Quality in Sport. 2026;51:68214. eISSN 2450-3118.

https://doi.org/10.12775/QS.2026.51.68214

Quality in Sport. eISSN 2450-3118

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

BRZOZA, Klaudia, MATUSIAK, Filip, KOCIŃSKI, Michał, IGLEWSKI, Patryk, PIETRASZ, Michał and KOMARCZEWSKA, Anna. Social Jetlag as an Independent Risk Factor for Cardiometabolic Diseases: The Role of Circadian Rhythm Irregularity. Quality in Sport. 2026;51:68214. eISSN 2450-3118. https://doi.org/10.12775/QS.2026.51.68214

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: 13.01.2026. Revised: 01.02.2026. Accepted: 01.02.2026. Published: 10.02.2026.

Social Jetlag as an Independent Risk Factor for Cardiometabolic Diseases: The Role of Circadian Rhythm Irregularity

AUTHORS

Klaudia Brzoza, ORCID: https://orcid.org/0009-0006-0950-4514

E-mail: klaudiabrzoza99@gmail.com

Jan Biziel University Hospital No. 2 in Bydgoszcz: Bydgoszcz, Kujawsko-Pomorskie, PL

Filip Matusiak, ORCID: https://orcid.org/0009-0002-0538-6443

E-mail: f.matusiak98@gmail.com

Jan Biziel University Hospital No. 2 in Bydgoszcz: Bydgoszcz, Kujawsko-Pomorskie, PL

Michał Kociński, ORCID: https://orcid.org/0009-0007-7651-7929
E-mail: michal.kocinski1999@gmail.com
Jan Biziel University Hospital No. 2 in Bydgoszcz: Bydgoszcz, Kujawsko-Pomorskie, PL

Patryk Iglewski, ORCID: https://orcid.org/0009-0004-6611-2168
E-mail: patryk.iglewski01@gmail.com
Ludwik Rydygier Provincial Combined Hospital in Torun: Torun, Kujawsko-Pomorskie, PL

Michał Pietrasz, ORCID: https://orcid.org/0009-0000-8148-7487
E-mail: michal.pietrasz252@gmail.com
Ludwik Rydygier Provincial Combined Hospital in Torun: Torun, Kujawsko-Pomorskie, PL

Anna Komarczewska, ORCID: https://orcid.org/0009-0006-7378-2607
E-mail: lek.komarczewska@wp.pl
Ludwik Rydygier Provincial Combined Hospital in Torun: Torun, Kujawsko-Pomorskie, PL

Corresponding author:
Klaudia Brzoza
E-mail: klaudiabrzoza99@gmail.com

Abstract

Background. Social jetlag (SJL) – misalignment of biological and social time – is a common experience in modern societies. Although weekend catch-up sleep (WCS) is a popular strategy for adapting to chronic insufficient sleep, its efficacy in recovering from accumulated sleep debt is uncertain.

Aim. This review examines the health consequences of sleep variability and the physiological effectiveness of catching up on lost weekend sleep.

Results. SJL causes internal desynchrony and results in marked metabolic, cardiovascular, and psychological derangements. Key findings included increased body weight, insulin resistance

and cardiovascular mortality. Research shows a “dual hormonal signature” of impaired melatonin and cortisol rhythms. Interestingly, although moderate WCS (1–2 hours) may result in modest metabolic benefits, excessive compensation (≥2 hours) aggravates the circadian misalignment and is a powerful predictor of depressive symptoms.

Conclusion. The idea of "repaying" lost sleep is a myth. Weekend payback can't make up for the harm of social jetlag. Attention should be paid to rhythm hygiene rather than total sleep duration in clinical practice, for long-lasting physiological homeostasis.

Keywords: social jetlag, sleep debt, weekend catch-up sleep, metabolic health, rhythm hygiene.

1. Introduction

Artificial light at night (ALAN), a worldwide problem in the contemporary environment, has changed the natural darkness level and can impact the performance of living organisms [1]. Combined with the demands of shift work and non-standard daily schedules, this leads to abrupt changes in the timing of light exposure and sleep. This leads to disruption of the endogenous circadian system and internal desynchronization, which is defined as a phase dissociation between the central circadian pacemaker located in the suprachiasmatic nucleus (SCN) and peripheral clocks [2]. A reflection of this way of life, in which our "sleep debt" – the difference between the number of hours we need (biologically) and the number of hours that so many Americans actually get – has never been greater. Ongoing sleep loss is linked to impaired cognitive performance, increased subjective sleepiness, diminished mood and elevated risk for incidents [3]. In addition, there is evidence suggesting that sleep patterns in terms of quantity (duration), quality (continuity) and timing have been strongly related to all-cause mortality, including cardiovascular [4]. Social jetlag is the term researchers use to define the gap between biologically and socially acceptable sleep timings. This is the mismatch between social and biological time which is measured as the difference of the mid-sleep time on free days and

workdays. It is mainly measured by questionnaires, like the Munich ChronoType Questionnaire (MCTQ) [5]. One of the most popular psychological methods of coping with sleep loss is "weekend catch-up sleep" (WCS). There is evidence in support of the hypothesis that moderate WCS (1–2 hours) is associated with a lower prevalence of depression [6]. However, the scientific literature is inconclusive with regard to recovery sleep dynamics. It is not known whether and/or to what extent chronic sleep loss-induced physiological and behavioral disruption can be fully restored by extra sleep [3].

The objective of this article was to examine the current literature on health outcomes associated with sleep timing variability and if weekend compensation is an effective strategy in reducing sleep debt.

2. Biological Mechanisms: Circadian Rhythm and Lifestyle

In order to elucidate health effects of sleep variability, the physiology behind the timing of our biological processes must first be considered. The mismatch between one's internal timing and the schedule of social time disrupts a cascade of body systems.

2.1. The Master Clock and Circadian Entrainment

Bará and Falchi describe the regulation of the timing of sleep and its opposites by an endogenous circadian system. In the core of this hierarchy is the suprachiasmatic nucleus (SCN) of the hypothalamus, which acts as the "master clock" or central pacemaker. This central pacemaker is constituted of an endogenous clock capable to produce circadian rhythms and entrain them to the external 24 h day. The light-dark cycle is the major environmental stimulus to entrain the SCN. Non-visual information that entrains the circadian clock is present in the light. In our current environment, however, this natural rhythm has been disrupted due to the ubiquitous use of artificial light at night (ALAN). Exposure to this sudden change in light results in the disruption of the endogenous circadian system and a situation of internal desynchronization [1,2].

2.2. Internal Desynchronization and Misalignment

Boivin et al. show that under normal conditions, the SCN coordinates the timing of physiological processes throughout the body, but when sleep timing changes suddenly – as it does with shift work or other non-standard schedules – the circadian system is forced to make an adjustment. The discrepancy is that recent research has shown that while the central circadian pacemaker (SCN) synchronizes to a new schedule, peripheral clock rhythms (e.g., in white

blood cells, oral mucosa cells) may take longer to acclimatize. This mismatch results in a condition of internal desynchronisation. As such, endogenous activities, including hormone secretion and metabolism are dysregulated with respect to the sleep-wake schedule. It has been shown that this misalignment leads to impaired glucose tolerance and reduced insulin sensitivity [2].

3. The Impact of "Sleep Catch-up" on Metabolism and Body Weight

3.1 Obesity and BMI

Associations of social jetlag with higher odds of obesity are consistently observed in observational studies and meta-analyses. Arab et al. involving 230,000 participants, suggested a positive relationship between SJL and higher body mass index (BMI), fat mass, body fat percentage and waist circumference. People with social jetlag had a 20% elevated risk of being overweight or obese compared to those with regular sleep patterns. [7]. Mathew et al. found that for adolescents, every additional hour of social jetlag up-regulates odds of eating fast food and drinking sweetened beverages, while down-regulating odds of eating both breakfast as well as vegetables. These unfavorable changes in eating styles may serve as a mechanism contributing to the weight increase. It is important to note, however, that while preliminary analyses suggest a monotonically increasing BMI percentile trend with higher level of this phenomenon; the association decreases and becomes nonsignificant after adjusting for race/ethnicity. This implies that in the younger age group, demographic characteristics can have a modifying effect on the direct association of irregular sleep and body mass index [8].

3.2 Carbohydrate Metabolism

Effects of weekend catch-up sleep on carbohydrate metabolism contribute to the importance of extended phenomenon for metabolic health. Wang et al. reported that in persons with diabetes it has been noted that a catch-up sleep pattern itself does not correlate significantly with average glycemic control, and the duration of this practice is important. Specifically, catch-up sleep lasting between 1 and 2 hours is significantly associated with lower levels of hemoglobin A1C (HbA1c) and fasting plasma glucose compared to individuals who do not utilize this form of compensation. This specific duration of catch-up sleep is also associated with a substantial decrease in the odds of poor glycemic control [9].

These tendencies are also observed in general population studies on insulin resistance. Liu et al. report a U-shaped relationship between the length of catch-up sleep and severe insulin resistance. In this condition, an optimal period for compensation exists with minimal risk of

metabolic perturbation at between 0.7 and 1.0 hours. Short catch-up sleep (≤ 1h) is also associated with a lower insulin resistance risk among those who work less than or equal to 7h per night. In contrast, very long catch-up sleep of 2 hours or more is implicated with an increasing likelihood of severe insulin resistance [10].

3.3 Hunger and Stress Hormones

Anomalous sleep, induced by social jetlag, causes metabolic changes that interfere with the mechanisms of appetite control. Studies show that circadian misalignment dampens the satiety hormone leptin and stimulates the appetite signal ghrelin. This metabolic imbalance promotes compensatory eating behaviors and hedonic hunger following acute sleep restriction or abrupt shifts in the circadian schedule. These physiological changes manifest as an increased preference for energy-dense foods and sugar-sweetened beverages. In younger populations, the impact of social jetlag is particularly pronounced, as it correlates with a significant increase in the consumption of ultra-processed foods and a concomitant reduction in the intake of fruits and vegetables.

Ungurianu and Marina have reported “dual hormonal signature” of circadian misalignment comprised of reduction in melatonin and dysregulation of cortisol concurrently. Social jetlag and irregular sleep schedules result in blunting of the nocturnal melatonin peak and decrease in its circadian amplitude. Melatonin acts not only as a regulator of sleep, but also has antioxidant, anti-inflammatory and cardioprotective properties; therefore, chronic deficiency by the disturbance of rhythms potentiates cardiovascular diseases and metabolic disorders. In addition, persons with sustained circadian misalignment manifest dysregulation of the hypothalamic-pituitary-adrenal system and have higher levels of morning cortisol with loss of the normal diurnal variation. This hyperactivation of hormonal stress signaling is a biology inscription of the chronic demand on the organism [11].

It’s this hormonal imbalance that is the reason behind catching up on sleep debt during weekends, not working as well to recover metabolism. The constant hormonal dyssynchronization is maintained even when sleep time is lengthened by keeping the body in a permanent state of physiological distress due to altered cortisol profiles and suppressed melatonin, thereby facilitating that a person will continue to maintain unhealthy eating behaviors and choose foods that promote overweight.

4. Cardiovascular Outcomes

4.1 Blood Pressure and Lipid Profile

Nakamura et al. found that even acute SJL – constituted by a 3h delay in bedtime on weekends – is associated with a significant enhancement of the morning blood pressure (BP). This is a well-known independent risk factor for cardiovascular events, which implies that acute sleep timing variability puts direct pressure on the circulatory system. The augmented morning BP surge in the setting of acute social jetlag may be due to greater central arterial stiffness. In particular, the loss of the arteriolar "Windkessel function" that normally dampens ejection pressure may account for the higher blood pressures seen [12].

Zhang et al. proved that social jetlag is a prominent contributor to fatty liver disease, also referred to as metabolic dysfunction-associated steatotic liver disease (MASLD). Studies have revealed that those afflicted by SJL tend to have elevated blood fats (triglycerides [TG]) and signs of liver distress like ALT and AST. This occurs because SJL disrupts the body's natural daily rhythm of a hormone called prolactin (PRL). Usually, a protein known as RORα regulates the timing of prolactin production in the pituitary gland, but SJL blocks that process. When that rhythm of prolactin release is disrupted, the liver loses its capacity to control fat in a normal fashion. The disruption reduces the amounts of cyclin D1 (CCND1), a protein that normally serves as a "brake" to prevent the liver from producing excess fat. Without this brake, the liver ramps up its fat production with enzymes such as FASN and ACC, causing fat accumulation in the liver cells. The study also indicates that the best treatment for this is “chronotherapy” – where prolactin is given at a time of day to mimic its normal cycle, and it works much better to heal liver than its prescription medicine [13].

4.2. Cardiovascular Outcomes and Mortality

4.2.1. Predictive Value for Global Cardiovascular Risk Studies by Gamboa Madeira et al. consider social jetlag as new and independent risk factor for high cardiovascular risk in shift or irregular workers. In accordance with the Systematic Coronary Risk Evaluation (SCORE) chart, calculated 10-year relative risk of cardiovascular death showed that 20.3% of manual workers fell into high-risk category. For each additional hour of SJL, the likelihood that these individuals were part of this high-risk cardiovascular group increased by over 30%. This reported correlation was not attenuated when age, gender, education, and lifestyle (ascertained as possible confounders from the literature) were held constant. Moreover, this article also

reported a dose-response association between magnitude of SJL and the proportion of both smokers and people with hypertension [14].

4.2.2. Autonomic Dysfunction and Ischemic Risk The analysis of Meléndez-Fernández et al. details how the disruption of endogenous circadian rhythms, including social jetlag (SJL), severely compromises autonomic control of the cardiovascular system. Circadian misalignment is significantly associated with an increased risk of severe outcomes, including of stroke and cardiovascular diseases (CVD). A principal physiological mechanism implicated is the lowering of awake cardiac vagal tone by 8% to 15% when there is a misalignment. This reduction of vagal tone frequently does not sufficiently offset sympathetic activation and may ultimately lead to ventricular tachycardia (VT), sudden cardiac death or heart failure. In addition to this the inability to normally decrease nocturnal blood pressure, which is often a result of rhythm disturbances, termed non-dipping hypertensive disorder where patients are at an increased risk for morbid cardiac events [15].

4.2.3. Metabolic and Biochemical Markers of Risk Belloir et al. compile proof about the biochemical and hemodynamic mechanisms by which sleep variability and SJL contribute to cardiovascular disease. Although there are limited or mixed results on epidemiological studies investigating a direct relationship between social jetlag and blood pressure, more social jetlag is associated with other important cardiovascular risk factors such as triglyceride, total cholesterol, and evening cortisol. SJL is further related to lower HDL concentrations and an elevated resting heart rate. These findings also imply that sleep irregularity – defined as day-to-day variability in the timing and duration of sleep – functions as a modifiable risk factor for cardiovascular events not influenced by habitual sleep quantity or quality [16].

4.2.4. The Myth of Weekend Compensation and Behavioral Synergies One study, conducted among college teachers (Galeno et al.) that such “paying” on weekends of a work-week sleep debt is associated with circadian misalignment and metabolic risk. Now, by contrast, scientists have discovered that professors who get less sleep on weekdays because of professional obligations often lengthen their average sleeps on free days – and this comes with an increased amount of social jetlag. This pattern is correlated positively with higher blood glucose levels and hyperglycemia, indicating that when individuals “make up” on the weekends they do not protect themselves against systemic metabolic stress deriving from irregularity of sleep patterns. Additionally, SJL was elevated in drinkers who may consume alcohol in an

attempt to cope with misalignment stress though it ultimately disrupts sleep architecture and aggravates cardiovascular risk factors [17].

5. Cognitive and Mental Health

Sun et al. points out that social jetlag is an important factor in the mental health of young individuals, especially adolescents and young adults. According to their meta-analysis high social jetlag (≥2h) is robustly related with higher odds ratios for depressive symptoms (OR=1.44). The neurobiological mechanism underlying these associations includes ventral striatal reward synergy with attenuation associated with depression, HPA axis overactivation, and phase-delayed cortisol rhythms. Moreover, behavioral issues, such as nighttime exposure to screens, augment these disruptions of the circadian cycle while also eliciting emotional problems [18].

A meta-analysis by Gao et al. from 25 observational studies which found a significant yet small association between social jetlag and depressive symptoms (pooled r=0.049). Although this effect accounts for only 0.24% of variance, it is an effect that should be considered in public health surveillance given its population level implications. No clear relation with anxiety was demonstrated, although SJL could deteriorate prefrontal cortex neuroplasticity and emotional control associated with disturbed melatonin and cortisol rhythms. Further, SJL is frequently related to lower levels of outdoor activity and physical exercise, which weaken the protective neurobiological effects mediated by natural exposure and positive emotions [19]. Moderie et al. show that 21.8% of resident physicians suffer from severe social jetlag, and that this represents a significant influence factor for increased depressive symptoms (OR=1.94). This relationship is not significantly attenuated even when adjusting for sleep disorders, and circadian misalignment appears to be an independent risk factor for resident psychological health. Severe SJL was more common in those at earlier stages of training and program (in which the social schedule conflicts can be particularly challenging with prevalence of late chronotypes seen in young adults). This indicates that chronotherapeutic approaches like light therapy and psychoeducation may be useful interventions for attenuating the psychological health risk in this population [20].

6. Discussion

The compilation of evidence suggests that the modern lifestyle – characterized by artificial light at night (ALAN) exposure and busy social schedules – and as such has induced a state of generalized biological desynchronization. A key result from this review is that there may be a

central importance in terms of maintenance of cardiometabolic health for the stability of the circadian rhythm compared to, or beyond, simply amount and distribution if sleep. Social jetlag (SJL) arises as a mismatch between an individual's endogenous biological rhythm and the timing of social activities, leading to a state of chronic physiologic stress.

Of particular interest is the paradox of "weekend catch-up sleep" (WCS). While a narrow "therapeutic window" of 1 to 2 hours of additional sleep may offer temporary benefits for glycemic control in sleep-deprived individuals, exceeding this limit exacerbates the phase shift. This exacerbates the metabolic and cardiovascular outcomes throughout the rest of the work week. There is evidence of a U-shaped relationship with compensation duration, that is both no recovery and excessive weekend oversleeping are associated with the increased likelihoods of insulin resistance and obesity.

This system is tightly linked with a “dual hormonal signature” – concomitant depression of melatonin and disruption of the HPA axis, as indicated by increased morning cortisol. This hormonal cascade, which induces adiposity and contributes to the pathogenesis of MASLD by inhibiting prolactin-mediated lipid homeostasis in the liver, as well as straining the cardiovascular system due to increased atherogenic activity, leads to an increased morning BP surge. Moreover, in the group of mental illness, SJL serves as an independent risk factor itself that contributes to depression by disarranging prefrontal cortex neuroplasticity and ventral striatum's reward system. Insofar, the practice of trying to “repay” sleep debt during weekends is a never-ending story and usually also counterproductive because it does not address internal desynchrony between central-driven pacemaker in the SCN as well as peripheral tissue oscillators.

7. Conclusions

In summary, our review provides evidence that sleep rhythm variability (i.e. as measured by social jetlag) is an independent and modifiable risk factor for major non-communicable diseases. The results showed a negative multisystem effect of irregular sleep timing in the human organism which was associated with higher BMI, worsening glucose metabolism and onset of dyslipidemia and an increased risk for cardiovascular events such as stroke and ischemic heart disease.

Also, the idea of recovering a "debt" in sleep has proven to be a myth; while it is the case that behaviorally 'making up' for lost sleep (i.e., extending sleep through longer time in bed over a weekend) will temporarily attenuate reports of subjective sleepiness, what cannot be restored are the deeply imbedded metabolic and hormonal responses to circadian phase shifts. For

clinical medicine and public health, the data emphasize the imperative of an “rhythm hygiene” approach – not only on quantity but also vigorous attention to pressing questions regarding optimal timing of sleep. Future interventions should focus on educating to avoid ALAN exposure, as much possible harmonize work and school hours with individual chronotypes. With a worldwide epidemic of metabolic and mood-disorders surging forward, stabilization of our natural (circadian) rhythm should be considered a linchpin in preventive medicine and a critical component for upholding homeostasis in our modern world.

Disclosure

Authors do not report any disclosures.

Authors’ contribution:

Conceptualization: Klaudia Brzoza, Michał Kociński, Patryk Iglewski
Methodology: Filip Matusiak, Michał Pietrasz, Michał Kociński
Investigation: Klaudia Brzoza, Filip Matusiak, Anna Komarczewska
Software: Michał Kociński, Patryk Iglewski, Michał Pietrasz
Formal analysis: Klaudia Brzoza, Michał Pietrasz, Anna Komarczewska
Writing- rough preparation: Klaudia Brzoza, Filip Matusiak, Anna Komarczewska
Writing- review and editing: Klaudia Brzoza, Patryk Iglewski, Filip Matusiak
Resources: Klaudia Brzoza, Michał Kociński, Michał Pietrasz

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 Statement:

The authors declare no conflict of interest.

Declaration on the use of AI: In preparing this work, the authors used Gemini for the purpose of improving language and readability, text formatting, and verification of bibliographic styles. After using this tool/service, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References

1. Bará S, Falchi F. Artificial light at night: a global disruptor of the night-time environment. *Philos Trans R Soc Lond B Biol Sci*. 2023;378(1892):20220352. doi:10.1098/rstb.2022.0352
2. Boivin DB, Boudreau P, Kosmadopoulos A. Disturbance of the Circadian System in Shift Work and Its Health Impact. *J Biol Rhythms*. 2022;37(1):3-28. doi:10.1177/07487304211064218
3. Guzzetti JR, Banks S. Dynamics of recovery sleep from chronic sleep restriction. *Sleep Adv*. 2022;4(1):zpac044. Published 2022 Nov 30. doi:10.1093/sleepadvances/zpac044
4. Lu J, Liu R, Wang J, et al. Associations of sleep characteristics with all-cause, cardiovascular and non-cardiovascular mortality among rural Chinese older adults: a cohort study. *BMJ Open*. 2025;15(6):e094928. Published 2025 Jun 22. doi:10.1136/bmjopen-2024-094928
5. Roenneberg T, Pilz LK, Zerbini G, Winnebeck EC. Chronotype and Social Jetlag: A (Self-) Critical Review. *Biology (Basel)*. 2019;8(3):54. Published 2019 Jul 12. doi:10.3390/biology8030054
6. Le L, Lan Z, Chen C. Association between weekend catch-up sleep and depression in US adults. *BMC Public Health*. 2025;25(1):439. Published 2025 Feb 4. doi:10.1186/s12889-025-21551-8
7. Arab A, Karimi E, Garaulet M, Scheer FAJL. Social jetlag and obesity: A systematic review and meta-analysis. *Obes Rev*. 2024;25(3):e13664. doi:10.1111/obr.13664
8. Mathew GM, Hale L, Chang AM. Social jetlag, eating behaviours and BMI among adolescents in the USA. *Br J Nutr*. 2020;124(9):979-987. doi:10.1017/S0007114520001804
9. Wang P, Li Q, Yu X, et al. Association between weekend catch-up sleep and glycemic control among individuals with diabetes: a population-based study. *Front Endocrinol (Lausanne)*. 2025;16:1461367. Published 2025 Jun 5. doi:10.3389/fendo.2025.1461367
10. Liu X, Chu A, Ding X. Investigating the associations between weekend catch-up sleep and insulin resistance: NHANES cross-sectional study. *BMC Med*. 2025;23(1):311. Published 2025 May 28. doi:10.1186/s12916-025-04154-3

11. Ungurianu A, Marina V. Melatonin and Cortisol Suppression and Circadian Rhythm Disruption in Burnout Among Healthcare Professionals: A Systematic Review. *Clin Pract*. 2025;15(11):199. Published 2025 Oct 29. doi:10.3390/clinpract15110199
12. Nakamura N, Akiyama H, Nishimura M, et al. Acute social jetlag augments morning blood pressure surge: a randomized crossover trial. *Hypertens Res*. 2023;46(9):2179-2191. doi:10.1038/s41440-023-01360-5
13. Zhang PZ, Shi YH, Guo YX, et al. Social jetlag elicits fatty liver via perturbed circulating prolactin rhythm-mediated circadian remodeling of hepatic lipid metabolism. *Mil Med Res*. 2025;12(1):29. Published 2025 Jun 3. doi:10.1186/s40779-025-00609-z
14. Gamboa Madeira S, Reis C, Paiva T, Moreira CS, Nogueira P, Roenneberg T. Social jetlag, a novel predictor for high cardiovascular risk in blue-collar workers following permanent atypical work schedules. *J Sleep Res*. 2021;30(6):e13380. doi:10.1111/jsr.13380
15. Meléndez-Fernández OH, Walton JC, DeVries AC, Nelson RJ. Clocks, Rhythms, Sex, and Hearts: How Disrupted Circadian Rhythms, Time-of-Day, and Sex Influence Cardiovascular Health. *Biomolecules*. 2021;11(6):883. Published 2021 Jun 14. doi:10.3390/biom11060883
16. Belloir J, Makarem N, Shechter A. Sleep and Circadian Disturbance in Cardiovascular Risk. *Curr Cardiol Rep*. 2022;24(12):2097-2107. doi:10.1007/s11886-022-01816-z
17. Galeno DML, Peixoto HJA, Carneiro BTS, Leocadio-Miguel MA. Cardiometabolic risk factors and social jetlag in university professors. *Braz J Med Biol Res*. 2023;56:e12539. Published 2023 Jun 30. doi:10.1590/1414-431X2023e12539
18. Sun S, Yang Y, Yu F, et al. Social jetlag and depressive symptoms among young people: a systematic review and meta-analysis. *BMC Psychiatry*. 2025;25(1):664. Published 2025 Jul 1. doi:10.1186/s12888-025-07066-x
19. Gao L, Gao J, Zeng Z. Social jet lag and mental health outcomes: A systematic review and meta-analysis. *Acta Psychol (Amst)*. 2025;259:105419. doi:10.1016/j.actpsy.2025.105419
20. Moderie C, Paradis C, Philippe FL, Geoffroy MC, Guay E, Paquin V. Sleep, chronotype, social jetlag, and mental health in resident physicians: a cross-sectional study. *J Clin Sleep Med*. 2024;20(12):1915-1922. doi:10.5664/jcsm.11288