Sleep Quality, Patterns, and Their Impact on Cardiovascular Disease Risk: A cross-sectional Study Among Omani Adults Attending Primary Healthcare in Muscat

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Abstract

Objectives: This study investigated the relationship between sleep quality, sleep patterns, and the 10-year cardiovascular disease (CVD) risk among Omani adults attending primary healthcare centres in Muscat.

Methods: A cross-sectional design was used and data were collected from patients between September 2023 and September 2024. Participants who were Omani and aged 30–75 years were included in the study using convenience sampling; however, those with established cardiovascular disease or showing signs of obstructive sleep apnea were excluded. The 10-year CVD risk was calculated using the Framingham Risk Score (FRS) and sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). The research explored the impact of various sleep patterns (monophasic, bi-phasic, polyphasic), work timings, socio-demographic factors, and body mass index on sleep quality and CVD risk.

Results: A total of 400 participants were included in this study with the majority being female (60%). The results indicate a significant association between poor sleep quality and an increased risk of developing CVD over 10 years (odds ratio [OR] = 1.734, P = 0.032), with flexible work timings showing a protective effect against CVD (OR = 0.514, P = 0.010). However, no significant difference was found between sleep patterns and CVD risk.

Conclusion: These findings emphasise the importance of sleep quality and work flexibility in reducing cardiovascular risk, with potential implications for public health interventions.

Keywords: Sleep Quality; Cardiovascular Diseases; Risk Assessment; Oman.

Introduction

Sleep quality and duration are well recognized for their significance in regulating and optimizing various metabolic and physiological functions in the human body. Studies have shown that poor sleep quality and inadequate sleep

duration are linked to negative health effects on both physical and mental well-being.[1] The National Sleep Foundation recommends that adults get between 7-9 hours of sleep per night for optimal health.[1,2] Factors such as extended working hours, cultural and religious practices, environmental influences, seasonal variations, and emerging communication technologies can all significantly affect sleep quality, duration, and patterns.[1,3]

The association between poor sleep quality and duration with cardiovascular disease (CVD) risk is supported by an expanding body of evidence.[4-7] Hypertension, type 2 diabetes mellitus, an atherogenic lipid profile, and obesity—all well-known risk factors for CVD—are closely associated with sleep quality and duration.[1,4,8,9] Both short (<7 hours per night) and long (>10 hours per night) sleep durations, as well as daytime napping, have been linked to an increased risk of CVD in large-scale prospective cohort studies and meta-analyses.[4-7] Data from the Sleep Heart Health Study (SHHS) demonstrated that sleep efficiency (SE)—the ratio of total time spent sleeping to time spent in bed—was significantly correlated with increased CVD risk and CVD-related mortality when SE was below 80%.[5]

A 2023 study conducted in the United States found significant associations between sleep duration and disturbances and various cardiovascular biomarkers, such as high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglycerides, C-reactive protein (CRP), glycated haemoglobin (HbA1c), insulin, and blood glucose. The findings indicated that both short and long sleep durations, as well as sleep disturbances, were linked to abnormal levels of these biomarkers, suggesting an increased risk of CVD.[7] Additionally, a meta-analysis of 249,324 adults reported a 50% increased risk of developing coronary heart disease among those sleeping less than 7 hours per night. The same study also associated long sleep duration (>10 hours) with an increased risk of coronary heart disease, stroke, and overall CVD risk.[4]

Sleep duration and quality are direct components of sleep patterns, with the internal circadian rhythm regulating specific sleep timing and ensuring sufficient duration for daytime alertness.[3] An individual's sleep pattern is defined as the clock-hour plan of sleep time and wake times, including nap habits and any disruptions to sleep.[10] There is growing evidence that not only sleep duration but also sleep patterns adopted in one's lifestyle can influence health outcomes.[9]

Two research studies conducted in Oman explored the relationship between sleep patterns and metabolic abnormalities.[9,11] The first study found that napping for over an hour in the afternoon was significantly associated with abnormal HbA1c and body mass index (BMI), increasing the risk of developing type 2 diabetes mellitus.[9] The second study reported that regular sleep deprivation was significantly linked to type 2 diabetes mellitus, irrespective of age, gender, BMI, or family history of diabetes.[11] Both studies underscored the need for public health interventions promoting healthy sleep habits.[9,11]

Sleep patterns are generally categorized into three types: monophasic, bi-phasic, and polyphasic.[12] Monophasic sleep consists of a single nighttime sleep period, bi-phasic sleep includes a longer nighttime sleep and a short daytime nap, and polyphasic sleep involves multiple sleep periods throughout the day.[12] Arab Muslims often exhibit distinct sleep patterns due to five daily prayers, with the first at dawn, thereby dividing nighttime sleep into two segments.[13] Furthermore, a recent study conducted in Oman identified four distinct sleep patterns: monophasic, bi-phasic (post-dawn), bi-phasic (afternoon siesta), and polyphasic.[3] The study found that the most prevalent sleep pattern among participants was bi-phasic (afternoon siesta), followed by polyphasic, monophasic, and bi-phasic (post-dawn).[3] There is a lack of research investigating the association between sleep patterns, sleep quality and CVD risk in certain regions of the world, particularly in Muslim Middle Eastern societies, such as Oman, where habitual sleep behaviour differs significantly from that in Western societies. While numerous studies have suggested an association between sleep quality, sleep duration, and CVD risk, few have explored this relationship within Arab Muslim societies, considering the distinct sleep patterns typically adopted in this region.

The objective of this study was to explore the potential relationship between sleep quality among Omani adults attending primary healthcare in Muscat with their 10-year risk of developing CVD. Additionally, the study aimed to assess sleep quality and patterns, alongside the 10-year CVD risk in the same population. The relationship between various sleep patterns—monophasic, bi-phasic (bi-dawn and siesta), and polyphasic—and cardiovascular risk is investigated, as well as the effects of work schedules and job nature on cardiovascular health and their impact on sleep quality. Furthermore, the research examines the influence of socio-demographic factors, such as education level, on

sleep quality and CVD risk. Another important focus is on the association between BMI and sleep quality, and the potential impact of BMI on the risk of developing CVD.

Methods

A cross-sectional design was employed to examine the relationship between sleep quality, sleep patterns, and the 10-year risk of developing CVD among Omani adults attending primary healthcare centres in Muscat. Conducted over a 12-month period, from September 2023 to September 2024, the data was collected at three selected healthcare centres: Al-Athaiba, Al-Shadi, and Al-Seeb, chosen for their representativeness, accessibility to participants, and high-quality facilities. The study targeted Omani adults aged 30 to 75 years, as this age range is specifically relevant for cardiovascular risk assessment using the Framingham Risk Calculator. To ensure reliability and generalizability, the initial sample size was calculated based on the prevalence of moderate to high cardiovascular risk in the Omani population, resulting in an optimal sample size of 331 participants. To enhance the robustness of the findings, the sample size was increased to 400 participants.

A simple random sampling technique was applied to select participants, minimizing selection bias and ensuring that every eligible individual had an equal chance of being included. Individuals with established cardiovascular disease, non-Omanis, those outside the specified age range, and those presenting with signs of obstructive sleep apnea as indicated by the STOP-Bang questionnaire were excluded.

Data were collected by trained staff nurses through direct interviews in the triage clinics of the selected healthcare centres. Participants' 10-year cardiovascular risk was estimated using the non-laboratory Framingham Risk Score (FRS), which incorporates variables such as age, history of diabetes, smoking status, systolic blood pressure, and BMI. Sleep quality and patterns were assessed using the Pittsburgh Sleep Quality Index (PSQI), a validated instrument that evaluates various dimensions of sleep over a one-month period. A score of 5 or higher indicated poor sleep quality. Additional demographic information and physical activity levels were also collected.

Data analysis was conducted using the Statistical Package for the Social Sciences (SPSS) Version 29.0 (IBM Coproration, Armonk, USA) with descriptive statistics, t-tests, ANOVA, and Chi-square tests employed to identify significant associations between variables. Data validity and reliability were ensured by utilizing standardized instruments, such as the FRS and PSQI, and by providing thorough training to data collectors.

Ethical approval was obtained from the Omani Ministry of Health (XXXX), and informed consent was obtained from all participants. For illiterate participants, the consent form and questionnaires were read aloud to ensure comprehension. Participants were assured of the confidentiality of their responses and the voluntary nature of their participation, with no impact on the healthcare services they received. Artificial Intelligence tools, specifically ChatGPT by OpenAI, were used to improve the readability of the manuscript and remove language-related errors.

Results

The study analysed data from 400 individuals across three healthcare centres: Al-Shadi (33.0%), Al-Athaiba (29.8%), and Al-Seeb (37.3%). The majority of participants were female (60%) and non-smokers (85.5%). Most participants did not have diabetes (81.5%), were not undergoing hypertension treatment (75.3%), and had no comorbid conditions (65.9%). A significant portion were married (82.5%) and educational status varied, with 28.0% having a college or university-level education and 17.8% being illiterate. Occupationally, participants were almost equally split among office-based work (33.5%), field-based work (34.0%), and non-working (32.5%). The majority (44.8%) worked regular fixed office hours, while 34.3% had flexible work timings and 21.0% worked shifts, reflecting diverse working conditions. In terms of age, 60.0% were in the 30–44 years range, 34.0% in the 45–64 years range, and 6.0% in the 65–75 years range. The mean BMI was 29.16 ± 6.4, indicating an overweight population, with 34.3% categorized as overweight and 35.3% as obese. The average systolic blood pressure was 126.98 ± 14 mmHg [Table 1].

Table 1: Demographic characteristics of participants attending three healthcare centres in Muscat, Oman (N = 400).

Category	n (%)

Age group (years)	
30–44	240 (60.0)
45–64	136 (34.0)
65–75	24 (6.0)
Gender	
Male	160 (40.0)
Female	240 (60.0)
Healthcare centre	
Al-Shadi	132 (33.0)
Al-Athaiba	119 (29.8)
Al-Seeb	149 (37.3)
Marital status	
Married	330 (82.5)
Single	51 (12.8)
Divorced	13 (3.3)
Widow	6 (1.5)
Education level	
College/University level	112 (28.0)
Secondary school level	82 (20.5)
Primary school level	44 (11.0)
Read and write	91 (22.8)
Illiterate	71 (17.8)
Smoking	
Smoker	58 (14.5)
Non-Smoker	342 (85.5)
BMI	
Underweight	20 (5.0)
Normal weight	102 (25.5)
Overweight	137 (34.3)
Obese	141 (35.3)
Comorbidities	
Yes	137 (34.3)
No	263 (65.7)
Family History of CVD	
Yes	108 (27.0)
No	292 (73.0)
Work nature	
Office-based work	134 (33.5)
Field-based work	136 (34.0)
Does not work	130 (32.5)
Work timings	
Fixed work timing	179 (44.8)
Free worker (flexible timing)	137 (34.3)
Shift system	84 (21.0)

BMI = body mass index; CVD = cardiovascular disease.

Sleep quality, as indicated by the PSQI, was reported as good by 60% of participants, with an average sleep duration of 6.8 ± 1.5 hours per night. The mean bedtime was 10:30 PM and wake-up time was 6:30 AM [Table 2]. Most participants (59.5%) follow a bi-phasic (afternoon siesta) sleep pattern, while 24.8% were monophasic sleepers. Additionally, 8.5% followed a bi-phasic (post-down) pattern, and 7.2% adhere to a polyphasic sleep pattern.

Table 2: Multivariate binary logistic regression analysis to determine the independent effect of sleep quality (PSQI) on the 10-year risk of cardiovascular disease.

				95% CI for OR	
Variable	**β**	***P*-value***	**OR**	**Lower**	**Upper**

Marital status					
Married (Reference)			1.000		
Single	-0.668	0.054	0.513	0.260	1.011
Divorced	-0.226	0.759	0.797	0.188	3.389
Widow			*NE*		
Education status					
College/University (Reference)			1.000		
Secondary school	1.008	0.007	2.740	1.316	5.705
Primary school	1.436	0.006	4.203	1.519	11.627
Read and write	0.720	0.046	2.054	1.012	4.173
Illiterate	-0.095	0.796	0.909	0.441	1.874
Work nature					
Not working (Reference)			1.000		
Office-based work	0.339	0.301	1.403	0.738	2.667
Field-based work	-0.256	0.416	0.774	0.417	1.435
Work timings					
Fixed work timing (regular) (Reference)			1.000		
Free worker (flexible timings)	-0.751	0.009	0.472	0.269	0.829
Shift system	-0.378	0.256	0.685	0.357	1.316
Family history of CVD					
No (Reference)			1.000		
Yes	-0.054	0.845	0.947	0.550	1.630
Sleep pattern					
Monophasic (Reference)			1.000		
Bi-phasic (afternoon siesta)	-0.224	0.483	0.799	0.427	1.496
Bi-phasic (post-dawn)	-0.440	0.373	0.644	0.245	1.696
Poly-phasic	0.118	0.823	1.125	0.399	3.171
Physical activity					
High intensity (Reference)			1.000		
Moderate intensity	1.458	<0.001	4.297	2.503	7.377
Low intensity	1.695	<0.001	5.447	2.806	10.572
PSQI					
Good sleep (Reference)			1.000		
Poor sleep	0.550	0.032	1.734	1.049	2.866

OR = odds ratio; CI = confidence interval; NE = not estimated; CVD = cardiovascular disease; PSQI = Pittsburgh Sleep Quality Index.

**P-values ≤0.05 are considered statistically significant.*

CVD risk assessment as measured by the non-laboratory FRS showed that 66.5% of participants had a moderate to high 10-year CVD risk (≥10%). The analysis showed a significant association between sleep quality and an increased 10-year risk of CVD [Figure 1]. Participants with poor sleep quality had a 73.4% higher risk of developing CVD over 10 years compared to those with good sleep quality. This finding was supported by both the Chi-square test results and multivariate logistic regression analysis (odds ratio [OR] = 1.734, 95% confidence interval [CI]: 1.049–2.866, $P = 0.032$) [Table 3].

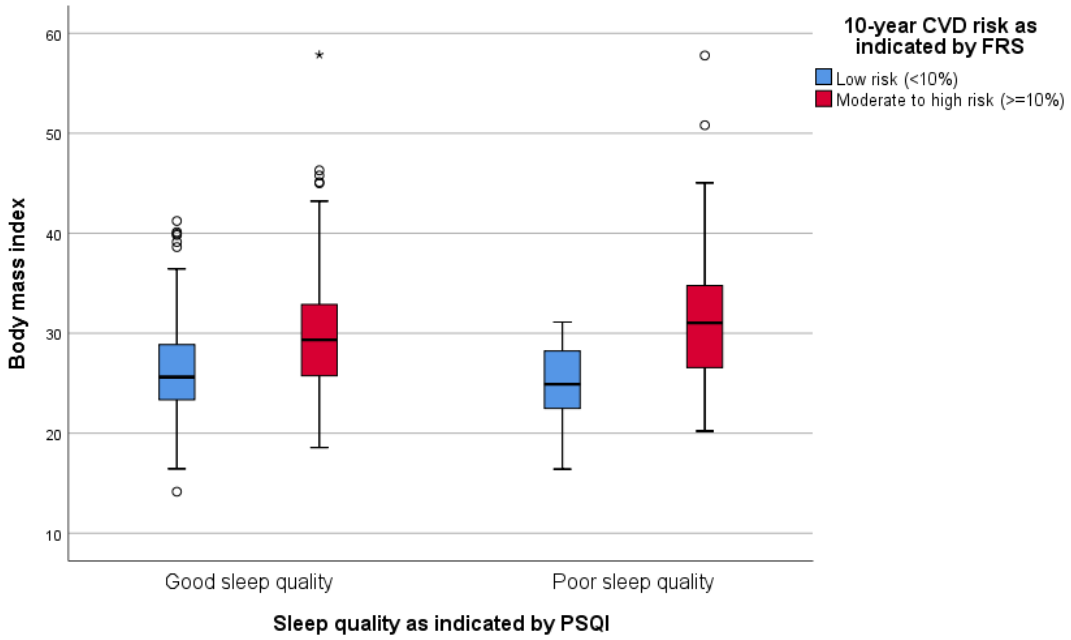

Figure 1: Impact of body mass index on sleep quality and 10-year cardiovascular disease (CVD) risk. PSQI = Pittsburgh Sleep Quality Index; FRS = Framingham Risk Score.

Table 3: Multivariate binary logistic regression analysis to determine the independent predictors of poor sleep quality (PSQI).

				95% CI for OR	
Variable	**β**	***P*-value***	**OR**	**Lower**	**Upper**
Age groups					
30-44 years (Reference)			1.000		
45-64 years	0.047	0.865	1.048	0.608	1.809
65-75 years	0.266	0.622	1.305	0.453	3.759
Gender					
Male (Reference)			1.000		
Female	0.314	0.189	1.369	0.857	2.187
Smoking					
No (Reference)			1.000		
Yes	-0.836	0.019	0.434	0.216	0.871
Diabetes					
No (Reference)			1.000		
Yes	0.283	0.378	1.327	0.707	2.488
BMI groups					
Normal weight (Reference)			1.000		
Underweight	0.104	0.851	1.110	0.375	3.281
Overweight	0.063	0.830	1.065	0.598	1.896
Obese	0.563	0.050	1.756	1.001	3.083
Marital status					
Married (Reference)			1.000		
Single	-0.678	0.063	0.508	0.248	1.038

Divorced	-0.094	0.876	0.910	0.277	2.991
Widow	-0.682	0.467	0.505	0.080	3.183
Education status					
College/University (Reference)			1.000		
Secondary school	-0.363	0.278	0.696	0.361	1.339
Primary school	-0.525	0.223	0.592	0.255	1.376
Read and write	-0.009	0.977	0.991	0.520	1.888
Illiterate	-0.572	0.111	0.564	0.279	1.140
Work nature					
Not working (Reference)			1.000		
Office-based work	0.385	0.196	1.469	0.820	2.634
Field-based work	0.017	0.953	1.018	0.572	1.810
Work timings					
Fixed work timing (regular) (Reference)			1.000		
Free worker (flexible timings)	-0.666	0.010	0.514	0.309	0.854
Shift system	-0.079	0.788	0.924	0.521	1.640
Sleep pattern					
Monophasic (Reference)			1.000		
Biphasic (afternoon siesta)	-0.076	0.785	0.927	0.536	1.602
Biphasic (post-dawn)	0.040	0.930	1.041	0.423	2.564
Polyphasic	0.289	0.520	1.335	0.554	3.217
Physical activity					
High intensity (Reference)			1.000		
Moderate intensity	0.153	0.549	1.166	0.706	1.923
Low intensity	0.050	0.883	1.052	0.536	2.065

PSQI = Pittsburgh Sleep Quality Index; OR = odds ratio; CI = confidence interval.
**P-values ≤0.05 are considered statistically significant.*

The 10-year CVD risk was significantly associated with education, work timings, and physical activity. However, the distribution of CVD risk among different sleep patterns did not show a statistically significant difference (P = 0.143), indicating that sleep patterns do not significantly impact the 10-year risk of developing CVD in this study population [Table 3].

The study revealed a significant association between work timings and the 10-year risk of developing CVD. Individuals with flexible work timings had a lower CVD risk, with Chi-squared analysis confirming that these differences were statistically significant (P = 0.009). Logistic regression further supported that flexible work timings were associated with a reduced risk of CVD (OR = 0.514, P = 0.010).

BMI and work timings emerged as significant independent predictors of sleep quality. Obese participants had a 75.6% higher risk of poor sleep quality compared to those with normal weight (OR = 1.756, 95% CI: 1.001–3.083, P = 0.050) [Figure 2].

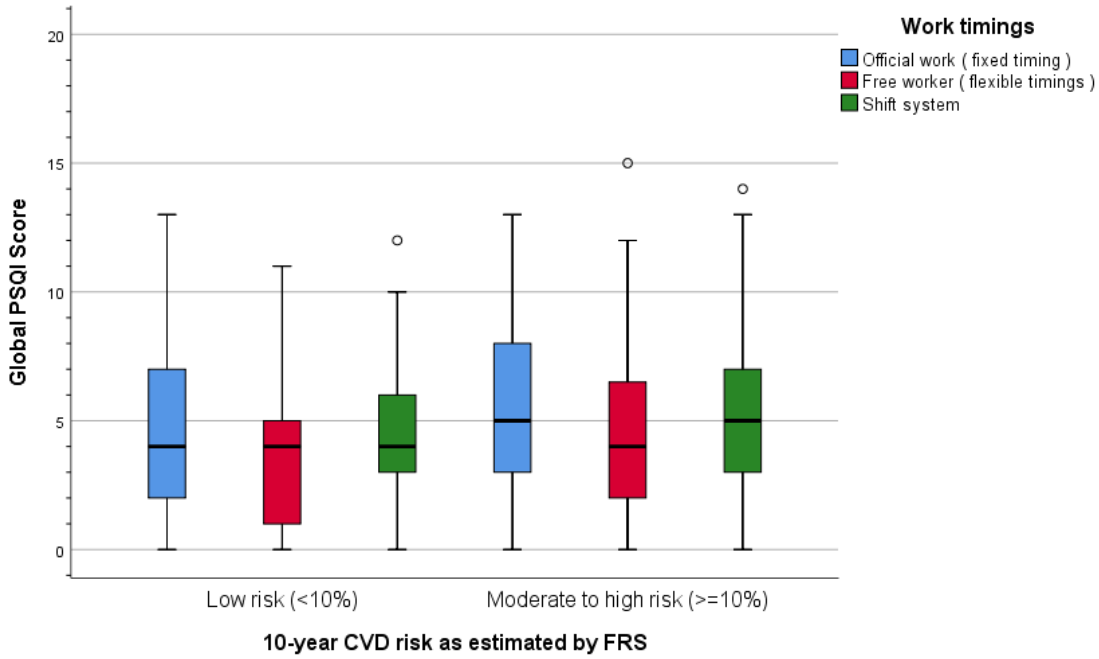

Figure 2: Impact of work timings on sleep quality and 10-year cardiovascular disease (CVD) risk. PSQI = Pittsburgh Sleep Quality Index; FRS = Framingham Risk Score.

Discussion

The primary objective of this study was to examine the relationship between sleep quality and the 10-year risk of developing CVD among Omani adults attending primary healthcare centres in Muscat, Oman, with an emphasis on whether poor sleep quality serves as a significant predictor of elevated CVD risk in this population. Additionally, the study aimed to assess sleep quality, sleep patterns, and the 10 years CVD risk, as well as explore association between different sleep patterns (monophasic, biphasic, and polyphasic) and CVD risk. The study further examined the impact of work timing and work nature on cardiovascular health and its relationship with sleep quality. Socio-demographic factors, such as education level, were also assessed for their influence on sleep quality and subsequent CVD risk, along with exploring the relationship between BMI and sleep quality as a potential modifier of CVD risk.

The average nocturnal sleep duration in our study was 6.8 hours, aligning with findings from recent regional studies, which reported approximately 7 hours of sleep among Omani adults and 7.2 hours among individuals in Saudi Arabia who adhere to the dawn (Fajr) prayer.[3,13] A study in Kuwait further supported this pattern, reporting a mean sleep duration of 6.28 hours using the PSQI and 6.7 hours with actigraphy.[14] These results suggest that the sleep durations in our sample are consistent with regional patterns influenced by cultural and religious practices, such as the Fajr prayer, which can disrupt consolidated sleep.[3,13,14] The Kuwait study highlights that differences in sleep duration between our study and earlier research in Oman may be due to the more accurate measurements provided by actigraphy compared to the self-reported methods used in our study.[14]

Our findings show that most participants followed a bi-phasic sleep pattern, aligning with the traditional Mediterranean siesta and common Middle Eastern sleep habits.[3,13] We also observed a bi-phasic post-dawn sleep pattern, with sleep episodes before and after the Fajr prayer. In contrast, a study conducted in Brazil, a region with different cultural, religious, and lifestyle contexts, found that monophasic sleep was predominant, with little evidence of bi-phasic or polyphasic sleep.[12] This contrast highlights how cultural, religious, and lifestyle factors uniquely shape sleep behaviours in different regions.

Our study demonstrates a significant association between sleep quality and an increased 10-year risk of CVD, aligning with findings from a similar study conducted among female nurses in Hong Kong.[15] In our research, participants with poor sleep quality had a 73.4% higher risk of developing CVD over 10 years compared to those with good sleep quality (OR = 1.734, 95% CI: 1.049–2.866, P = 0.032). The Hong Kong study also found that female nurses with sleep disturbances had a higher CVD risk (OR 1.82; 95% CI: 1.04–3.18), similar to our observed odds ratio.[15] Numerous other studies have supported this association, showing that poor sleep quality, insomnia, and short sleep duration are consistent predictors of increased cardiovascular risk. For example, studies by Covassin and Singh (2016), Wang *et al.* (2022), and Bertisch *et al*. (2018) reinforce the evidence that poor sleep is a significant contributor to elevated CVD risk across different populations.[1,6,16] Several mechanisms may explain this relationship, as poor sleep quality is known to contribute to various physiological disruptions, including increased sympathetic nervous system activity, elevated blood pressure, inflammation, and metabolic dysregulation—all of which are well-established risk factors for CVD.[11,17]

Additionally, poor sleep can exacerbate conditions like obesity and type 2 diabetes, further increasing the likelihood of cardiovascular issues.[11] A meta-analysis exploring the impact of sleep deprivation on CVD risk found that sleep deprivation significantly increases the risk of CVD by contributing to inflammation, hypertension, and insulin resistance, all of which are critical contributors to the development and progression of atherosclerosis.[18]

While our study found a general association between poor sleep quality and increased CVD risk, the Hong Kong study also highlighted specific components of sleep quality, such as daytime dysfunction, which had a more complex relationship with CVD risk.[15] Interestingly, higher daytime dysfunction scores in the Hong Kong study were associated with a lower CVD risk—contrary to the expected relationship between poor sleep quality and cardiovascular health.[15] This finding suggests that while overall sleep quality is an important factor, individual components of sleep may interact with cardiovascular risk factors in nuanced ways.[15] This nuanced interaction calls for more targeted research to unravel these relationships, particularly how different aspects of sleep can independently or synergistically influence cardiovascular outcomes.

Our results indicated that the distribution of CVD risk among different sleep patterns did not show a statistically significant difference (P = 0.143), suggesting that sleep patterns alone do not significantly influence the 10-year risk of developing CVD. In the Omani study by Al-Abri *et al*. (2020), different sleep patterns were identified, including monophasic, bi-phasic-siesta, bi-phasic-dawn, and polyphasic patterns.[3] Although these patterns varied significantly in terms of sleep duration and timing, the study did not find a clear association between these sleep patterns and health outcomes related to sleep.[3] The lack of a significant difference in CVD risk across sleep patterns in our study may be attributable to the complex interplay of factors influencing cardiovascular health. The Omani study highlighted that lifestyle, environmental factors, and cultural practices, such as afternoon siestas, might mitigate the potential negative effects of segmented or polyphasic sleep patterns.[3]

Our statistical analysis revealed no significant association between sleep patterns and sleep quality (P = 0.529). We also did not identify any significant predictors of poor sleep quality across different sleep patterns, although there was a slight, non-significant trend toward poorer sleep quality among participants with a polyphasic sleep pattern (OR = 1.335, P = 0.520). These findings suggest that, in our sample, sleep patterns do not significantly impact sleep quality as measured by the PSQI, aligning with recent studies that have shown no strong link between sleep patterns and sleep quality.[3,13] Notably, no previous studies have specifically examined the relationship between monophasic, bi-phasic (afternoon siesta and post-dawn), and polyphasic sleep patterns and sleep quality.

Our study revealed a significant association between work timings and the 10-year risk of developing CVD (P = 0.009). Individuals with flexible work timings showed a significantly lower CVD risk (OR = 0.516, P = 0.013). Additionally, a significant association between work timings and sleep quality was observed (P = 0.012). Further analysis showed that individuals with flexible work timings had significantly lower odds of experiencing poor sleep quality (OR = 0.514, 95% CI: 0.309–0.854, P = 0.010), indicating that flexibility in work hours may improve sleep quality.

These findings align with existing research exploring the impact of work schedules on individual health, demonstrating that flexible work timings are associated with improved health outcomes, particularly in reducing cardiovascular risk.[19-22] Improved sleep quality, circadian rhythm stability, and reduced stress levels are likely

mechanisms through which flexible work timings exert their protective effects on cardiovascular health. In Japan, individuals with flexible schedules reported better sleep quality compared to those with fixed hours.[22] Joyce *et al.* (2011) highlighted that flexible working conditions improve sleep quality and reduce psychological stress, both of which are linked to lower cardiovascular risk.[19] Similarly, Haley and Miller (2015) showed that flexible schedules lower stress and improve sleep quality, indirectly contributing to better cardiovascular health.[21] Kelly *et al.* (2011) provided evidence that flexible work arrangements improve sleep and reduce stress, enhancing cardiovascular outcomes by promoting better alignment with circadian rhythms.[20]

Our study also showed that individuals with lower education levels are at a significantly higher risk of developing CVD over a 10-year period. Educational attainment is a well-established determinant of CVD risk.[20] Studies suggest that individuals with higher education levels tend to have better cardiovascular health, partly due to greater health literacy, healthier lifestyle choices, and better access to healthcare resources.[23,24] People with higher educational levels are more likely to have stable jobs, regular work hours, and better living conditions, which contribute to better sleep.[23] Conversely, those with lower education levels may experience job insecurity, irregular work hours, and higher stress levels, leading to poorer sleep quality.[23] This suggests that lower educational levels not only directly contributes to higher CVD risk through lifestyle and healthcare access but also indirectly through its impact on sleep quality.

Our findings strongly suggest that increasing the intensity of physical activity is associated with a lower risk of developing CVD over a 10-year period. This aligns with extensive research showing that regular, vigorous physical activity plays a crucial role in reducing cardiovascular risk factors, including hypertension, obesity, and poor lipid profiles.[25] Encouraging higher levels of physical activity could, therefore, be an effective strategy in CVD prevention.

Most research has focused on the effects of sleep duration on physiological changes like obesity and metabolic markers, but there is limited investigation into the relationship between overall sleep quality and other anthropometric measures in the absence of sleep apnea, particularly among the general adult population of Oman.[4,9,14,26] One of the objectives of our study was to assess the independent effects of sociodemographic factors and health-related issues like obesity on sleep quality in the adult Omani population. In our study, 60% of participants reported good sleep quality, compared to 55% in a previous Omani study.[3] The slight variation may be due to broader work types and flexible schedules in our sample, which positively influenced sleep quality. The previous Omani study did not examine work schedules in detail, potentially contributing to their lower percentage of good sleepers. Additionally, our study used subjective self-reports, whereas the Omani study employed actigraphy, which may have detected sleep disruptions not captured in self-assessments, further explaining the differences in findings.

The higher prevalence of poor sleep quality observed in our study and other regional studies, such as the Kuwaiti study, may be attributed to the greater prevalence of obesity in Gulf countries.[14] In our study, 40% of participants reported poor sleep quality, with BMI emerging as a significant independent predictor. Obese participants faced a 75.6% higher risk of poor sleep quality compared to those with normal weight (OR = 1.756, 95% CI: 1.001–3.083, $P = 0.050$). Similarly, the Kuwaiti study reported a higher prevalence of poor sleep quality (59.6%) and a positive correlation between BMI and sleep quality ($r = 0.348$, $P \leq 0.0001$).[14] The rising trend toward higher BMI in Middle Eastern countries has been linked to an increasing prevalence of obesity, driven by the adoption of Westernized diets high in fat, sugar, and salt, increased eating outside the home, and low physical activity levels. These factors were also believed to influence both obesity and sleep quality.[14,27] Other studies across different populations and settings have consistently demonstrated a strong relationship between sleep quality and obesity.[28-30]

The relationship between poor sleep quality and obesity appears to be bidirectional, although the underlying mechanisms remain unclear.[31] Even without sleep apnea, individuals with obesity may experience impaired sleep quality and duration.[32] Poor sleep quality can increase the risk of obesity through its impact on metabolism, hormone secretion, and appetite regulation.[28,29] Disruptions in the secretion of hormones such as ghrelin and leptin during sleep restriction can lead to an energy imbalance, affecting food choices and calorie intake.[28,29] Additionally, poor sleep quality contributes to fatigue, reducing physical activity, and promoting abdominal fat accumulation. Overall, these factors may lead to an imbalance between energy intake and expenditure, thereby promoting weight gain.[28,29]

Obesity, in turn, significantly impacts sleep quality, largely through the accumulation of excessive adipose tissue, which narrows and obstructs the upper airway, contributing to sleep dysfunction, a key component of sleep quality assessment.[30] While this is more common in cases of severe obesity, sleep dysfunction also affects individuals with

grade I obesity or overweight, suggesting additional contributing mechanisms beyond airway obstruction.[30] Visceral adipose tissue plays a significant role in releasing inflammatory cytokines such as IL-1, IL-6, and TNF-α, which influence sleep regulation, particularly slow-wave sleep. A previous study found that waist circumference, an indirect measure of visceral fat, was higher in individuals with sleep dysfunction and positively correlated with sleep quality as measured by the PSQI.[30] In contrast, only a few studies found no significant relationship between sleep quality and obesity, indicating that this association may vary depending on other individual or contextual factors.[29,33]

Our study did not find a significant association between sleep quality and work nature (office-based or field-based; $P = 0.499$). This is consistent with findings from Kuwait, which showed no differences in sleep quality between desk and non-desk jobs.[14] These findings suggest that other factors, such as work schedule flexibility and individual health behaviours, may be more influential in determining sleep quality than the physical nature of one's work.

This study contributes to the growing body of evidence on the relationship between sleep quality, CVD risk, work schedules, and obesity within the Middle Eastern population, focusing specifically on the Omani context. However, several limitations must be acknowledged. First, the cross-sectional design of this study limits the ability to infer causality between variables. The associations observed, such as those between flexible work timings and improved sleep quality or between obesity and poor sleep quality, may reflect bidirectional influences that cannot be clarified without longitudinal data. Additionally, the use of self-reported data for sleep quality through the PSQI introduces the potential for recall bias, which may affect the accuracy of the findings. The convenience sampling of participants from three healthcare centers in Muscat may limit the generalizability of the results to the broader population. While cultural and religious practices unique to the region influence sleep patterns, their broader impact on CVD outcomes remains understudied. Finally, the use of the non-laboratory FRS for CVD risk estimation, while valid, lacks detailed clinical markers, and the exclusion of participants with established CVD or obstructive sleep apnea may have resulted in an underestimation of the associations observed.

Conclusion

This study highlights the critical role of sleep quality in predicting the 10-year risk of CVD among Omani adults. Poor sleep quality was found to significantly elevate CVD risk, whereas flexible work timings demonstrated a protective effect. The lack of a significant association between sleep patterns and CVD risk suggests that sleep quality, rather than specific sleep patterns, plays a more influential role in cardiovascular health. Furthermore, lower educational attainment was identified as a significant contributor to increased CVD risk, and obesity was linked to both poor sleep quality and elevated CVD risk. These findings underscore the importance of public health initiatives aimed at promoting better sleep hygiene, particularly targeting vulnerable grounds, including individuals with shift work schedules, lower educational backgrounds, and higher BMI. Addressing these factors may be key in mitigating long-term cardiovascular risks in the population.

Disclosure

The authors declare no conflicts of interest. No funding was received for this study.

Data Availability Statement

The data for this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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