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Association between Sleep Duration and Dyslipidemia: Insights from the Fasa Adults Cohort Study (FACS), 2014–2021

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Abstract

Background & Objectives: Sleep duration is increasingly recognized as a critical determinant of overall health, particularly in relation to cardiovascular risk. Dyslipidemia, a leading contributor to cardiovascular disease, has been linked inconsistently to sleep duration, especially among rural populations.

Materials & Methods: This population-based cohort study utilized data from the Fasa Adults Cohort Study (2014–2021), comprising 10,118 adults aged 35 to 70 years from the rural districts of Sheshdeh and Qarabolagh, Fasa County, Iran. Participants were categorized based on self-reported sleep duration (**<7** hours vs. ≥7 hours). Anthropometric measurements, blood pressure, and fasting lipid profiles were collected. Dyslipidemia was defined according to Adult Treatment Panel III (ATP III) criteria. Statistical analyses included chi-square tests, t-tests, and multivariable logistic regression.

Results: Individuals with short sleep duration (<7 hours) exhibited significantly higher body mass index (BMI), waist circumference, and low-density lipoprotein cholesterol (LDL-C) levels, as well as lower high-density lipoprotein cholesterol (HDL-C) levels (all p-values < 0.001). Short sleep duration was significantly associated with increased odds of dyslipidemia (adjusted odds ratio [OR] = 1.119; 95% confidence interval [CI]: 1.024–1.223; p = 0.013) and low HDL-C (adjusted OR = 1.239; 95% CI: 1.140–1.347; p < 0.001). No significant associations were observed with total cholesterol, triglycerides, or LDL-C.

Conclusion: Short sleep duration is significantly associated with dyslipidemia and adverse lipid profiles, particularly reduced HDL-C levels. These findings highlight the importance of sleep health in mitigating cardiovascular risk, especially in underserved rural populations. Promoting sufficient sleep may serve as a viable preventive strategy against metabolic disorders. Further longitudinal studies are warranted to clarify underlying causal mechanisms.

Keywords: Sleep Duration, Dyslipidemia, Cohort Study, Cardiovascular Risk, Metabolic Health

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Introduction

Sleep is a critical determinant of health, and its duration, along with its quality, are essential for maintaining overall well-being (1). Various factors—ranging from cultural, social, and psychological to pathophysiological and environmental—influence sleep patterns.

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Numerous studies have linked both short and long sleep durations to adverse health outcomes, including cardiovascular disease (CVD) (2, 3), diabetes mellitus (DM) (4), hypertension (HTN) (5), obesity (6), psychiatric disorders (7), and mortality (2). Thus, sleep duration serves as an important variable in assessing health risks and should be examined in relation to conditions such as dyslipidemia, a significant risk factor for CVD.

Dyslipidemia is characterized by an abnormal lipid profile—namely, high triglycerides, elevated total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and low high-density lipoprotein cholesterol (HDL-C)—and is a well-established risk factor for atherosclerosis, contributing significantly to CVD (8, 9). Several studies have explored the relationship between sleep duration and lipid abnormalities, indicating potential associations with conditions such as hypercholesterolemia (10), hyperlipidemia (11), and variations in LDL-C and HDL-C levels (12, 13). However, other research has not identified significant connections (14, 15), suggesting a complex and inconsistent relationship that merits further investigation.

The growing concern over sleep duration and its impact on public health is underscored by studies highlighting its influence on various chronic conditions. For example, in China, the prevalence of dyslipidemia has surged in recent years, with studies indicating that approximately 40.4% of adults are affected by this condition (16). Since dyslipidemia is a major contributor to the morbidity and mortality associated with CVD, understanding its risk factors—including sleep duration—is essential for developing effective prevention strategies. Similar concerns have emerged globally, as inadequate sleep—both in terms of quality and quantity—has been linked to increased risks of diseases such as obesity, HTN, and metabolic syndrome (17).

The existing literature on sleep duration and dyslipidemia presents conflicting findings, with some studies observing U-shaped associations, while others suggest that only short or long sleep durations may be linked to dyslipidemia (15, 18). A meta-analysis of 137 prospective cohort studies involving more than 5 million participants did not establish a clear association between sleep duration and dyslipidemia (14). Moreover, limited research has examined the influence of sleep quality and potential gender-specific associations with lipid profiles (19-21), revealing gaps in the literature that require further exploration.

The need for additional research is particularly evident in rural populations, where lifestyle factors and access to healthcare services often differ from those in urban settings. Since sleep patterns can vary substantially depending on geographic location and socio-cultural influences, it is essential to investigate how sleep duration and quality interact with health outcomes in specific contexts. The Fasa region of Iran, with its distinct rural demographic, presents a valuable opportunity to examine these relationships within a large adult cohort. This study aims to assess the association between sleep duration and dyslipidemia among adults in the Fasa Cohort, comprising over 10,000 individuals aged 35 to 70 years. By examining these associations, the study will offer critical insights into the role of sleep in lipid metabolism and cardiovascular risk among rural populations, thereby informing targeted public health strategies and interventions.

Material and Methods

Study Design and Population

This cross-sectional, descriptive-analytical study was conducted using baseline data from the population-based Fasa Cohort Study (FACS), which commenced in 2014 and continued through 2021. A total of 10,118 adults aged 35 to 70 years from the Sheshdeh and Qarabolagh districts of Fasa County, southern Iran, were included. A random sampling method was employed, and

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written informed consent was obtained from all participants.

To ensure the sample was representative of the target population, rigorous inclusion and exclusion criteria were established. Eligible participants were required to be Iranian nationals, residents of the target region for at least one year, aged between 35 and 70 years, capable of verbal communication, and willing to provide informed consent. Exclusion criteria applied to individuals who failed to attend after three phone invitations or had severe medical conditions that could impede participation or influence study outcomes. These criteria were carefully formulated to enhance the validity and reliability of the data, thereby ensuring an accurate representation of the health status and risk factors associated with noncommunicable diseases in rural Iran.

Informed consent procedures adhered to ethical guidelines, with all participants receiving a comprehensive explanation of the study's objectives, procedures, and confidentiality measures in alignment with the ethical principles outlined in the Declaration of Helsinki (1975, revised 2008). The study was approved by the Ethics Committee of Fasa University of Medical Sciences (Approval Code: IR.FUMS.REC.1402.168). The methodology and data collection procedures employed in this study are consistent with prior publications from the Fasa Cohort Study (22, 23).

Physical and Biochemical Analyses

Anthropometric measurements, including height, hip circumference, waist circumference (in centimeters), and weight (in kilograms), as well as blood pressure, were each measured twice at separate times by trained professionals. Demographic data, including age, sex, marital status, and sleep duration, were systematically collected through face-to-face interviews conducted by trained healthcare professionals and local health workers (Behvarz). Blood samples were obtained in the morning after an 8- to 12-hour fasting period and were immediately stored at –80°C for subsequent analysis of lipid profiles and fasting blood sugar. Serum levels of TC, LDL-C, HDL-C, and TG were determined using the enzymatic colorimetric assay. All laboratory procedures and measurement protocols adhered to the standard guidelines established within the Fasa Cohort infrastructure (22, 23).

Diagnostic Criteria

Dyslipidemia was diagnosed according to the Adult Treatment Panel III (ATP III) criteria. Individuals were classified as having dyslipidemia if they met any of the following conditions: total cholesterol (TC) ≥ 200 mg/dL, triglycerides (TG) ≥ 150 mg/dL, LDL-C ≥ 130 mg/dL, or HDL-C < 40 mg/dL for men or < 50 mg/dL for women. Sleep duration was assessed through a validated self-report questionnaire, capturing both nighttime and daytime sleep durations. Total sleep duration over a 24-hour period was calculated by summing these values. Sleep duration was then categorized into two groups: less than 7 hours and 7 hours or more. DM was diagnosed based on self-reports and/or fasting blood glucose levels >120 mg/dL. High blood pressure was defined as a reading exceeding 120/80 mm Hg. These criteria ensured a standardized approach to the diagnosis of these health conditions.

Data Analysis

Descriptive statistics were used to summarize the demographic and clinical characteristics of the study participants by gender and sleep duration. Continuous variables were expressed as means ± standard deviations (SDs), and categorical variables were presented as frequencies and percentages. Comparisons of continuous variables between males and females were conducted using independent-samples t-tests, while chi-square tests were employed to assess differences in categorical variables. To investigate the association between sleep duration and various health factors, multiple logistic regression models were applied.

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These models were adjusted for potential confounders, including age, gender, BMI, waist circumference (WC), hip circumference (HC), marital status (categorized as: 1. single, 2. married, 3. widowed, 4. divorced), DM, and HTN. Odds ratios (ORs) with 95% confidence intervals (CIs) were reported to evaluate the strength of associations between sleep duration and health outcomes. For continuous variables such as total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), linear regression analyses were conducted. Statistical significance was defined as a p-value < 0.05 for all tests. All analyses were performed using SPSS software.

Results

The study included 10,118 participants, categorized into two groups based on self-reported sleep duration: those sleeping less than 7 hours (n = 5,905) and those sleeping 7 hours or more (n = 4,203). Table 1 presents the demographic, anthropometric, and biochemical characteristics of participants, stratified by sleep duration. Participants sleeping less than 7 hours were significantly older (49.37 ± 9.49 years) than those sleeping 7 hours or more (47.60 ± 9.61 years, $p < 0.001$). Sleep duration itself differed significantly between the groups, with the "<7 hours" group averaging 5.95 ± 1.13 hours and the "≥7 hours" group averaging 8.42 ± 0.86 hours ($p < 0.001$).

Anthropometric measurements, including body mass index (BMI) and waist circumference (WC), were significantly higher in the "<7 hours" group (BMI: 25.80 ± 4.90; WC: 93.50 ± 11.80 cm) than in the "≥7 hours" group (BMI: 25.42 ± 4.80; WC: 92.63 ± 11.78 cm, $p < 0.001$ for both). Hip circumference (HC) also differed significantly, with the "<7 hours" group exhibiting a higher mean value (99.86 ± 8.86 cm) compared to the "≥7 hours" group (99.23 ± 8.83 cm, $p < 0.001$).

Biochemical analyses revealed significant differences in lipid profiles between the two groups. Participants with less than 7 hours of sleep had higher levels of LDL-C: 108.70 ± 32.63 mg/dL) compared to those with 7 or more hours of sleep (LDL-C: 106.21 ± 33.23 mg/dL, $p < 0.001$). Conversely, HDL-C levels were significantly

Table 1. Comparison of Demographic, Anthropometric, and Biochemical Characteristics by Sleep Duration Groups

Variable	Sleep Duration Groups N (Mean ± SD)		P-Value
	Under 7 Hours	Above 7 Hours	
Age	5905 (49.37 ± 9.485)	4203 (47.60 ± 9.612)	<0.001
Sleep Duration	5905 (5.95 ± 1.13)	4203 (8.42 ± 0.86)	<0.001
BMI	5895 (25.80 ± 4.90)	4198 (25.42 ± 4.80)	<0.001
WC	5892 (93.50 ± 11.80)	4197 (92.63 ± 11.78)	<0.001
HP	5892 (99.86 ± 8.86)	4197 (99.23 ± 8.83)	<0.001
FBG	5881 (92.56 ± 28.76)	4189 (92.73 ± 30.56)	0.77
TC	5881 (184.89 ± 39.26)	4189 (185.40 ± 39.11)	0.54
TG	5881 (132.82 ± 82.82)	4189 (130.50 ± 81.98)	0.16
LDL	5881 (108.70 ± 32.63)	4188 (106.21 ± 33.23)	<0.001
HDL	5881 (49.59 ± 14.15)	4189 (53.04 ± 17.89)	<0.001

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lower in the "<7 hours" group (49.59 ± 14.15 mg/dL) than in the "≥7 hours" group (53.04 ± 17.89 mg/dL, $p < 0.001$). No significant differences were observed in fasting blood glucose (FBG), TC levels between the two groups ($p > 0.05$ for all). These findings suggest that shorter sleep duration is associated with older age, higher BMI, larger waist and hip circumferences, and less favorable lipid profiles, particularly lower HDL-C and higher LDL-C levels.

The table presents the means ± standard deviations (SDs) for age, sleep duration, BMI, waist circumference (WC), hip circumference (HC), FBG, TC, triglycerides, LDL, and HDL among participants, stratified by sleep duration: less than 7 hours (n = 5,905) and 7 hours or more (n = 4,203). P-values were calculated using independent-samples t-tests to compare the means between the two groups. Significant differences ($p < 0.05$) were observed for age, sleep duration, BMI, WC, HC, LDL, and HDL, while no significant differences were found for FBG, TC, or TG. Table 2 displays the distribution of dyslipidemia, gender, marital status, DM, and HTN across the two sleep duration groups.

Dyslipidemia was significantly more prevalent in the <7 hours group (69.5%) compared to the ≥7 hours group (66.9%, $p = 0.007$), suggesting that shorter sleep duration is associated with a greater prevalence of dyslipidemia.

Gender distribution also differed significantly between the groups ($p < 0.001$). The <7 hours group had a higher proportion of males (47.2%) than the ≥7 hours group (42.3%), while the proportion of females was higher in the ≥7 hours group (57.7%) compared to the <7 hours group (52.8%). Marital status varied significantly between the two groups ($p < 0.001$). The ≥7 hours group had a higher proportion of participants in Status 1 (single) (4.9% vs. 2.8%) and Status 4 (divorced) (1.2% vs. 0.9%), whereas the <7 hours group had slightly higher proportions of participants in Status 2 (married) (89.5% vs. 88.1%) and Status 3 (widowed) (6.7% vs. 5.8%). The prevalence of DM was marginally higher in the <7 hours group (12.7%) compared to the ≥7 hours group (11.7%, $p < 0.001$). Similarly, the prevalence of HTN was significantly higher in the <7 hours group (21.5%) relative to the ≥7 hours group (18.0%, $p < 0.001$).

These findings indicate that shorter sleep duration is associated with a higher prevalence of dyslipidemia, DM, and HTN, as well as notable differences in gender and marital status distributions.

The table displays the frequencies and percentages of categorical variables, including dyslipidemia (yes/no), gender (male/female),

Table 2. Distribution of Dyslipidemia, Gender, Marital Status, Diabetes Mellitus, and Hypertension by Sleep Duration Groups

Variable	Category	Sleep Duration		P-Value
		Under 7 Hours	Above 7 Hours	
Dyslipidemia	No	1793 (30.5%)	1385 (33.1%)	0.007
	Yes	4088 (69.5%)	2804 (66.9%)	
Gender	Male	2787 (47.2%)	1777 (42.3%)	<0.001
	Female	3118 (52.8%)	2426 (57.7%)	
Marital Status	Status 1	168 (2.8%)	205 (4.9%)	<0.001
	Status 2	5286 (89.5%)	3704 (88.1%)	
	Status 3	398 (6.7%)	245 (5.8%)	
	Status 4	53 (0.9%)	49 (1.2%)	
DM	No	5152 (87.3%)	3707 (88.3%)	<0.001
	Yes	751 (12.7%)	493 (11.7%)	
HTN	No	4634 (78.5%)	3442 (82.0%)	<0.001
	Yes	1269 (21.5%)	758 (18.0%)	

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Table 3. Adjusted Odds Ratios (OR) and 95% Confidence Intervals (CI) for Associations Between Sleep Duration and Lipid Profile Variables

Variable	95% C.I.	P-Value
Dyslipidemia	1.119 (1.024–1.223)	0.013
TC	0.924 (0.846–1.008)	0.075
TG	1.067 (0.972–1.170)	0.174
LDL	1.042 (0.947–1.147)	0.401
HDL	1.239 (1.140–1.347)	<0.001

marital status (classified into four categories: 1. single, 2. married, 3. widowed, 4. divorced), DM (yes/no), and HTN (yes/no). P-values were calculated using chi-square tests to assess differences in proportions between the two sleep duration groups. Statistically significant differences ($p < 0.05$) were identified for dyslipidemia, gender, marital status, DM, and HTN. The study examined the associations between sleep duration and lipid profile variables using multiple logistic regression models, adjusted for potential confounders, including age, gender, marital status, DM, and HTN. Table 3 presents the adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for dyslipidemia and individual lipid profile components.

Dyslipidemia was significantly associated with shorter sleep duration, with an adjusted OR of 1.119 (95% CI: 1.024–1.223, $p = 0.013$). This indicates that participants who slept less than 7 hours had approximately 11.9% greater odds of having dyslipidemia compared to those who slept 7 hours or more. Among the lipid profile components, high-density lipoprotein cholesterol (HDL) showed a statistically significant inverse association with shorter sleep duration, with an adjusted OR of 1.239 (95% CI: 1.140–1.347, $p < 0.001$). This finding suggests that reduced sleep duration is associated with lower HDL levels, which is typically considered an adverse lipid profile.

No significant associations were observed for total cholesterol (TC) (OR: 0.924, 95% CI: 0.846–1.008, $p = 0.075$), triglycerides (TG) (OR: 1.067, 95% CI: 0.972–1.170, $p = 0.174$), or low-density lipoprotein cholesterol (LDL) (OR: 1.042, 95% CI: 0.947–1.147, $p = 0.401$). These results indicate that sleep duration was not significantly associated with TC, TG, or LDL levels after adjusting for confounders. In summary, shorter sleep duration was significantly associated with increased odds of dyslipidemia and reduced HDL levels, but not with TC, TG, or LDL levels. These findings underscore the potential role of sleep duration as a modifiable factor influencing lipid metabolism and cardiovascular risk.

The table displays the odds ratios (ORs) along with their 95% confidence intervals (CIs), obtained from logistic regression analyses adjusted for potential confounders, including age, gender, BMI, waist circumference (WC), hip circumference (HC), marital status, diabetes, and hypertension. Statistically significant associations ($p < 0.05$) were identified for dyslipidemia and high-density lipoprotein cholesterol (HDL). However, no significant associations were observed for total cholesterol (TC), triglycerides (TG), or low-density lipoprotein cholesterol (LDL). P-values were computed to evaluate the magnitude and significance of these associations.

Discussion

This cross-sectional study of 10,118 adults aged 35–70 years from Fasa County, Iran, aimed to explore the association between sleep duration and health outcomes. Participants were categorized based on their sleep duration (<7 hours vs. ≥7 hours). Those with shorter sleep durations were older, had higher BMI, larger

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waist and hip circumferences, and less favorable lipid profiles, including elevated LDL-C and reduced HDL-C levels. Dyslipidemia, diabetes, and HTN were more prevalent among individuals with shorter sleep durations. Adjusted logistic regression analyses indicated that shorter sleep was associated with a higher likelihood of dyslipidemia (OR: 1.119, $p = 0.013$) and decreased HDL-C levels (OR: 1.239, $p < 0.001$), whereas no significant associations were found with total cholesterol, triglycerides, or LDL-C levels. These findings underscore the potentially adverse metabolic and cardiovascular effects of insufficient sleep.

A review of the literature reveals a complex relationship between sleep duration and lipid profiles, as well as the prevalence of dyslipidemia. While there is general agreement that sleep duration affects lipid metabolism, the direction and strength of this relationship vary depending on population characteristics, methodologies, and study designs. For instance, Abdurahman et al. (2020) and Kinuhata et al. (2014) both observed that longer sleep durations might influence lipid profiles, though their findings diverged regarding specific lipid components. Abdurahman et al. identified a significant association with elevated total cholesterol, whereas our study did not observe this relationship. In contrast, our research focused on shorter sleep durations (<7 hours), which were associated with higher LDL-C and lower HDL-C levels—findings consistent with Kinuhata et al., who reported protective effects of moderate to long sleep durations on triglycerides. These discrepancies may be attributed to differences in study populations (e.g., age, cultural factors), research designs (e.g., sample size, follow-up periods), or the extent of statistical adjustment (12, 24).

Kruisbrink et al. (2017) found no significant relationship between sleep duration and dyslipidemia, which contrasts with our findings, where shorter sleep was strongly associated with an increased prevalence of dyslipidemia (19). This divergence may be explained by differences in study design, such as the inclusion of a more heterogeneous participant sample in our study, as well as a more comprehensive adjustment for confounding variables. Shin et al. (2016) and Song et al. (2020) both investigated the effects of prolonged sleep on lipid profiles but did not identify significant associations with short sleep durations (20, 21). In contrast, our study found a direct link between shorter sleep and dyslipidemia, particularly in relation to LDL-C and HDL-C levels. These prior studies primarily focused on long sleep durations, while our research emphasizes the deleterious impact of short sleep on lipid metabolism and broader metabolic health risks, including HTN and diabetes.

Similarly, Tsiptsios et al. (2022) and Wang et al. (2019) identified significant associations between short sleep duration and dyslipidemia, while also highlighting the role of factors such as sleep disorders and poor sleep quality (25, 26). Our study, which focused exclusively on sleep duration, confirmed the link between shorter sleep and lipid abnormalities but did not find associations with total cholesterol or triglycerides. These results differ from those of Wang et al. (2019), who associated longer sleep durations with lipid abnormalities (26).

Zhang et al. (2022) and Zhu et al. (2023) suggested that the impact of sleep duration on lipid profiles may vary by age group or circadian sleep timing (11, 27). Our study, which focused on adults, found a significant association between shorter sleep and elevated LDL-C and decreased HDL-C levels, in alignment with findings from adult-focused studies such as those by Du et al. (2022) and Kwon et al. (2021) (18, 28). However, Kwon et al. identified a U-shaped association, suggesting that both short and long sleep durations may contribute to dyslipidemia (28). This differs from our study's emphasis on the specific risks associated with insufficient sleep.

In conclusion, while commonalities exist across studies, our findings suggest that

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short sleep duration (less than 7 hours) exerts particularly harmful effects on lipid profiles, with significant associations observed for elevated LDL-C and reduced HDL-C levels, as well as increased dyslipidemia prevalence. These results underscore the negative metabolic consequences of inadequate sleep and align with studies emphasizing the detrimental effects of short sleep, such as those by Tsiptsios et al. (2022) and Wang et al. (2019) (25, 26). Discrepancies across studies may stem from variations in how sleep duration is categorized, differences in population characteristics, and methodological approaches. The broad sample size and thorough adjustment for confounding factors in our study enhance the robustness of the observed association between short sleep and impaired lipid metabolism. Future research should prioritize standardized methodologies and aim to elucidate the biological mechanisms underlying these associations, particularly within diverse demographic populations.

The relationship between sleep duration and dyslipidemia (DL) has been extensively studied; however, findings remain inconsistent regarding its effects on cardiovascular outcomes. Both short and long sleep durations have been implicated in adverse health conditions, including CVD, as highlighted in systematic reviews (7, 29). Nonetheless, some meta-analyses have reported no direct association between sleep duration and blood lipid levels, suggesting that other mediating factors—such as obesity, HTN, and inflammation—may underlie the observed relationships between sleep patterns and cardiovascular health (30-32). These results raise important questions about whether sleep duration directly influences lipid levels or if the association is modulated through more complex physiological pathways.

Despite the lack of conclusive evidence in some studies, the possibility that sleep duration influences blood lipid levels through various biological and behavioral mechanisms remains biologically plausible (19). Sleep deprivation has been shown to alter the secretion of key metabolic hormones, such as growth hormone, cortisol, leptin, and ghrelin, which regulate hunger and energy balance. These hormonal changes can lead to modifications in eating behavior, particularly in the context of short sleep duration, during which individuals may experience increased caloric and fat intake (33, 34). Additionally, inadequate sleep can reduce physical activity, further exacerbating poor dietary choices and contributing to obesity, a well-established risk factor for dyslipidemia. As sleep deprivation is associated with both diminished physical activity and elevated energy intake, it may lead to increased adiposity and deterioration of lipid profiles (6). Short-term experimental studies support these findings, suggesting that sleep restriction may result in adverse alterations in blood lipid levels (35, 36).

The mechanisms underlying the effects of long sleep duration on health remain less well understood. While the impact of extended sleep on lipid profiles has received comparatively little attention, it is possible that long sleep duration functions more as a risk indicator rather than a direct cause of dyslipidemia. Prolonged sleep may reflect underlying health conditions, rather than exerting a direct influence on lipid metabolism. Previous studies have suggested a potential association between long sleep duration and an increased risk of CVD, indicating that hyperlipidemia could serve as a mediator of this relationship (37, 38).

Moreover, sleep duration can influence the balance of leptin and ghrelin, hormones that play a crucial role in regulating appetite and metabolism. Leptin, which suppresses appetite, has been found to decrease with insufficient sleep, while ghrelin, which stimulates hunger, increases. These hormonal shifts may lead to elevated caloric consumption, particularly of high-fat foods, thereby negatively impacting blood lipid profiles (39, 40). Furthermore,

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the detrimental effects of insufficient sleep on physical activity levels may contribute to the development of dyslipidemia. Short sleep duration is associated with reduced physical activity, which in turn may increase LDL-C levels and decrease HDL-C levels, thereby exacerbating lipid imbalances (41).

Psychological stress also plays an important role in the relationship between sleep and dyslipidemia. Sleep deprivation has been shown to elevate stress levels, which can significantly raise total cholesterol (TC) and LDL-C concentrations. Animal studies have demonstrated that chronic stress impairs lipid profiles, suggesting that the relationship between insufficient sleep and lipid dysregulation may be partially mediated by stress-induced metabolic changes (42).

In addition to hormonal and stress-related mechanisms, genetic factors may also modulate lipid profiles in response to sleep deprivation. Some studies propose that sleep restriction may interact with genetic susceptibilities, affecting lipid metabolism and indicating a complex gene-environment interaction in the development of dyslipidemia (43, 44).

Finally, although the mechanisms linking long sleep duration to hyperlipidemia remain speculative, it is worth considering that prolonged sleep may influence lipid metabolism through changes in physical activity patterns. Individuals who sleep longer may have reduced waking hours for exercise. Moreover, dietary intake patterns differ between long and short sleepers, with long sleepers generally consuming more carbohydrates and fats, potentially contributing to unfavorable lipid profiles.

In conclusion, the relationship between sleep duration and dyslipidemia is driven by a constellation of biological and behavioral mechanisms. Short sleep duration may contribute to dyslipidemia through hormonal dysregulation, altered dietary behavior, and decreased physical activity. Conversely, long sleep duration may serve as a surrogate marker for underlying health conditions that contribute to poor lipid profiles. Further research is warranted to elucidate the precise pathophysiological pathways linking sleep duration to dyslipidemia, particularly in diverse populations such as those residing in rural Iran, where cultural, environmental, and lifestyle factors may influence these associations.

Strengths and Limitations

This study possesses several strengths, including a large sample size of 10,118 participants, which enhances the generalizability of the findings to the adult population of Fasa County, Iran. The study's cross-sectional design enables a comprehensive analysis of sleep duration and its association with health outcomes in a diverse cohort encompassing a wide age range and various sociodemographic factors. Furthermore, the adjustment for potential confounders—such as BMI, waist and hip circumferences, and other lifestyle factors—enhances the validity of the findings. The study's focus on a specific rural Iranian population provides valuable insights into the health implications of sleep duration in a context that may differ substantially from urban populations, where lifestyle and environmental influences are likely to vary.

However, several limitations should be acknowledged. The cross-sectional design precludes causal inferences between sleep duration and lipid profiles. Longitudinal research is required to establish a more definitive cause-and-effect relationship. Additionally, the reliance on self-reported sleep duration may introduce recall bias, as participants may not accurately report their sleep habits. While the study adjusted for numerous confounders, unmeasured variables—such as genetic predispositions or specific sleep disorders—may still influence the results. Moreover, the study focused solely on sleep duration, without accounting for other aspects of sleep quality, which could have provided a more nuanced understanding of the sleep-health relationship. Finally, the findings

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may not be directly generalizable to other populations with differing cultural or lifestyle characteristics.

Conclusion

In conclusion, this study provides compelling evidence that short sleep duration is associated with dyslipidemia and unfavorable lipid profiles, particularly characterized by elevated LDL-C and reduced HDL-C levels. These findings underscore the importance of adequate sleep for metabolic health, emphasizing the detrimental effects of insufficient sleep on cardiovascular and metabolic outcomes. While the results are consistent with those of previous studies examining short sleep duration, discrepancies among studies highlight the complexity of this relationship, which may be modulated by differences in study design, population characteristics, and confounding variables. The large sample size and rigorous adjustments for potential confounders bolster the validity of these findings; however, further research—particularly prospective longitudinal studies—is necessary to confirm causality and elucidate the underlying biological mechanisms. Future investigations should also explore the roles of sleep quality, genetic factors, and environmental and lifestyle influences across diverse populations, including those in rural settings. Overall, these findings contribute to a deeper understanding of the metabolic consequences of insufficient sleep, particularly among populations at heightened risk for CVD.

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Conflict of Interests

The authors declare no conflicts of interest.

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Ethical Considerations

Informed consent was obtained from all participants, who were thoroughly briefed on the study's objectives, procedures, and confidentiality protocols, in accordance with the ethical standards of the Declaration of Helsinki (1975, revised 2008). The study received approval from the Ethics Committee of Fasa University of Medical Sciences.

Code of Ethics

IR.FUMS.REC.1402.168

Authors' Contributions

Zahra Rahpeyma: Investigation

Susan Darroudi: Conceptualization, Methodology

Mohammad Ebrahim Astaneh: Investigation, Writing - Review & Editing

Narges Fereydouni: Conceptualization, Writing - Original Draft

Data Availability Statement

All data generated during the current study are available from the corresponding author on reasonable request.

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