ORIGINAL ARTICLE

Investigating the Role of Short and Long Sleep Durations in Cardiovascular Diseases: Results from Kharameh PERSIAN Cohort Study in Iran

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Abstract

Background: Cardiovascular Diseases (CVDs) are now among the most common chronic conditions and are the leading cause of disability, morbidity, and mortality worldwide. Also, sleep duration is considered a neglected modifiable risk factor for CVDs. Therefore, this study aimed to investigate the association between sleep duration and the prevalence of CVD in a large scale of the Iranian population.

Methods: In this cross-sectional study, baseline data from 9,632 participants aged 40 to 70 years were collected between 2014 and 2016 and analyzed. Sleep duration was categorized into five groups (<6, 6–6.9, 7–7.9, 8–8.9, and ≥9 hours). The association between categorized sleep duration and the prevalence of CVD was assessed using both crude and adjusted multiple logistic regression models. In addition, a sensitivity analysis was performed.

Results: In the crude analysis, both males and females who slept <6 hours showed similar odds ratios for CVD (males: OR=1.42, P=0.046; females: OR=1.44, P<0.001). In the adjusted model, both short (<6 hours) and long (≥9 hours) sleep durations were associated with increased odds of CVD for both genders, reaching statistical significance exclusively in females. A sensitivity analysis was then performed, which resulted in modest attenuation of the associations in both genders.

Conclusion: Overall, our findings indicate that both short and long sleep duration are associated with an increased risk of CVD, particularly among females. These results emphasize the importance of maintaining a balanced sleep duration for cardiovascular health and support the potential benefit of sleep-related interventions in reducing CVD risk.

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Introduction

With the global epidemiological transition toward Non-Communicable Diseases (NCDs), the prevalence of cardiovascular diseases (CVDs) has increased significantly. CVDs are now among the most prevalent chronic conditions and serve as major determinants of morbidity, mortality, and disability on a global scale.[1,2] In 2020, CVDs were estimated to account for approximately 19 million deaths, representing 32% of all global deaths.[3] Moreover, 11 million deaths from CVDs were recorded in Asia in 2019[4] and more than 165 thousand deaths in

Iran, which is about 46% of the total deaths.[5]

The etiology of CVDs is multifactorial, with genetics and environmental factors involved, such as gender, age, physical activity, smoking, lipid profiles, blood pressure, type 2 diabetes, and obesity which represent well-recognized risk factors for CVD. One of the modifiable risk factors for CVDs is sleep disorders,[6] which are strongly influenced by lifestyle.[7] Any sleep disorder, such as short and long sleep duration, has been linked to an elevated risk of CVD-related mortality.[8, 9] However, due to differences in population structures, studies have reported contradictory results for the type of relationship between sleep duration and CVD risk. Sleep duration may influence CVD risk through its effects on biological processes. Short sleep may lead to metabolic and hormonal dysregulation, while prolonged sleep may reflect underlying comorbidities that predispose individuals to CVD.[10] Several studies have demonstrated a relationship between sleep duration and all-cause mortality in both sexes. The Whitehall II cohort study found a U-shaped relationship between sleep duration and CVD for males and females.[11] A study in Japan reported that short sleep (<7 hours) was significantly associated with increased CVD risk.[12] An Iranian study demonstrated a nearly 31% rise in the prevalence of cardiovascular diseases among men with short sleep duration (<6 hours per night), and by about 19% in those with longer than 9 hours sleep durations.[13]

CVD remains a major and growing public health concern that is increasing rapidly in Mediterranean and Middle Eastern countries, especially Iran.[14] The incidence and prevalence of CVDs and their risk factors differ across populations, potentially affecting their interrelationships.[15] Sleep duration, as a modifiable behavioral risk factor for CVD,[16] also varies globally. However, large-scale and population-based studies capable of generating generalizable evidence on the relationship between sleep duration and CVD are notably scarce in Iran. Moreover, the inconsistency of previous findings highlights the need for further investigation to fill this scientific gap. Therefore, this study seeks to examine the association between sleep duration and prevalence of CVD in the 40-to 70-year-old population of Kharameh County, southern Iran, as part of the PERSIAN Cohort Study.

Methods

Participants and study design

This cross-sectional study was conducted using the baseline data of Kharameh cohort study, gathered between June 2014 and August 2016. Kharameh cohort study is a branch of the PERSIAN (Prospective Epidemiological Research Studies in Iran) cohort study in the south of Iran. The PERSIAN study is a national cohort study that has been running since 2014 to establish the risk factors for NCDs and, as a result, provide appropriate solutions for controlling chronic diseases.[17]

Residents of Kharameh (10,663), aged 40 to 70 years old, were included in the study. After excluding patients with a history of taking sleeping pills (920), the data from 9,632 participants were analyzed.

Data Collection

Data for the Kharameh Cohort Study were collected through a census approach. After obtaining written informed consent, the participants were assessed by nutritionists, physicians, and trained interviewers. Relevant data were gathered using standardized questionnaires and electronically recorded. Medical and general questionnaires included a total of 482 questions. Each questionnaire was filled out by a trained expert. All relevant information and medical documentation of the individuals, including any illnesses documented during their self-reporting, were systematically examined and confirmed by two qualified specialist physicians in this investigation. CVD events were characterized by the presence of chronic ischemic heart disease, heart failure, myocardial infarction, hypertension, or stroke. Also, some of the samples were analyzed to examine the required laboratory items to record in the baseline data of each participant. Diastolic blood pressure (DBP) and systolic blood pressure (SBP) measurements were obtained following standard protocols. The Metabolic Equivalent of Task (MET score) assessment was carried out to determine the participants' physical activity level.

Calculation of sleep duration

Questions from the Kharameh Cohort questionnaire were used to categorize self-reported sleep duration: less than 6 hours (<6) as short sleep duration, 6 to 6.9 hours (6-6.9), 7 to 7.9 hours (7-7.9) as standard sleep duration, 8 to 8.9 hours (8-8.9), and more than 9 hours (≥9) as long sleep duration.

Statistical Analysis

Qualitative data were presented as frequency (percentage), while quantitative data were expressed as mean±standard deviation. The chi-square test was employed for categorical variables, and one-way ANOVA was used for continuous variables. Logistic regression was used for the calculation of odds ratio (OR) with a 95% confidence interval (CI). To evaluate the association between sleep duration and the odds of CVD, two logistic regression models were constructed. The first model was a crude (unadjusted) model that estimated the univariate association between sleep duration and CVD. The second model was an adjusted model that controlled the potential confounding

variables, specifically age, body mass index (BMI), lipid profiles (CHOL, HDL), systolic and diastolic blood pressure, diabetes, smoking, and MET score. The intercorrelation among these variables may have influenced the regression estimates by attenuating the independent effect of sleep duration on CVD.

To assess the robustness of our adjusted logistic regression model results and evaluate the sensitivity of the observed association to extreme sleep duration categories, we conducted a sensitivity analysis. In this analysis, individuals in the extreme sleep duration categories, those with short (<6) or long (≥9) sleep duration, were excluded from the dataset. The adjusted model, which incorporated several control variables, was re-run for the sensitivity analysis. Subsequently, the adjusted logistic regression model was executed separately for each gender using only the remaining three sleep duration categories: 6-6.9, 7-7.9, 8-8.9. The objective of this sensitivity analysis was to determine whether the association between sleep duration and CVD prevalence was driven primarily by the extreme sleep groups and evaluate the stability of the adjusted ORs in their absence.

Statistical significance was set at a P-value<0.05. All analyses were performed using IBM SPSS statistics, version 23 (IBM Corp., Armonk, NY, USA).

Results

As shown in Table 1, several demographic and clinical variables varied significantly across sleep duration categories. Among the participants, 19.5% reported sleeping less than 6 hours, 25.4% reported 6–6.9 hours, 28.4% reported 7–7.9 hours, 18.0% reported 8–8.9 hours, and 8.6% reported sleeping 9 or more hours. BMI differed significantly among the sleep categories (p=0.004), with the highest mean BMI seen in the 6–6.9 hours group (26.22±4.29 kg/m²). In contrast, serum cholesterol, HDL cholesterol, and both systolic and diastolic blood pressures did not show significant differences.

Participants in the extreme sleep groups, those sleeping <6 hours and those sleeping ≥9 hours, demonstrated distinct profiles compared to those with intermediate sleep durations. Individuals with short sleep (<6) were slightly older (mean age: 52.93±7.99 years), had a higher proportion of males (46.2%) and higher MET score (39.49±6.24), and showed the highest prevalence of CVD (30.2%) and a greater percentage of smokers (27.1%). Conversely, those with long sleep durations (≥9) had the lowest proportion of males (38.8%) and smokers (21.6%) but still exhibited a relatively high prevalence of CVD (28.6%) and diabetes (15.7%). Their MET scores were the lowest among all groups (36.87±5.61). In contrast, participants with sleep durations between 6 and 8.9 hours showed lower prevalence of CVD overall. These findings support the focus on short and long sleep durations as potential risk factors in the evaluation of cardiovascular outcomes.

In the initial analysis, the crude logistic regression was employed to assess the unadjusted association between sleep duration categories and the prevalence of CVD, separately for males and females (Table 2). The odds of CVD were 42% higher in males sleeping <6 hours (95% CI: 1.006–2.02; p=0.046) compared with those who slept 7–7.9 hours (the reference group). In females, both short (<6) and long (≥9) sleep durations were significantly associated with increased odds of CVD. Specifically, the odds of CVD were 44% higher in females sleeping <6 hours (OR=1.44, 95% CI: 1.22–1.70, $p < 0.001$), and 19% higher in those sleeping ≥9 hours (OR=1.19, 95% CI: 1.01–1.40, p=0.028), compared with those who slept 7–7.9 hours (the reference group). For the other sleep duration

Table 1: Baseline characteristics (sociodemographic, clinical and laboratory characteristics) by sleep duration categories

Variables	Sleep duration (Hour)					P-value
	<6	6-6.9	7-7.9	8-8.9	≥9	
N (%)	1877 (19.5)	2446 (25.4)	2740 (28.4)	1736 (18.0)	833 (8.6)	-
Gender (Male) [a]	868 (46.2)	1138 (46.5)	1270 (46.4)	775 (44.6)	323 (38.8)	0.001 *
Age [b]	52.93±7.99	51.83±8.16	51.87±8.06	51.23±8.24	52.48±8.88	<0.001 **
BMI [b]	26.14±4.46	26.22±4.29	25.92±4.33	25.75±4.55	25.82±4.40	0.004 **
Chol [b]	186.37±41.45	187.88±42.05	186.04±41.02	186.60±42.08	186.28±40.92	0.576 **
HDL [b]	47.70±12.82	47.58±12.18	47.86±12.51	47.92±13.15	47.51±12.97	0.865 **
SBP [b]	115.42±17.70	115.12±16.92	115.31±17.77	114.73±18.94	114.93±18.47	0.782 **
DBP [b]	72.33±10.19	72.42±9.86	72.41±10.58	71.87±10.68	72.15±10.51	0.432 **
Met score [b]	39.49±6.24	38.98±6.23	38.70±6.06	38.31±6.42	36.87±5.61	<0.001 **
Has diabetes [a]	311 (16.6)	330 (13.5)	394 (14.4)	226 (13.0)	131 (15.7)	0.013 *
Smoker [a]	509 (27.1)	620 (25.3)	712 (26.0)	432 (24.9)	180 (21.6)	0.041 *
CVD prevalence [a]	566 (30.2)	648 (26.5)	700 (25.5)	440 (25.3)	238 (28.6)	0.003 *

[a] Number (%); [b] Mean±Standard Deviation; P-value reported as the result of * Chi-square and ** Analysis of variance (ANOVA). Statistically significant p-values are bolded (p-value < 0.05)

Table 2: Crude and adjusted odds ratios (ORs) with 95% confidence intervals (CI) for CVD risk by sleep duration categories, stratified by gender using logistic regression

Gender	Variable	Crude Model			Adjusted Model [1]		
	Sleep Duration	OR	95% CI	P-Value	OR	95% CI	P-Value
Male	< 6	1.42	1.006-2.02	0.046	1.31	0.90-1.92	0.153
	6-6.9	1.32	0.91-1.91	0.131	1.17	0.81-1.70	0.388
	Reference (7-7.9)	-	-		-	-	-
	8-8.9	1.31	0.91-1.89	0.144	1.11	0.74-1.66	0.594
	≥ 9	1.34	0.95-1.90	0.089	1.30	0.88-1.93	0.183
Female	< 6	1.44	1.22-1.70	<0.001	1.37	1.13-1.67	0.001
	6-6.9	1.11	0.89-1.38	0.323	1.14	1.01-1.29	0.032
	Reference (7-7.9)	-	-	-	-	-	-
	8-8.9	1.04	0.87-1.25	0.596	1.10	0.85-1.42	0.436
	≥ 9	1.19	1.01-1.40	0.028	1.25	1.04-1.50	0.016

[1] Adjusted for age, body mass index (BMI), lipid profiles (CHOL, HDL), systolic and diastolic blood pressure, diabetes, smoking and MET score. Statistically significant p-values are bolded (p-value<0.05).

category, although the crude associations were not statistically significant, they were generally associated with increased odds of CVD across both genders.

Because the crude model does not account for potential confounders, an adjusted logistic regression model was subsequently employed. This model controlled for a range of demographic, clinical, and laboratory variables that might influence the association between sleep duration and CVD risk. In the adjusted model, the strength of associations changed. In males, while the OR for the <6 hours category decreased to 1.31 (95% CI: 0.90–1.92; p=0.153), none of the sleep duration categories reached statistical significance. By contrast, in females, the associations of both short and long sleep durations with CVD persisted. Short sleep duration (<6 hours) was significantly associated with higher odds of CVD (OR=1.37, 95% CI: 1.13–1.67, p=0.001), as was long sleep duration (≥9) (OR=1.25, 95% CI: 1.04–1.50, p=0.016). Interestingly, even sleeping 6–6.9 hours was associated with a modest but significant increase in CVD risk among females (OR=1.14, 95% CI: 1.01–1.29, p=0.032). These findings underscore that, after adjusting for confounders, the association between suboptimal sleep durations (both short and long) and CVD is more pronounced among females.

To assess the robustness of the findings from the adjusted model, we conducted a sensitivity analysis (Table 3). In this analysis, the extreme sleep categories (i.e., <6 hours and ≥9 hours) were excluded, and the adjusted model was re-run using only the intermediate sleep duration groups (6–6.9, 7–7.9, and 8–8.9 hours). For males, the ORs for the remaining categories slightly attenuated (6-6.9 hours: 1.13 [95% CI: 0.93–1.38], p=0.215; 8- 8.9 hours: 1.10 [95% CI: 0.87–1.39], p=0.389). In females, the previously significant association for the 6-6.9 hours category also diminished (OR=1.02, 95% CI: 0.854–1.22, p=0.799) after excluding the extreme groups, and the 8-8.9 hours category similarly lost significance (OR=0.91, 95% CI: 0.79–1.06, p=0.257). This attenuation in effect size upon the exclusion of the extreme sleep categories indicates that the significant associations observed in the main analysis were largely driven by participants at the extremes of the sleep distribution.

Discussion

This population-based study among Iranian adults indicated that both short (<6 hours) and long (≥9 hours) sleep durations were significantly associated with a higher prevalence of cardiovascular disease. These findings are in line with numerous studies, indicating that deviations from an optimal sleep duration may exert deleterious effects on cardiovascular health.[18, 19] Our initial crude analyses indicated that extreme sleep durations, particularly short (<6 hours) and long (≥9 hours) sleep duration categories, were associated

Table 3: Comparison of odds ratios from adjusted and sensitivity analyses of the association between sleep duration and prevalence of cardiovascular disease, stratified by gender

Gender	Variable	Adjusted Model [1]			Sensitivity Analysis Model		
	Sleep Duration	OR	95% CI	P-Value	OR	95% CI	P-Value
Male	6-6.9	1.17	0.81-1.70	0.388	1.13	0.93-1.38	0.215
	Reference (7-7.9)	-	-	-	-	-	-
	8-8.9	1.11	0.74-1.66	0.594	1.10	0.87-1.39	0.389
Female	6-6.9	1.14	1.01-1.29	0.032	1.02	0.854-1.22	0.799
	Reference (7-7.9)	-	-	-	-	-	-
	8-8.9	1.10	0.85-1.42	0.436	0.91	0.79-1.06	0.257

[1] Adjusted for age, body mass index (BMI), lipid profiles (CHOL, HDL), systolic and diastolic blood pressure, diabetes, smoking and MET score. Statistically significant p-values are bolded (p-value<0.05).

with increased CVD odds in both males and females. However, once potential confounders were controlled in the adjusted model, the associations persisted predominantly in females, suggesting a gender-specific vulnerability that may modulate the effect of sleep duration on cardiovascular risk.

In the adjusted analyses, females with short sleep duration (<6 hours) revealed 37% increased odds of CVD, and those with long sleep duration (≥9 hours) had 25% increased odds, when compared to the reference group (7–7.9 hours). Among males, the associations with extreme sleep durations were attenuated and lost statistical significance. These findings align with previous reports suggesting a U-shaped relationship between sleep duration and cardiovascular outcomes, where both insufficient and excessive sleep may predispose individuals to adverse cardiovascular events. For instance, Cappuccio et al. conducted a meta-analysis that concluded that short sleep duration and long sleep duration were related with elevated odds of stroke and coronary heart disease.[20] Similarly, another study in the Korean population found that both long and short sleep durations are associated with elevated risk of CVD.[21] In line with our study, a large-scale study in Japan confirmed that individuals with less than 6 hours or more than 9 hours of sleep had a higher risk of cardiovascular mortality, relative to the reference group with 7–8 hours of sleep.[22]

The biological mechanisms underlying these associations are multifaceted. Short sleep duration has been implicated in the dysregulation of the autonomic nervous system, resulting in heightened sympathetic activity and reduced parasympathetic activity,[23] which can contribute to increased blood pressure and increased heart rate variability.[24] Additionally, insufficient sleep is associated with metabolic disturbances, including insulin resistance, glucose intolerance, and increased inflammatory markers, such as interleukin-6 and C-reactive protein.[25] These changes collectively may accelerate atherosclerotic processes and ultimately increase the risk of cardiovascular events. On the other hand, long sleep duration may be indicative of underlying health issues or may directly contribute to a sedentary lifestyle, both of which have been linked to adverse cardiovascular outcomes. As an illustration, long sleep has been associated with elevated levels of inflammatory cytokines and a higher prevalence of comorbid conditions such as depression and metabolic syndrome, which are recognized as contributors to cardiovascular risk.[26]

Our study findings are also consistent with several mechanistic studies that have investigated the impact of sleep duration on cardiovascular risk factors. For example, experimental research has shown that sleep restriction leads to an increase in blood pressure and impaired endothelial function,[27] both of which are critical in the pathogenesis of atherosclerosis.[28]Obstructive/complications/*epidemiology</keyword></keywords><dates><year>2002</year><pub-dates><date>Mar 1</date></pub-dates></dates><isbn>1073-449X (Print Moreover, the literature consistently shows that persistent short sleep is associated with a gradual worsening of metabolic parameters, including dyslipidemia and increased body mass index, which are clinically validated risk factors for CVD.[29] Conversely, while long sleep duration is often considered less detrimental in experimental settings, epidemiological evidence suggests that habitual long sleep may serve as a marker for underlying health conditions that elevate cardiovascular risk.[30]

Our sensitivity analysis further explored the robustness of these associations by excluding the extreme sleep groups. The results showed that, in the absence of the short and long sleep categories, the associations were notably attenuated in both genders. This suggests that the significant findings in the main analysis were primarily driven by participants at the extremes of the sleep duration spectrum. The attenuation observed upon exclusion of these groups may indicate that individuals with extreme sleep patterns possess unique risk profiles, possibly related to underlying sleep disorders, comorbid conditions, or other unmeasured factors that substantially contribute to their increased CVD risk.

The gender differences observed in our study are consistent with previous studies that have highlighted sex-specific biological and behavioral pathways linking sleep to cardiovascular health. Females may exhibit greater susceptibility to the adverse cardiovascular impacts associated with disrupted sleep patterns due to hormonal variations, differences in stress response, or differential exposure to psychosocial stressors. Estrogen, for instance, has a protective effect on the cardiovascular system; however, postmenopausal changes and sleep disturbances in females may diminish this protective effect and exacerbate cardiovascular risk.[31] Moreover, females may be more vulnerable to the cardiometabolic consequences of insufficient or excessive sleep due to complex interactions between sleep, mood disorders, and autonomic regulation, which can negatively impact both sleep quality and duration.[32]

The evidence presented herein aligns with and expands upon prior research, emphasizing the importance of sleep in the etiology of CVDs. As the global burden of CVD continues to rise,[33, 34] identification of modifiable risk factors such as sleep duration is critical. Future research should aim to elucidate the underlying biological mechanisms through which sleep influences cardiovascular health,

and investigate whether interventions designed to improve sleep can effectively reduce CVD risk. Additionally, given the gender differences observed in our study, further investigation into the interplay between hormonal status, sleep, and cardiovascular risk is warranted.

Strengths and Limitations

Even though this study is strengthened by factors such as a large sample size, the employment of trained questioners and supervisors, and the stratified approach to analysis, several limitations must be acknowledged. Primarily, the cross-sectional nature of the study limits the ability to draw causal inferences, and reverse causality remains possible. Second, sleep duration was self-reported, which may introduce recall bias or misclassification. Third, residual confounding from unmeasured variables, such as socioeconomic status and mental health, cannot be entirely ruled out. Future prospective studies with objective measures (e.g., actigraphy or polysomnography) of sleep are warranted to validate these findings and elucidate the underlying mechanisms.

Conclusion

In summary, our findings suggest that both short and long sleep durations are associated with increased CVD risk, particularly among females, and these associations are largely driven by participants at the extremes of the sleep distribution. These results highlight the potential importance of maintaining an optimal sleep duration as part of cardiovascular risk management, emphasizing the need for targeted interventions and further research into the gender-specific mechanisms linking sleep and cardiovascular health. These findings underscore the importance of identifying individuals with extreme sleep behaviors as a potentially high-risk group for CVD, particularly females. Sleep assessment should, be considered a routine part of cardiovascular risk evaluation, especially in primary care settings.

Ethical Consideration

All experimental procedures were conducted in accordance with the relevant guidelines and regulations established by the regional and national research ethics committees. The study protocol received formal approval from both regional and national ethical review boards (the equivalent of institutional review boards) of Shiraz University of Medical Sciences (Ethics ID: IR.SUMS.REC.1403.062).

Authors' Contribution

A.D. conceived and designed the study. M.S. developed the analysis plan and supervised all stages of the project. A.R. was responsible for data acquisition from the cohort database. A.D. was responsible for data cleaning and preparing the analytical dataset, performed the statistical analyses, and interpreted the results. A.D. and M.S. drafted the initial version of the manuscript. M.S. critically revised the manuscript for important intellectual content and contributed to refining the interpretation of findings. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Conflict of Interest: None declared.

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