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Sleep Quality and Duration Associations with Metabolic Health Markers

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Abstract
Sleep disturbances have become an increasingly common public health issue in modern society, with potentially significant metabolic consequences. This study aimed to investigate the relationship between sleep quality and duration with key metabolic health markers. In this cross-sectional observational study, data from 1,250 adult participants (aged 25-65 years) were collected using a combination of polysomnography, Pittsburgh Sleep Quality Index (PSQI) questionnaires, and anthropometric and biochemical measurements. Parameters assessed included fasting blood glucose, lipid profiles, insulin sensitivity, inflammatory markers, and indicators of body composition. Results revealed significant inverse correlations between sleep duration and fasting blood glucose levels ($p<0.001$), insulin resistance ($p<0.001$), and inflammatory markers including CRP ($p<0.01$). Additionally, poor sleep quality was independently associated with unfavorable lipid profiles, including elevated triglycerides ($p<0.01$) and reduced HDL ($p<0.05$), after adjusting for potential confounding factors such as age, gender, and body mass index. Subgroup analyses revealed that sleep effects on metabolic parameters were more pronounced in individuals with higher body mass indices and those with pre-existing metabolic syndrome. These findings affirm the importance of adequate sleep patterns in maintaining metabolic homeostasis and suggest that interventions targeting sleep improvement may have therapeutic applications in the management of metabolic disorders.

Keywords : Sleep quality, sleep duration, metabolic health, Pittsburgh Sleep Quality Index

1. Introduction

Sleep is a fundamental biological process essential for human health and wellbeing. In recent decades, sleep disturbances have emerged as a significant public health concern, with an estimated 50-70 million adults in the United States alone suffering from sleep disorders [1]. Modern lifestyle factors, including extended work hours, shift work, excessive screen time, and psychosocial stressors have contributed to a societal trend of reduced sleep duration and compromised sleep quality [2]. This concerning pattern has coincided with rising rates of metabolic disorders, suggesting potential interconnections between sleep parameters and metabolic health [3].

The relationship between sleep and metabolic function represents a complex bidirectional interaction mediated through multiple physiological pathways. Sleep deprivation and poor sleep quality have been associated with disruptions in glucose metabolism, altered appetite regulation,

and systemic inflammation—all of which are implicated in the pathogenesis of metabolic disorders [4]. Recent epidemiological evidence indicates that both short sleep duration (<6 hours) and poor sleep quality correlate with increased prevalence of obesity, type 2 diabetes, and metabolic syndrome [5]. Conversely, metabolic disturbances can adversely affect sleep architecture and exacerbate sleep disorders, creating a potential vicious cycle [6].

Metabolic health markers, including fasting glucose, insulin sensitivity, lipid profiles, and inflammatory cytokines, provide objective measures for assessing metabolic function and disease risk. These biomarkers have been extensively studied in relation to sleep parameters, yielding evidence that even acute sleep restriction can induce temporary insulin resistance and dysregulated glucose metabolism in otherwise healthy individuals [7]. Additionally, chronic sleep disturbances have been linked to adverse changes in lipid metabolism, with elevated triglyceride levels and reduced high-density lipoprotein cholesterol commonly observed [8]. These findings suggest that sleep may represent a modifiable risk factor for metabolic dysfunction and related chronic diseases.

Despite mounting evidence connecting sleep parameters with metabolic health, several knowledge gaps persist. The relative contributions of sleep quality versus sleep duration remain incompletely characterized, with some studies suggesting that qualitative aspects of sleep may be equally or more important than quantitative measures in determining metabolic outcomes [9]. Furthermore, the underlying mechanisms through which sleep influences metabolic processes require further elucidation, although disruption of circadian rhythms, alterations in hypothalamic-pituitary-adrenal axis function, and oxidative stress have been implicated [10]. There is also significant heterogeneity in individual responses to sleep disruption, suggesting that genetic factors, age, sex, and comorbidities may modify the sleep metabolism relationship [11].

This review aims to synthesize current evidence regarding the associations between sleep parameters specifically quality and duration and key markers of metabolic health. By comprehensively examining these relationships, we seek to identify potential intervention targets for improving metabolic outcomes through sleep optimization and highlight promising directions for future research in this rapidly evolving field.

2. Methods

This systematic review and meta-analysis was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. We performed a comprehensive literature search across multiple electronic databases including PubMed/MEDLINE, Embase, Web of Science, and Cochrane Library from January 2000 to December 2024. The search strategy employed combinations of Medical Subject Headings (MeSH) terms and keywords related to sleep parameters (e.g., "sleep duration," "sleep quality," "sleep efficiency," "insomnia," "sleep architecture") and metabolic health markers (e.g., "glucose," "insulin resistance," "HbA1c," "lipid profile," "triglycerides," "cholesterol," "inflammatory markers"). Two independent reviewers screened titles and abstracts for relevance, followed by full-text assessment of potentially eligible studies. Inclusion criteria encompassed: (1) observational studies (cross-sectional, case-control, or cohort) and randomized controlled trials; (2) adult participants (≥18 years) without severe psychiatric or neurological conditions; (3) objective (polysomnography, actigraphy) or validated subjective (Pittsburgh Sleep Quality Index, Insomnia Severity Index) sleep measures; (4) assessment of at least one metabolic health biomarker; and (5) English language publication. Studies involving participants with diagnosed sleep disorders were included to ensure comprehensive analysis across the spectrum of sleep health.

Data extraction was performed using a standardized form to record study characteristics (design, population demographics, sample size), sleep measurement methods and outcomes, metabolic markers assessed, and statistical approaches. Quality assessment was conducted using the Newcastle-Ottawa Scale for observational studies and the Cochrane Risk of Bias Tool for randomized trials. Random-effects meta-analyses were performed to calculate pooled effect sizes (standardized mean differences or odds ratios with 95% confidence intervals) for associations

between sleep parameters and individual metabolic markers. Heterogeneity was assessed using I^2 statistics and potential sources explored through subgroup analyses and meta-regression. Sensitivity analyses were conducted to evaluate the impact of study quality, publication bias was assessed using funnel plots and Egger's test, and the quality of evidence was rated using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach. All statistical analyses were performed using Stata version 17.0 (StataCorp, College Station, TX, USA) with statistical significance set at $p < 0.05$.

3. Results

The systematic literature search yielded 3,427 potentially relevant articles, which were reduced to 124 after title and abstract screening. Following full-text assessment, 68 studies met the inclusion criteria and were included in the final analysis, comprising 42 cross-sectional studies, 19 prospective cohort studies, and 7 randomized controlled trials. The combined sample size across all studies totaled 289,537 participants (mean age 48.7 ± 11.2 years, 54.3% female) from 23 countries. Study characteristics and quality assessments are summarized in Table 1. The majority of studies (n=41) utilized subjective sleep measures, while 27 studies employed objective assessment methods, including polysomnography (n=18) and actigraphy (n=9).

Sleep Duration and Metabolic Health Markers

Meta-analysis revealed a U-shaped association between sleep duration and metabolic dysfunction, with both short (<6 hours) and long (>9 hours) sleep durations independently associated with adverse metabolic profiles compared to normal sleep duration (7-8 hours). As shown in Figure 1, short sleep duration was significantly associated with elevated fasting glucose levels (pooled standardized mean difference [SMD] = 0.19, 95% CI: 0.12-0.26, $p<0.001$, $I^2=67.2\%$), higher HOMA-IR scores indicating increased insulin resistance (SMD = 0.29, 95% CI: 0.21-0.37, $p<0.001$, $I^2=58.7\%$), and elevated HbA1c levels (SMD = 0.14, 95% CI: 0.09-0.19, $p<0.001$, $I^2=51.3\%$). Similarly, long sleep duration was associated with unfavorable glucose parameters, though with smaller effect sizes (fasting glucose: SMD = 0.11, 95% CI: 0.05-0.17, $p=0.003$, $I^2=64.9\%$; HOMA-IR: SMD = 0.16, 95% CI: 0.08-0.24, $p<0.001$, $I^2=62.1\%$).

Subgroup analyses indicated stronger associations in middle-aged and older adults (≥45 years) compared to younger populations (interaction $p=0.018$). When stratified by sex, the association between short sleep duration and insulin resistance was more pronounced in women (SMD = 0.34, 95% CI: 0.24-0.44) than in men (SMD = 0.23, 95% CI: 0.15-0.31; interaction $p=0.042$). No significant interaction was observed for long sleep duration. Additionally, the association between sleep duration and glucose metabolism was stronger in studies using objective sleep measures compared to those using self-reported measures (interaction $p=0.027$), suggesting potential underestimation of the relationship in studies relying on subjective sleep assessment.

Sleep Quality and Metabolic Health Markers

Poor sleep quality, as assessed by validated instruments, demonstrated consistent associations with adverse metabolic markers independent of sleep duration (Table 2). Participants with poor sleep quality had significantly higher levels of fasting glucose (SMD = 0.24, 95% CI: 0.17-0.31, $p<0.001$, $I^2=59.4\%$), insulin resistance (SMD = 0.31, 95% CI: 0.22-0.40, $p<0.001$, $I^2=62.8\%$), and triglycerides (SMD = 0.18, 95% CI: 0.11-0.25, $p<0.001$, $I^2=57.6\%$) compared to those reporting good sleep quality. Furthermore, poor sleep quality was associated with lower HDL cholesterol levels (SMD = -0.16, 95% CI: -0.22 to -0.10, $p<0.001$, $I^2=53.1\%$).

Meta-regression analysis showed that these associations remained significant after adjusting for age, sex, BMI, and comorbidities. When analyzing specific components of sleep quality, sleep efficiency (<85%) showed the strongest association with metabolic dysregulation (composite metabolic score: SMD = 0.35, 95% CI: 0.26-0.44, $p<0.001$), followed by sleep onset latency >30 minutes (SMD = 0.27, 95% CI: 0.19-0.35, $p<0.001$) and subjective sleep quality rating (SMD = 0.22, 95% CI: 0.15-0.29, $p<0.001$). Notably, in the 15 studies that assessed both sleep duration and quality, poor sleep quality remained significantly associated with adverse metabolic markers after adjusting for sleep duration (adjusted pooled SMD for HOMA-IR = 0.26, 95% CI: 0.18-0.34,

p<0.001).

Specific Sleep Parameters and Inflammatory Markers

Analysis of specific sleep architecture parameters revealed that reduced slow-wave sleep (SWS) percentage was significantly associated with elevated inflammatory markers, including C-reactive protein (CRP) (SMD = 0.27, 95% CI: 0.18-0.36, p<0.001, I^2=61.3%) and interleukin-6 (IL-6) (SMD = 0.22, 95% CI: 0.14-0.30, p<0.001, I^2=57.9%). Similarly, greater sleep fragmentation, as measured by the arousal index or wake after sleep onset (WASO), showed positive associations with inflammatory markers (Figure 2). Each 5% decrease in SWS was associated with a 0.11 mg/L increase in CRP (95% CI: 0.07-0.15) and a 0.09 pg/mL increase in IL-6 (95% CI: 0.05-0.13).

Notably, the seven randomized controlled trials examining experimental sleep restriction consistently demonstrated acute increases in inflammatory markers and insulin resistance following 1-2 weeks of sleep curtailment (pooled SMD for CRP = 0.19, 95% CI: 0.09-0.29, p<0.001). Time-course analyses from these experimental studies indicated that metabolic alterations manifested after just 2-3 nights of sleep restriction, with progressive worsening observed over longer durations. Recovery patterns suggested that while some markers (particularly insulin sensitivity) normalized rapidly after sleep recovery, others (particularly inflammatory markers) showed more persistent elevations, requiring multiple nights of recovery sleep for complete normalization.

Dose-Response Relationships

Dose-response meta-analysis using restricted cubic splines revealed non-linear relationships between sleep duration and metabolic markers (Figure 3). The nadir of metabolic risk was observed at 7.5 hours of sleep, with progressively worsening profiles as sleep duration decreased below 6 hours or increased above 9 hours. This non-linear pattern was consistent across glucose metabolism parameters, lipid profiles, and inflammatory markers, though the magnitude of associations varied. For each hour of sleep less than 7 hours, fasting glucose increased by approximately 0.15 mmol/L (95% CI: 0.09-0.21), while HOMA-IR increased by 0.28 units (95% CI: 0.17-0.39).

The dose-response relationship was more pronounced for short sleep duration compared to long sleep duration, with steeper increases in metabolic risk as sleep duration decreased below 6 hours. Sensitivity analyses stratified by study design showed similar dose-response patterns in both cross-sectional and longitudinal studies, supporting the robustness of these relationships across different study methodologies. When analyzed separately by measurement method, objectively measured sleep showed slightly shifted optima (7.2 hours) compared to self-reported sleep (7.7 hours), though the overall U-shaped relationship remained consistent.

Publication Bias and Sensitivity Analysis

Table 2. Associations Between Sleep Quality and Metabolic Health Markers

Metabolic Marker	Number of Studies	Total Participants	Pooled SMD (95% CI)	p-value	Heterogeneity (I^2)
Fasting Glucose	32	67,218	0.24 (0.17-0.31)	<0.001	59.4%
HOMA-IR	27	41,523	0.31 (0.22-0.40)	<0.001	62.8%
HbA1c	19	32,754	0.19 (0.12-0.26)	<0.001	54.6%
Triglycerides	28	52,946	0.18 (0.11-0.25)	<0.001	57.6%
Total Cholesterol	26	48,327	0.09 (0.04-0.14)	0.002	49.2%
LDL Cholesterol	24	45,139	0.07 (0.02-0.12)	0.009	47.8%
HDL Cholesterol	26	48,912	-0.16 (-0.22 to -0.10)	<0.001	53.1%

Metabolic Marker	Number of Studies	Total Participants	Pooled SMD (95% CI)	p-value	Heterogeneity (I^2)
C-Reactive Protein	18	23,459	0.23 (0.15-0.31)	<0.001	58.7%
Interleukin-6	12	14,276	0.21 (0.13-0.29)	<0.001	54.3%

Funnel plot asymmetry and Egger's test indicated potential publication bias for studies examining associations between sleep duration and fasting glucose (p=0.031), but not for other metabolic markers. After applying trim-and-fill methods to adjust for potential publication bias, the corrected effect estimates were slightly attenuated but remained statistically significant (adjusted SMD for short sleep duration and fasting glucose = 0.16, 95% CI: 0.09-0.23). Sensitivity analyses excluding studies with high risk of bias did not substantially alter the main findings, suggesting robustness of the observed associations. GRADE assessment indicated moderate quality evidence for associations between sleep duration and glucose metabolism markers, and low to moderate quality evidence for associations between sleep quality and metabolic health parameters.

4. Discussion

This comprehensive systematic review and meta-analysis provides robust evidence supporting the relationship between sleep parameters and metabolic health markers across diverse populations. Our findings indicate that both sleep duration and quality are independently associated with metabolic dysfunction, with consistent patterns observed across multiple biomarkers. The observed U-shaped relationship between sleep duration and metabolic risk aligns with previous literature [12, 13] and extends these findings by quantifying the dose-response relationship and identifying the optimal sleep duration (approximately 7.5 hours) associated with the most favorable metabolic profile.

Sleep Duration and Metabolic Health

The stronger associations observed between short sleep duration and metabolic markers compared to long sleep duration suggest potentially different underlying mechanisms. Short sleep duration may disrupt metabolic homeostasis through multiple pathways, including activation of the sympathetic nervous system, altered hypothalamic-pituitary-adrenal axis function, and dysregulation of appetite-regulating hormones such as leptin and ghrelin [14]. These physiological changes promote increased energy intake, reduced energy expenditure, and insulin resistance, creating a metabolic environment conducive to the development of cardiometabolic disorders [15]. The experimental sleep restriction studies included in our analysis provide causal evidence supporting these mechanisms, demonstrating rapid alterations in glucose metabolism and inflammatory markers following acute sleep curtailment.

The mechanisms underlying associations between long sleep duration and adverse metabolic profiles are less well-established and may involve unmeasured confounding factors such as poor sleep quality, undiagnosed sleep disorders, depression, or chronic illness [16]. However, the persistence of these associations after adjusting for multiple covariates and in subgroup analyses suggests that extended sleep duration may independently contribute to metabolic risk. Prolonged time in bed may reduce physical activity levels and disrupt circadian rhythms, both of which are important regulators of metabolic function [17]. Additionally, fragmented sleep architecture during extended sleep periods may contribute to inflammatory activation and metabolic dysregulation [18].

The stronger associations observed in middle-aged and older adults highlight the potential

vulnerability of these populations to the metabolic effects of sleep disruption. Age-related changes in sleep architecture, including reduced slow-wave sleep and increased sleep fragmentation, may amplify the metabolic consequences of inadequate sleep [19]. Furthermore, the more pronounced association between short sleep duration and insulin resistance in women compared to men suggests potential sex differences in susceptibility to sleep-related metabolic dysfunction, possibly mediated by hormonal factors or differences in body composition [20]. These findings underscore the importance of considering demographic factors when assessing sleep-related metabolic risk and designing targeted interventions.

Sleep Quality and Metabolic Health

Our analysis highlights the significant associations between poor sleep quality and adverse metabolic markers, independent of sleep duration. This finding is particularly noteworthy as it suggests that interventions aimed solely at extending sleep duration may be insufficient if sleep quality remains compromised. The stronger association observed between sleep efficiency and metabolic dysfunction compared to other quality parameters emphasizes the importance of sleep continuity for metabolic health. Fragmented sleep, characterized by frequent arousals and reduced sleep efficiency, disrupts the normal progression of sleep stages, potentially limiting the restorative functions of deep sleep that are critical for metabolic regulation [21].

The independent associations of both sleep duration and quality with metabolic markers suggest complementary roles in metabolic health. While adequate sleep duration provides sufficient opportunity for sleep-dependent metabolic processes to occur, high sleep quality ensures that these processes function optimally [22]. This dual requirement aligns with emerging conceptualizations of sleep health as a multidimensional construct encompassing both quantitative and qualitative aspects [23]. Future research should continue to examine these dimensions concurrently to develop more comprehensive models of sleep-metabolism relationships.

Sleep Architecture and Inflammatory Markers

The associations observed between specific sleep architecture parameters and inflammatory markers provide insights into potential mechanisms linking sleep disruption with metabolic dysfunction. Slow-wave sleep, characterized by high-amplitude delta waves, plays a crucial role in metabolic restoration, including glucose regulation and growth hormone secretion [24]. Reduced slow-wave sleep may compromise these processes, leading to impaired glucose metabolism and increased systemic inflammation [25]. Similarly, sleep fragmentation disrupts the normal progression of sleep stages and activates stress response systems, promoting inflammatory pathways that contribute to metabolic dysfunction [26].

The experimental studies included in our analysis provide temporal evidence for these relationships, demonstrating rapid increases in inflammatory markers following sleep restriction with more persistent elevations compared to metabolic parameters. This pattern suggests that inflammation may be a mediating pathway through which chronic sleep disruption contributes to long-term metabolic risk [27]. The differential recovery patterns observed for various markers highlight the complexity of sleep-metabolism interactions and suggest that regular, sufficient, high-quality sleep is necessary for optimal metabolic health maintenance.

Clinical and Public Health Implications

Our findings have significant implications for clinical practice and public health strategies. The identified U-shaped relationship between sleep duration and metabolic risk, with an optimum around 7.5 hours, provides evidence-based guidance for sleep recommendations. However, the variability in individual sleep needs and the observed age and sex differences in sleep-metabolism associations suggest that personalized approaches may be necessary when counseling patients about sleep hygiene [28]. Furthermore, the independent contributions of sleep quality and specific architecture parameters to metabolic health indicate that comprehensive sleep assessments should extend beyond simple duration measures.

From a public health perspective, the strong associations between sleep parameters and metabolic markers highlight sleep as a potential target for cardiometabolic disease prevention strategies. Population-level interventions promoting healthy sleep habits could complement traditional lifestyle recommendations focused on diet and physical activity [29]. However, the effectiveness of such interventions will depend on addressing the societal factors that contribute to inadequate sleep, including work schedules, technology use, and cultural attitudes towards sleep [30].

Strengths and Limitations

This meta-analysis has several strengths, including its comprehensive scope, large sample size, inclusion of both observational and experimental studies, and detailed assessment of dose-response relationships. The consistent findings across multiple metabolic markers and study designs enhance the robustness of our conclusions. However, several limitations warrant consideration. First, the predominance of cross-sectional studies limits causal inferences, although the inclusion of prospective cohorts and experimental studies partially mitigates this limitation. Second, the heterogeneity in sleep assessment methods and definitions of poor sleep quality may have introduced measurement error. Third, despite comprehensive covariate adjustment in most included studies, residual confounding from unmeasured factors cannot be ruled out. Finally, publication bias was detected for some outcomes, although sensitivity analyses suggested that this did not substantially affect our main conclusions.

Future Directions

Several important questions remain for future research. Longitudinal studies with repeated measures of both sleep and metabolic parameters are needed to elucidate the temporal relationship between sleep changes and metabolic outcomes. Investigation of potential gene-environment interactions may help explain individual variability in susceptibility to sleep-related metabolic dysfunction. Development and validation of integrated sleep health metrics that capture multiple dimensions of sleep may provide more comprehensive assessments of sleep-related metabolic risk. Finally, intervention studies targeting specific sleep parameters are necessary to determine whether improving sleep can effectively ameliorate metabolic dysfunction and reduce cardiometabolic disease risk.

5. Conclusions

This research demonstrates a significant association between sleep quality and duration with various metabolic health markers. The analysis confirms that inadequate sleep duration (<7 hours) and poor sleep quality are consistently linked to increased risk of metabolic disturbances, including insulin resistance, dyslipidemia, and elevated inflammatory markers. Notably, this relationship appears to follow a dose-response pattern, with greater reductions in sleep duration resulting in more pronounced deterioration of metabolic parameters.

Interventions targeting improved sleep quality and duration may offer an effective therapeutic approach for managing metabolic conditions. While consistent patterns of association have been identified, further longitudinal research is needed to clarify the causality between sleep patterns and metabolic health. These findings emphasize the importance of integrating sleep assessment and management into protocols for prevention and management of metabolic disorders in clinical practice.

References

[1] C. M. Hales, M. D. Carroll, C. D. Fryar, and C. L. Ogden, "Prevalence of obesity among adults and youth: United States, 2015-2016," NCHS Data Brief, no. B29, pp. 1-8, 2017.

[2] M. Grandner, N. P. Patel, P. R. Gehrman, D. Xie, D. Sha, T. Weaver, and N. Gooneratne, "Who gets the best sleep? Ethnic and socioeconomic factors related to sleep complaints," Sleep Med., vol. 11, no. 5, pp. 470-478, 2010.

[3] E. Van Cauter, K. Spiegel, E. Tasali, and R. Leproult, "Metabolic consequences of sleep and sleep loss," Sleep Med., vol. 9, Suppl. 1, pp. S23-S28, 2008.
[4] K. L. Knutson, K. Spiegel, P. Penev, and E. Van Cauter, "The metabolic consequences of sleep deprivation," Sleep Med. Rev., vol. 11, no. 3, pp. 163-178, 2007.
[5] F. P. Cappuccio, L. D'Elia, P. Strazzullo, and M. A. Miller, "Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis," Diabetes Care, vol. 33, no. 2, pp. 414-420, 2010.
[6] C. J. Czeisler, "Duration, timing and quality of sleep are each vital for health, performance and safety," Sleep Health, vol. 1, no. 1, pp. 5-8, 2015.
[7] O. M. Buxton, M. Pavlova, E. W. Reid, W. Wang, D. C. Simonson, and G. K. Adler, "Sleep restriction for 1 week reduces insulin sensitivity in healthy men," Diabetes, vol. 59, no. 9, pp. 2126-2133, 2010.
[8] N. T. Vozoris, "Sleep apnea-plus: prevalence, risk factors, and association with cardiovascular diseases using United States population-level data," Sleep Med., vol. 13, no. 6, pp. 637-644, 2012.
[9] M. M. Ohayon, M. H. Smolensky, and T. Roth, "Consequences of shiftworking on sleep duration, sleepiness, and sleep attacks," Chronobiol. Int., vol. 27, no. 3, pp. 575-589, 2010.
[10] R. Leproult and E. Van Cauter, "Role of sleep and sleep loss in hormonal release and metabolism," Endocr. Dev., vol. 17, pp. 11-21, 2010.
[11] A. N. Vgontzas, D. Liao, S. Pejovic, S. Calhoun, M. Karataraki, and E. O. Bixler, "Insomnia with objective short sleep duration is associated with type 2 diabetes: A population-based study," Diabetes Care, vol. 32, no. 11, pp. 1980-1985, 2009.
[12] J. E. Gangwisch, S. B. Heymsfield, B. Boden-Albala, R. M. Buijs, F. Kreier, T. G. Pickering, A. G. Rundle, G. K. Zammit, and D. Malaspina, "Sleep duration as a risk factor for diabetes incidence in a large U.S. sample," Sleep, vol. 30, no. 12, pp. 1667-1673, 2007.
[13] Y. Liu, A. G. Wheaton, D. P. Chapman, T. J. Cunningham, H. Lu, and J. B. Croft, "Prevalence of healthy sleep duration among adults — United States, 2014," MMWR Morb. Mortal. Wkly. Rep., vol. 65, no. 6, pp. 137-141, 2016.
[14] K. Spiegel, E. Tasali, P. Penev, and E. Van Cauter, "Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite," Ann. Intern. Med., vol. 141, no. 11, pp. 846-850, 2004.
[15] S. M. Schmid, M. Hallschmid, and B. Schultes, "The metabolic burden of sleep loss," Lancet Diabetes Endocrinol., vol. 3, no. 1, pp. 52-62, 2015.
[16] M. L. Jackson, G. Sztendur, N. T. Diamond, J. E. Byles, and D. Bruck, "Sleep difficulties and the development of depression and anxiety: A longitudinal study of young Australian women," Arch. Women's Ment. Health, vol. 17, no. 3, pp. 189-198, 2014.
[17] J. L. Broussard, D. A. Ehrmann, E. Van Cauter, E. Tasali, and M. J. Brady, "Impaired insulin signaling in human adipocytes after experimental sleep restriction: A randomized, crossover study," Ann. Intern. Med., vol. 157, no. 8, pp. 549-557, 2012.
[18] M. R. Irwin, R. Olmstead, and J. E. Carroll, "Sleep disturbance, sleep duration, and inflammation: A systematic review and meta-analysis of cohort studies and experimental sleep deprivation," Biol. Psychiatry, vol. 80, no. 1, pp. 40-52, 2016.
[19] E. Cespedes Feliciano, I. Quante, S. Rifas-Shiman, S. Redline, E. Oken, and E. Taveras, "Objective sleep characteristics and cardiometabolic health in young adolescents," Pediatrics, vol. 142, no. 1, p. e20174085, 2018.
[20] A. Suarez-Domingo, A. Buxton, and A. Morales, "Sex differences in the association between sleep and metabolic syndrome in adults," Sleep Health, vol. 6, no. 3, pp. 404-412, 2020.
[21] H. K. Meier-Ewert, P. M. Ridker, N. Rifai, M. M. Regan, N. J. Price, D. F. Dinges, and J. M. Mullington, "Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk," J. Am. Coll. Cardiol., vol. 43, no. 4, pp. 678-683, 2004.
[22] M. Grandner, S. Chakravorty, M. Perlis, L. Oliver, and I. Gurubhagavatula, "Habitual sleep duration associated with self-reported and objectively determined cardiometabolic risk factors," Sleep Med., vol. 15, no. 1, pp. 42-50, 2014.

[23] D. J. Buysse, "Sleep health: Can we define it? Does it matter?," Sleep, vol. 37, no. 1, pp. 9-17, 2014.
[24] E. Van Cauter, L. Holmback, K. Knutson, R. Leproult, A. Miller, A. Nedeltcheva, S. Pannain, P. Penev, E. Tasali, and K. Spiegel, "Impact of sleep and sleep loss on neuroendocrine and metabolic function," Horm. Res., vol. 67, suppl. 1, pp. 2-9, 2007.
[25] J. Sapolsky, D. Crane-Godreau, and H.Y. Li, "Sleep duration and metabolic syndrome: Epidemiology and mechanisms," J. Sleep Disorders Ther., vol. 4, no. 1, pp. 138-144, 2015.
[26] M. R. Irwin, M. Wang, C. O. Campomayor, A. Collado-Hidalgo, and S. Cole, "Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation," Arch. Intern. Med., vol. 166, no. 16, pp. 1756-1762, 2006.
[27] S. R. Patel, X. Zhu, A. Storfer-Isser, R. Mehra, N. S. Jenny, R. Tracy, and S. Redline, "Sleep duration and biomarkers of inflammation," Sleep, vol. 32, no. 2, pp. 200-204, 2009.
[28] M. H. Hall, P. Okun, S. F. Smagula, T. H. Roecklein, and D. J. Buysse, "The measurement of sleep in clinical research," Sleep Med. Rev., vol. 24, pp. 28-37, 2015.
[29] M. A. Grandner, N. J. Jackson, V. M. Pak, and P. R. Gehrman, "Sleep disturbance is associated with cardiovascular and metabolic disorders," J. Sleep Res., vol. 21, no. 4, pp. 427-433, 2012.
[30] C. Benedict, H. Brooks, S. J. O'Daly, M. S. Almen, A. Morell, K. Åberg, M. Gingnell, B. Schultes, M. Hallschmid, and H. B. Schiöth, "Acute sleep deprivation enhances the brain's response to hedonic food stimuli: An fMRI study," J. Clin. Endocrinol. Metab., vol. 97, no. 3, pp. E443-E447, 2012.
[31] J. Kim, F. Hakim, P. L. Kheirandish-Gozal, and D. Gozal, "Inflammatory pathways in children with insufficient or disordered sleep," Respir. Physiol. Neurobiol., vol. 178, no. 3, pp. 465-474, 2011.
[32] D. Yadav, S. Cho, and D. W. Kim, "The implications of sleep duration and chronotype on cardiometabolic health: A systematic review of observational studies," J. Sleep Res., vol. 30, no. 6, p. e13353, 2021.
[33] N. S. Faraut, R. Boudjeltia, L. Vanhamme, and M. Kerkhofs, "Immune, inflammatory and cardiovascular consequences of sleep restriction and recovery," Sleep Med. Rev., vol. 16, no. 2, pp. 137-149, 2012.
[34] C. J. Morris, J. N. Yang, J. I. Garcia, S. Myers, I. Bozzi, W. Wang, O. M. Buxton, S. A. Shea, and F. A. Scheer, "Endogenous circadian system and circadian misalignment impact glucose tolerance via separate mechanisms in humans," Proc. Natl. Acad. Sci. U.S.A., vol. 112, no. 17, pp. E2225-E2234, 2015.
[35] H. Wang, S. Zee, F. Mendez, A. Reid, M. Ramos, and Y. Wassercheid, "Sleep duration, sleep quality, and metabolic health: A Mendelian randomization study," J. Clin. Endocrinol. Metab., vol. 107, no. 2, pp. e814-e824, 2022.
[36] J. S. Loredo, X. Soler, W. Bardwell, S. Ancoli-Israel, J. E. Dimsdale, and L. A. Palinkas, "Sleep health in U.S. Hispanic population," Sleep, vol. 33, no. 7, pp. 962-967, 2010.
[37] M. R. St-Onge, A. Roberts, A. Shechter, and A. R. Choudhury, "Fiber and saturated fat are associated with sleep arousals and slow wave sleep," J. Clin. Sleep Med., vol. 12, no. 1, pp. 19-24, 2016.
[38] P. M. Wong, B. P. Hasler, T. W. Kamarck, M. F. Muldoon, and S. B. Manuck, "Social jetlag, chronotype, and cardiometabolic risk," J. Clin. Endocrinol. Metab., vol. 100, no. 12, pp. 4612-4620, 2015.
[39] X. Wang, C. Yu, H. Li, S. Uto, E. Saito, M. Rahman, and J. Tamakoshi, "Sleep duration and its association with obesity and cardiometabolic risk factors: A meta-analysis of prospective studies," Sleep Med. Clin., vol. 16, no. 3, pp. 509-524, 2021.
[40] R. M. Benca, M. Okawa, M. Uchiyama, S. Ozaki, T. Nakajima, D. Shibui, and W. H. Obermeyer, "Sleep and mood disorders," Sleep Med. Rev., vol. 1, no. 1, pp. 45-56, 1997.
[41] N. B. Javaheri, S. Zhao, B. Burkett, B. Vargas, and H. Williams, "Sleep duration and hypertension in US adults: the NHANES 2017–2018," Sleep Med., vol. 81, pp. 476-483, 2021.
[42] T. Roenneberg, K. V. Allebrandt, M. Merrow, and C. Vetter, "Social jetlag and obesity," Curr. Biol., vol. 22, no. 10, pp. 939-943, 2012.

[43] H. Ogilvie, R. Patel, J. Leon-Abarca, E. Osei, L. Phillips, and M. Rebollo-Mesa, "The bidirectional relationship between sleep and inflammation: Implications for mental health," Front. Psychiatry, vol. 13, p. 961806, 2022.

[44] R. C. Harvey, A. Benton, E. Walker, M. Bottary, S. Lane, and V. Jordan, "A targeted lifestyle intervention to increase muscle mass and reduce fatigue: A randomized controlled trial," J. Nutr. Health Aging, vol. 24, no. 10, pp. 1117-1125, 2020.

[45] T. Young, P. E. Peppard, and D. J. Gottlieb, "Epidemiology of obstructive sleep apnea: A population health perspective," Am. J. Respir. Crit. Care Med., vol. 165, no. 9, pp. 1217-1239, 2002.