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ASSOCIATION OF SLEEP PATTERNS WITH DYSLIPIDAEMIA: A NARRATIVE REVIEW

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ABSTRACT

Background: Dyslipidaemia, a major cardiovascular risk factor, involves abnormal lipid profiles including elevated LDL-C, triglycerides, and reduced HDL-C. Emerging evidence links sleep disturbances to metabolic dysregulation, yet associations with specific sleep disorders remain underexplored. **Methods:** This narrative review synthesises evidence from five open-access observational studies (PubMed Central, up to January 2025) on sleep characteristics (duration, quality, catch-up sleep, night-shift work, OSA, insomnia) and dyslipidaemia in adults. Studies were selected using Boolean searches (e.g., "Sleep AND Dyslipidaemia"), with qualitative synthesis across domains like circadian disruption and gender effects. **Results:** Weekend catch-up sleep showed inverse dyslipidaemia associations in

optimal sleepers; night-shift work heightened risks via circadian misalignment. Severe OSA linked to hypercholesterolaemia and low HDL-C primarily in males, potentially moderated by oestrogen. Poor sleep quality correlated with adverse lipids in diabetics; insomnia lacked direct ties except elevated LDL-C in hypnotic users. **Conclusions:** Sleep duration, OSA, and shift work emerge as heterogeneous dyslipidaemia correlates with sex-specific effects. Longitudinal studies are needed for causality and prevention.

Keywords: circadian disruption; dyslipidaemia; insomnia; obstructive sleep apnoea; sleep duration

INTRODUCTION

Blood lipids comprise fatty substances circulating freely in the plasma or bound to other molecules. They play essential roles in energy storage, maintenance of cell membrane integrity, cytokine synthesis, signal transduction, and regulation of basal metabolic processes **[1]**. Dyslipidaemia is characterized by elevated plasma levels of low-density lipoprotein (LDL) cholesterol or triglycerides, reduced levels of high-density lipoprotein (HDL) cholesterol, alterations in total cholesterol, or any combination of these abnormalities. It constitutes a major modifiable risk factor for cardiovascular diseases, including ischemic heart disease and stroke, with elevated plasma LDL-cholesterol being particularly implicated **[2]**. Metabolic syndrome has emerged as a significant global health concern, with atherosclerosis representing one of its key clinical manifestations. Epidemiological data suggest that approximately 80% of atherosclerosis cases are attributable to dyslipidaemia, which is strongly linked to the onset and progression of cardiovascular complications **[3]**. Adequate restorative sleep is fundamental to human health; nevertheless, its precise role in regulating metabolic homeostasis remains incompletely understood **[4]**. Sleep disorders represent one of the most prevalent clinical conditions. With the increasing public awareness of sleep health and its associated disorders, issues related to sleep are emerging as a significant focus within both general internal medicine and various medical specialities **[5]**.

Circulating lipid concentrations are significantly modulated by lifestyle determinants such as dietary patterns, smoking habits, and levels of physical activity. However, the potential association between sleep duration and quality with serum lipid profiles remains to be fully elucidated **[6]**. Physiologically, sleep restriction has been shown to induce hormonal perturbations that favour the development of an atherogenic lipid profile, characterized by elevated cortisol and ghrelin concentrations, decreased leptin

levels, and altered sympatho-vagal balance **[7]**. According to the American Academy of Sleep Medicine, sleep disorders are classified into six major categories: (1) insomnia, (2) sleep-related breathing disorders, (3) central disorders of hypersomnolence, (4) circadian rhythm sleep–wake disorders (CRSWDs), (5) sleep-related movement disorders, and (6) parasomnias **[8]**. Nonetheless, existing literature has explored the association between dyslipidaemia and only two of these sleep disorder categories, underscoring the need for further investigation into this relationship.

MATERIALS AND METHODS-

This review focuses on analysing the existing relationship between dyslipidaemia and sleep. **(Figure 1)**

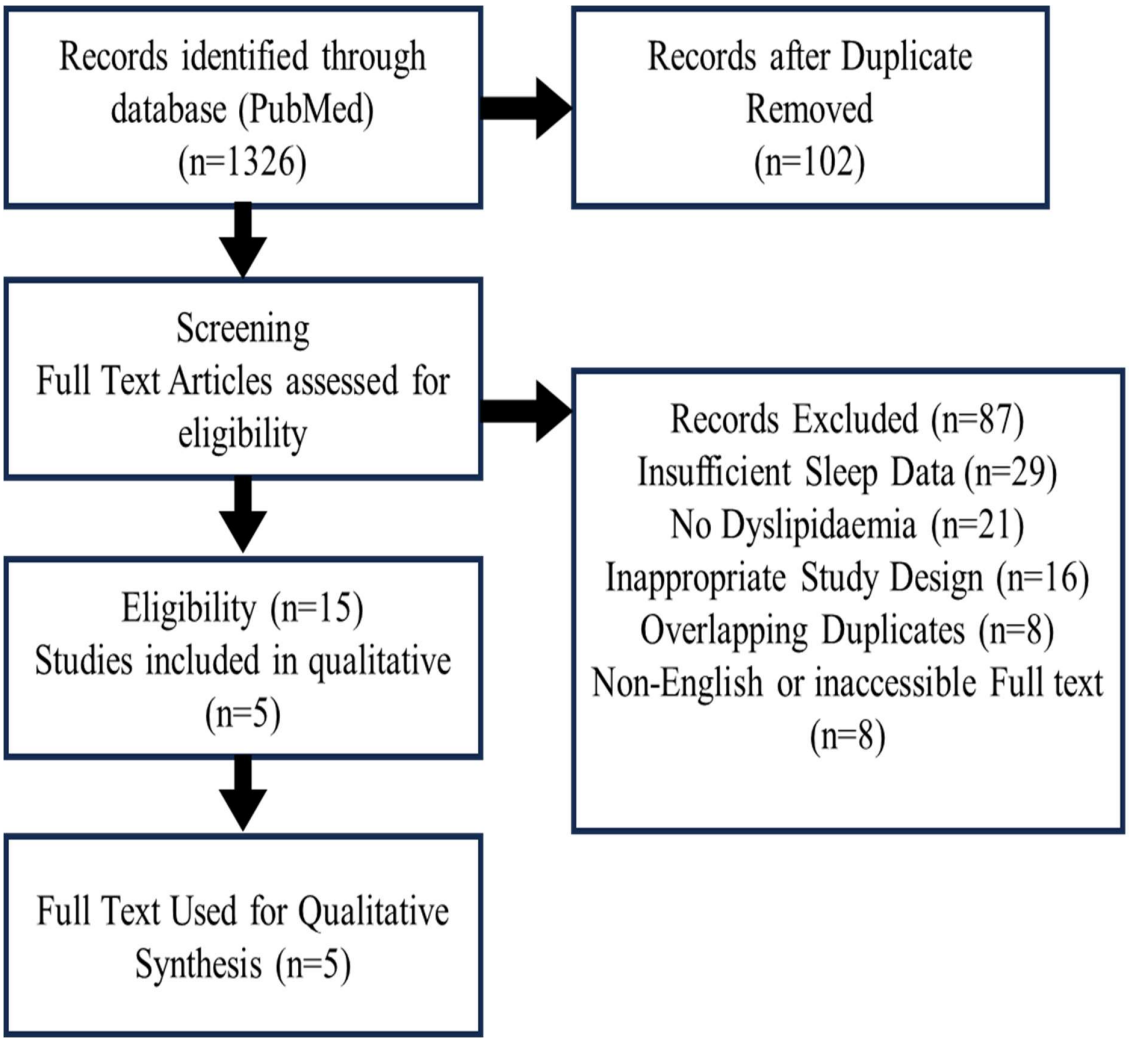

Figure 1: Review Methodology

Search Strategy

A thorough literature search was conducted in PubMed Central for studies published up until January 2025. A total of 10 studies met the inclusion criteria, of which 5 open-access studies were selected for review. The following keywords and Boolean combinations were employed: Sleep AND Dyslipidaemia, Sleep Disorders AND Dyslipidaemia, Lipid Disturbances AND Sleep, and Metabolic Syndrome AND Sleep. Additionally, reference lists of the selected articles were examined for further relevant studies.

Study Design

This narrative review was performed to synthesise and integrate current evidence regarding the association between sleep characteristics and dyslipidaemia in adults. The narrative methodology was selected to facilitate the comparison of findings across diverse study designs, populations, and methods of sleep assessment.

Eligibility Criteria

Studies were included if they m*et al*l of the following criteria:

1. **Population:** Adults (≥18 years) from community, hospital, or occupational settings.
2. **Exposure:** Any sleep-related factor, including sleep duration, insomnia, sleep quality, weekend catch-up sleep, night-shift work, or obstructive sleep apnoea.
3. **Outcome:** Dyslipidaemia or individual lipid parameters (TC, LDL-C, HDL-C, TG).
4. **Design:** Cross-sectional, cohort, case–control, or clinical observational studies.
5. **Measurement:** Objective or validated subjective sleep measures and laboratory-based or guideline-defined lipid assessment.
6. **Language:** Articles published in English.

Exclusion Criteria

The following categories of studies were excluded from consideration: reviews, editorials, case reports, paediatric studies, those lacking lipid outcome data, and investigations focused solely on genetic dyslipidaemias.

Sources of Evidence Included

Five full-text studies provided by the author, which fulfilled eligibility criteria, were used as core evidence in the review. The studies are listed in **Table 1**.

Table 1: Studies Included in the Review

S.No.	Title of the Study	PMID/PMCID	Year of Publication	Place of Study Conducted
1.	Association between weekend catch-up sleep and dyslipidaemia among Korean workers. [9]	PMID: 36650276 PMCID: PMC9845206	2023	Korea
2.	Association between night work and dyslipidaemia in South Korean men and women: a cross-sectional study [10]	PMID: 30922333 PMCID: PMC6440094	2019	Korea

3.	**Effect modification by gender of the influence of obstructive sleep apnoea characteristics on dyslipidaemia in China: a cross-sectional study. [11]**	**PMID: 31488475 PMCID: PMC6731808**	**2019**	**China**
4.	**The correlation between stress score. Quality and duration of sleep with lipid profile and glycaemic status. [12]**	**PMID: 39400097**	**2024**	**India**
5.	**Insomnia Symptoms Are Not Associated with Dyslipidaemia: A Population-Based Study [13]**	**PMID: 26612387 PMCID: PMC4763367**	**2016**	**Canada**

Data Extraction

A structured data-extraction template was used to collect the following information from each included article:

1. **Study characteristics:** author, year, country, design, sample size
2. **Participant characteristics:** age range, gender distribution, comorbidities
3. **Sleep exposure variables:** self-reported sleep duration, sleep quality (PSQI), insomnia symptoms, weekend catch-up sleep (weekday vs weekend difference), shift-work schedules and night-work definitions and polysomnography-derived indices (AHI, ODI, MAI)
4. **Outcome measures:** dyslipidaemia definitions (cut-offs for TC, LDL-C, HDL-C, TG)
5. **Confounding variables:** BMI, smoking, alcohol, socioeconomic factors, diabetes, hypertension, physical activity
6. **Statistical methods used in each study:** Data extraction was performed manually based on full-text review.

Quality Assessment

Given the narrative design, formal scoring was not mandated; however, all studies were qualitatively assessed based on:

1. Appropriateness of sleep measurement tools (PSQI-Pittsburgh Sleep Quality Index, NHANES questionnaires, Polysomnography)
2. Validity of lipid assessment methods and guideline cut-offs
3. Adequacy of confounder adjustments
4. Risk of bias related to self-reported sleep, cross-sectional design, or selection bias
5. Sample size sufficiency and representativeness

Studies using objective sleep assessments (e.g., PSG in OSA research) were considered higher quality than those relying solely on self-report.

Data Synthesis Approach

Due to the variability in sleep metrics, study cohorts, and statistical methodologies among the studies reviewed, conducting a meta-analysis was not feasible. Consequently, a qualitative narrative synthesis was employed to integrate the findings. Findings were organized into the following conceptual domains:

1. Sleep duration and catch-up sleep
2. Night-shift work and circadian disruption
3. Insomnia and sleep quality

4. Obstructive sleep apnoea and intermittent hypoxia
5. Population-specific effects (gender, metabolic status, occupation)

Within each domain, results were compared and contrasted, and biological plausibility was explored based on mechanisms proposed in the literature.

RESULTS

Overview of Included Studies

A synthesis of five observational studies examined the relationship between sleep patterns and dyslipidaemia: (1) weekend catch-up sleep among Korean workers (national survey), (2) night shift work and its association with dyslipidaemia (KNHANES), (3) a gender-stratified analysis of obstructive sleep apnoea (OSA) using polysomnography (hospital cohort in China), (4) correlations between sleep quality, stress, and lipid levels in diabetic and pre-diabetic patients (Indian hospital sample), and (5) insomnia symptoms and lipid profiles in a US population sample (NHANES). The observations are reported in **Table 2**.

Table 2: Review Results

Study (Year, Country)	Population & Design	Sleep Exposure	Dyslipidaemia Measures	Key Results	Notable Subgroup Findings
Weekend Catch-Up Sleep and Dyslipidaemia (2023, Korea)	N = 4,085 working adults; KNHANES; cross-sectional	Weekend catch-up sleep (CUS = weekend − weekday sleep)	Fasting TC, LDL-C, HDL-C, TG; Korean guideline cut-offs	CUS was protective in men: OR 0.76 (95% CI 0.61–0.95). No significant association in women.	Men with 7–8 h total sleep, night workers, and white-collar workers benefited most. CUS ≤2 h provided the strongest protective effect.
Night Work and Dyslipidaemia (2019, Korea)	N = 5,813 adults ≥30 y; KNHANES; cross-sectional	Night work (6 PM–12 AM; 12 AM–8 AM)	Fasting TC, HDL-C, LDL-C, TG	Men: night work increased dyslipidaemia odds (OR 1.53; 95% CI 1.05–2.24). Women: no overall association.	Male night workers who skipped meals or slept <7 h had highest risk. Female white-collar night workers had nearly 3-fold increased risk (OR ≈ 2.95).
Gender-Modified Effects of OSA on Dyslipidaemia (2019, China)	N = 3,760; hospital-based; full polysomnography	OSA severity: AHI, ODI, MAI	Hyper-TC; Hypo-HDL-C; Hyper-LDL-C; TC/HDL ratio	Severe OSA (AHI >30) and high ODI were associated with worse lipid profiles in men only. Significant increase in hyper-TC, hypo-HDL, and high TC/HDL ratio among males.	Positive additive and multiplicative interactions between male sex and high AHI/ODI. Little to no dyslipidaemia effect in females.
Sleep Quality, Stress, and Lipids in Diabetic/Prediabetic Patients (2024, India)	N = 90 (45 diabetics, 45 prediabetics); hospital-based	PSQI sleep quality; sleep duration; Perceived Stress Scale	Fasting TC, LDL-C, HDL-C, TG; TC:HDL ratio	In diabetics: TC and TC:HDL ratio varied with sleep quality and stress; TG significantly higher vs prediabetics. In prediabetics: HDL correlated with HbA1c; sleep duration varied with stress.	Illustrates sleep–lipid–glycaemic interactions but limited by small sample size. Findings are exploratory.
Insomnia Symptoms and Dyslipidaemia (NHANES) (2016)	Large population sample; NHANES 2005–2008	Insomnia symptoms ≥5×/month	LDL-C, HDL-C, TG	No significant association between insomnia symptoms and dyslipidaemia after adjustments.	Insomnia + sleeping pill use was linked to higher LDL-C (OR 2.18; 95% CI 1.14–4.15).

DISCUSSION

Reduced sleep duration and poor sleep quality have been identified as potential risk factors for adverse physical and psychological health outcomes **[14]**. Workers who maintained a total sleep duration of 7–8 hours, particularly night-shift employees and white-collar workers exhibiting catch-up sleep (CUS) behaviour, demonstrated a relatively lower risk of dyslipidaemia compared with those without CUS **[15]**. Conversely, evidence suggests that excessively prolonged sleep may have detrimental effects on various health outcomes **[16]**. To counterbalance weekly sleep deficits, individuals often engage in weekend CUS, a behavioural adaptation associated with a reduced prevalence of hypertension, obesity, and elevated serum high-sensitivity C-reactive protein concentrations **[17]**. In the referenced investigation, workers who exhibited CUS

and maintained an optimal weekday sleep duration (7–8 hours) demonstrated an inverse association with dyslipidaemia relative to those with suboptimal sleep durations (<7 hours or >8 hours) **[18]**. Furthermore, prior research indicates that abnormal sleep duration during weekdays is correlated with increased mortality risk among individuals younger than 65 years **[19]**. Night-shift work has been observed to have a stronger association with dyslipidaemia compared to day or other shift work patterns **[20]**. Chronic sleep deprivation adversely affects metabolic regulation, promoting the development of an atherogenic lipid profile. Conversely, habitual excessive sleep may elevate mortality risk, and with greater circadian misalignment, the compensatory benefits of extended sleep may become attenuated **[21]**. The study demonstrated a positive association between weekend catch-up sleep and dyslipidaemia. However, the findings have several limitations. Definitive conclusions regarding the relationship between catch-up sleep, dyslipidaemia, and female workers cannot be drawn from this investigation, as these aspects were not specifically addressed. Moreover, factors such as individual sleep quality, metabolic variation, occupational stress, and dietary habits may have acted as potential confounders, thereby influencing the observed outcomes.

In male participants, an increased risk of dyslipidaemia was observed. Physiological processes, including eating behaviour, lipid, carbohydrate, and glucose metabolism, as well as sleep regulation, follow circadian rhythms governed by the endogenous biological clock **[22]**. Disruption of this circadian system, as seen in night-shift work, has been linked to obesity, impaired insulin secretion, and dysregulation of glucose homeostasis **[23]**. Furthermore, shared pathophysiological pathways between insulin resistance and atherosclerosis, an established consequence of dyslipidaemia, have been identified. These include hyperglycaemia, elevated free fatty acids leading to oxidative stress, activation of pro-inflammatory cascades, reduced HDL levels, and increased triglycerides **[24]**.

The circadian clock plays a pivotal role in regulating lipid metabolism, and periodic disturbances in circadian rhythm can adversely affect lipid homeostasis. Consequently, night-shift work exhibits a stronger association with dyslipidaemia compared with day or rotating shifts **[25]**. Sleep deprivation further exacerbates metabolic dysregulation, impairing homeostatic control of macronutrient intake (proteins, fats, and carbohydrates) **[26]** and contributing to the development of an atherogenic lipid profile **[27]**. Additionally, age demonstrates a positive correlation with

the prevalence of dyslipidaemia in women, particularly among postmenopausal women above 50 years. This may be attributed to menopause-related alterations in lipoprotein composition, including increased total and low-density lipoprotein (LDL) cholesterol levels **[28]**.

The present study acknowledges several limitations. The cross-sectional design precluded the establishment of causal relationships between night work and dyslipidaemia. Furthermore, the influence of night shift work on dyslipidaemia among female participants could not be conclusively determined. The study emphasized the potential impact of irregular eating habits, such as meal skipping and binge eating during night shifts, on lipid abnormalities among night-shift workers **[29]**. Severe obstructive sleep apnoea (OSA), as indicated by an apnea-hypopnea index (AHI) greater than 30, was associated with elevated risks of hypercholesterolemia (hyper-TC), reduced high-density lipoprotein cholesterol (hypo-HDL-C), and an increased hyper-TC/HDL-C ratio, but these associations were observed only in males. One of the key pathophysiological features of OSA, intermittent hypoxia, reflected by the oxygen desaturation index (ODI), was similarly correlated with elevated hypo-HDL-C status and a higher hyper-TC/HDL-C ratio in males, but not in females **[29]**. Additive interaction analyses demonstrated synergistic effects between male sex and AHI on hypo-HDL-C and hyper-TC/HDL-C ratios. Additionally, both multiplicative and additive interactions were identified between male sex and ODI in relation to the hyper-TC/HDL-C ratio, suggesting that male gender amplifies the adverse effects of severe OSA and intermittent hypoxia on lipid metabolism **[30]**. Collectively, these findings indicate that the impact of severe OSA and intermittent hypoxia on dyslipidaemia is more pronounced in males compared to females.

Oestrogen may confer protective effects against dyslipidaemia in females by modulating hypoxia-related pathways. Intermittent hypoxia elevates hepatic levels of hypoxia-inducible factor-1α (HIF-1α), which subsequently activates sterol regulatory element-binding protein 1 (SREBP-1) and stearoyl-coenzyme A desaturase-1 (SCD-1), thereby promoting hepatic fatty acid synthesis and increasing circulating lipid levels, including triglycerides and cholesterol **[31]**. Previous rodent studies have shown that oestrogen downregulates HIF-1α expression, potentially mitigating the effects of intermittent hypoxia on lipid metabolism in females **[32]**. Moreover, males with OSA typically exhibit a greater volume of visceral adipose tissue (VAT) compared to females with OSA, even when body mass index

(BMI) and waist circumference (WC) are similar. Excess VAT is strongly correlated with an atherogenic dyslipidaemia profile **[33]**.

This study attempts to elucidate the relationship between sleep apnea and dyslipidaemia among males and females. The authors report that females exhibit a lower predisposition to dyslipidaemia compared to males, possibly due to the protective effects of estrogen. However, the study does not elaborate on age-related changes in estrogen levels, menopausal transition, or their potential influence on the interplay between sleep apnea and dyslipidaemia **[34][35]**. While evaluating alterations in lipid parameters concerning stress and sleep among diabetic participants, the research demonstrated significant variations in total cholesterol (TC), low-density lipoprotein (LDL), and the TC:HDL ratio with sleep quality, and a significant variation of TC with stress levels. No significant correlations were observed in the prediabetic cohort. A separate investigation concluded that poor sleep was associated with elevated TC and triglyceride (TG) levels, identifying sleep duration and Pittsburgh Sleep Quality Index (PSQI) scores as independent predictors of TC and LDL cholesterol. The proposed mechanisms underlying these associations include heightened sympathetic nervous system activity, increased cortisol secretion, reduced leptin levels, and diminished insulin sensitivity **[36]**.

Insomnia symptoms were generally not found to be associated with dyslipidaemia. Negative associations persisted across analyses considering symptom frequency, daytime fatigue, or short sleep duration, all indicators of insomnia severity. A distinctive finding, however, revealed that individuals with insomnia symptoms who used hypnotic medications were 118% more likely to exhibit elevated LDL-C levels after adjusting for confounding variables **[37]**. Despite these findings, the study did not adequately address the relationship between chronic insomnia and dyslipidaemia, as all participants reported insomnia symptoms persisting for only one month. Additionally, information regarding the duration and dosage of hypnotic use, participant age, and existing comorbidities was not provided.

CONCLUSION

Sleep duration, circadian disruption, OSA-related intermittent hypoxia, and stress-related sleep disturbance collectively emerge as important, but heterogeneous, correlates of dyslipidaemia in adults. Evidence suggests sex-specific and metabolic-context–dependent effects, while insomnia per se shows largely null associations, except among hypnotic users with elevated LDL-C. Larger longitudinal and mechanistic studies are required to

clarify causality and guide targeted sleep-based dyslipidaemia prevention strategies.

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Conflicts of Interest

There are no conflicts of interest.

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