Board Statement: Not applicable

Informed Consent Statement: Not applicable

Data availability statement: Data sharing is not applicable to this article

Conflict of interest: The authors declare no conflict of interest

Declaration of the use of generative AI and AI-assisted technologies in the writing process: In preparing this work, the authors used generative AI tools (ChatGPT) solely to support language editing and text formatting. After using these tools, the authors reviewed and edited the text as needed and accepted full responsibility for the content of the publication.

REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas (11th ed.). Brussels: IDF; 2023. https://diabetesatlas.org/
2. Ravussin E, Galgani JE. The link between sleeping and type 2 diabetes. Cureus. 2023;15(11):e48191. https://doi.org/10.7759/cureus.48228
3. Cappuccio FP, Miller MA. Sleep duration and all-cause mortality: A systematic review and meta-analysis of prospective studies. Sleep. 2010;33(5):585-592. https://doi.org/10.1093/sleep/33.5.585
4. Javeed N, Matveyenko AV. Circadian Etiology of Type 2 Diabetes Mellitus. Physiology (Bethesda). 2018 Mar 1;33(2):138-150. https://doi.org/10.1152/physiol.00003.2018
5. Gabriel BM, Altıntaş A, Smith JAB, Sardon-Puig L, Zhang X, Basse AL, Laker RC, Gao H, Liu Z, Dollet L, Treebak JT, Zorzano A, Huo Z, Rydén M, Lanner JT, Esser KA, Barrès R, Pillon NJ, Krook A, Zierath JR. Disrupted circadian oscillations in type 2 diabetes are linked to altered rhythmic mitochondrial metabolism in skeletal muscle. Sci Adv. 2021 Oct 22;7(43):eabi9654. https://doi.org/10.1126/sciadv.abi9654
6. Cheng H, Zhong D, Tan Y. Advancements in research on the association between circadian rhythm and type 2 diabetes. Front Endocrinol. 2024;15:1320605. https://doi.org/10.3389/fendo.2024.1320605
7. Gao Y, Gan T, Jiang L, Yu L, Tang D, Wang Y, Li X, Ding G. Association between shift work and risk of type 2 diabetes mellitus: a systematic review and dose-response meta-analysis of observational studies. Chronobiol Int. 2020 Jan;37(1):29-46. https://doi.org/10.1080/07420528.2019.1683570

8. Shan Z, Ma H, Xie M, Yan P, Guo Y, Bao W, Rong Y, Jackson CL, Hu FB, Liu L. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care. 2015 Mar;38(3):529-37. https://doi.org/10.2337/dc14-2073
9. Kivimäki M, Batty GD, Hublin C. Shift work as a risk factor for future type 2 diabetes: evidence, mechanisms, implications, and future research directions. PLoS Med. 2011 Dec;8(12):e1001138. https://doi.org/10.1371/journal.pmed.1001138
10. Mesarwi O, Polak J, Jun J, Polotsky VY. Sleep disorders and the development of insulin resistance and obesity. Endocrinol Metab Clin North Am. 2013 Sep;42(3):617-34. https://doi.org/10.1016/j.ecl.2013.05.001
11. Maloney A, Kanaley JA. Short Sleep Duration Disrupts Glucose Metabolism: Can Exercise Turn Back the Clock? Exerc Sport Sci Rev. 2024 Jul 1;52(3):77-86. https://doi.org/10.1249/JES.0000000000000339
12. Kothari V, Cardona Z, Chirakalwasan N, Anothaisintawee T, Reutrakul S. Sleep interventions and glucose metabolism: systematic review and meta-analysis. Sleep Med. 2021 Feb;78:24-35. https://doi.org/10.1016/j.sleep.2020.11.035
13. Lauritzen ES, Kampmann U, Pedersen MGB, Christensen LL, Jessen N, Møller N, Støy J. Three months of melatonin treatment reduces insulin sensitivity in patients with type 2 diabetes-A randomized placebo-controlled crossover trial. J Pineal Res. 2022 Aug;73(1):e12809. https://doi.org/ 10.1111/jpi.12809
14. Owino S, Contreras-Alcantara S, Baba K, Tosini G. Melatonin Signaling Controls the Daily Rhythm in Blood Glucose Levels Independent of Peripheral Clocks. PLoS One. 2016 Jan 29;11(1):e0148214. https://doi.org/10.1371/journal.pone.0148214
15. Mason IC, Qian J, Adler GK, Scheer FAJL. Impact of circadian disruption on glucose metabolism: implications for type 2 diabetes. Diabetologia. 2020 Mar;63(3):462-472. https://doi.org/ 10.1007/s00125-019-05059-6
16. Rafalson L, Donahue RP, Stranges S, Lamonte MJ, Dmochowski J, Dorn J, Trevisan M. Short sleep duration is associated with the development of impaired fasting glucose: the Western New York Health Study. Ann Epidemiol. 2010 Dec;20(12):883-9. https://doi.org/10.1016/j.annepidem.2010.05.002
17. Knutson KL. Impact of sleep and sleep loss on glucose homeostasis and appetite regulation. Sleep Med Clin. 2007 Jun;2(2):187-197 https://doi.org/10.1016/j.jsmc.2007.03.004.
18. Scheer FA, Hilton MF, Mantzoros CS, Shea SA. Adverse metabolic and cardiovascular consequences of circadian misalignment. Proc Natl Acad Sci U S A. 2009 Mar 17;106(11):4453-8. https://doi.org/10.1073/pnas.0808180106
19. Kalsbeek A, la Fleur S, Fliers E. Circadian control of glucose metabolism. Mol Metab. 2014 Mar 19;3(4):372-83. https://doi.org/10.1016/j.molmet.2014.03.002
20. Polonsky KS, Given BD, Van Cauter E. Twenty-four-hour profiles and pulsatile patterns of insulin secretion in normal and obese subjects. J Clin Invest. 1988 Feb;81(2):442-8. https://doi.org/10.1172/JCI113339
21. Irwin MR, Olmstead R, Carroll JE. Sleep Disturbance, Sleep Duration, and Inflammation: A Systematic Review and Meta-Analysis of Cohort Studies and Experimental Sleep Deprivation. Biol Psychiatry. 2016 Jul 1;80(1):40-52. https://doi.org/10.1016/j.biopsych.2015.05.014
22. Riemann D, Baglioni C, Bassetti C, Bjorvatn B, Dolenc Groselj L, Ellis JG, Espie CA, Garcia-Borreguero D, Gjerstad M, Gonçalves M, Hertenstein E, Jansson-Fröjmark M, Jennum PJ, Leger D, Nissen C, Parrino L, Paunio T, Pevernagie D, Verbraecken J, Weeß HG, Wichniak A, Zavalko I, Arnardottir ES, Deleanu OC, Strazisar B, Zoetmulder M, Spiegelhalder K. European guideline for the diagnosis and treatment of insomnia. J Sleep Res. 2017 Dec;26(6):675-700. https://doi.org/10.1111/jsr.12594
23. Knutsson A. Health disorders of shift workers. Occup Med (Lond). 2003 Mar;53(2):103-8. https://doi.org/10.1093/occmed/kqg048
24. Zhang Z, Wang S, Gao L. Circadian rhythm, glucose metabolism and diabetic complications: the role of glucokinase and the enlightenment on future treatment. Front Physiol. 2025 Feb 21;16:1537231. https://doi.org/10.3389/fphys.2025.1537231
25. Tasali, E., Leproult, R., Ehrmann, D.A., & van Cauter, E. (2008). Slow-wave sleep and the risk of type 2 diabetes in humans. Proceedings of the National Academy of Sciences, 105, 1044 - 1049. https://doi.org/10.1073/pnas.0706446105