BMC Public Health

RESEARCH

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Association between sleep duration and long-term changes in novel anthropometric and atherogenic indices: a cohort study

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Abstract

Background Adequate sleep is a crucial aspect of overall health. While existing research has highlighted the impact of factors like obesity and lipid profiles on sleep patterns, limited attention has been given to exploring the relationship between sleep duration with novel anthropometric and atherogenic indices.

Methods This study was part of the Mashhad Stroke and Heart Atherosclerotic Disorders (MASHAD) cohort study. 7,449 participants' sleep duration was assessed at baseline using a questionnaire, and they were followed for 10 years to calculate novel anthropometric and atherogenic indices.

Results Participants who slept over 9 h had a 41.7% higher likelihood of reduced body shape index (ABSI) (ABSI < 0.086, OR: 1.417, $P = 0.021$) and a 49.5% greater chance of a lower weight-adjusted-waist index (WWI) (WWI < 12, OR: 1.495, $P = 0.011$). In contrast, less than 6 h of sleep was associated with a 40.1% decrease in the odds of reduced ABSI (OR: 0.599, $P < 0.001$) and a 32.7% lower likelihood of reduced WWI (OR: 0.673, $P < 0.001$). Long sleepers also had a 31.3% lower chance of body adiposity index (BAI) < 37.5 (OR: 0.687, $P = 0.027$) and a 27% reduced chance of high-density lipoprotein (HDL) < 55 mg/dl (OR: 0.730, $P = 0.037$). Shortened sleep decreased the odds of a lower abdominal volume index (AVI) (AVI < 21) by 19.1% (OR: 0.809, $P = 0.010$).

Conclusion Longer sleep durations were associated with lower ABSI and WWI, while shorter sleep durations correlated with increased ABSI, WWI, and AVI. Additionally, extended sleep was associated with elevated HDL levels, although it also contributed to increased BAI.

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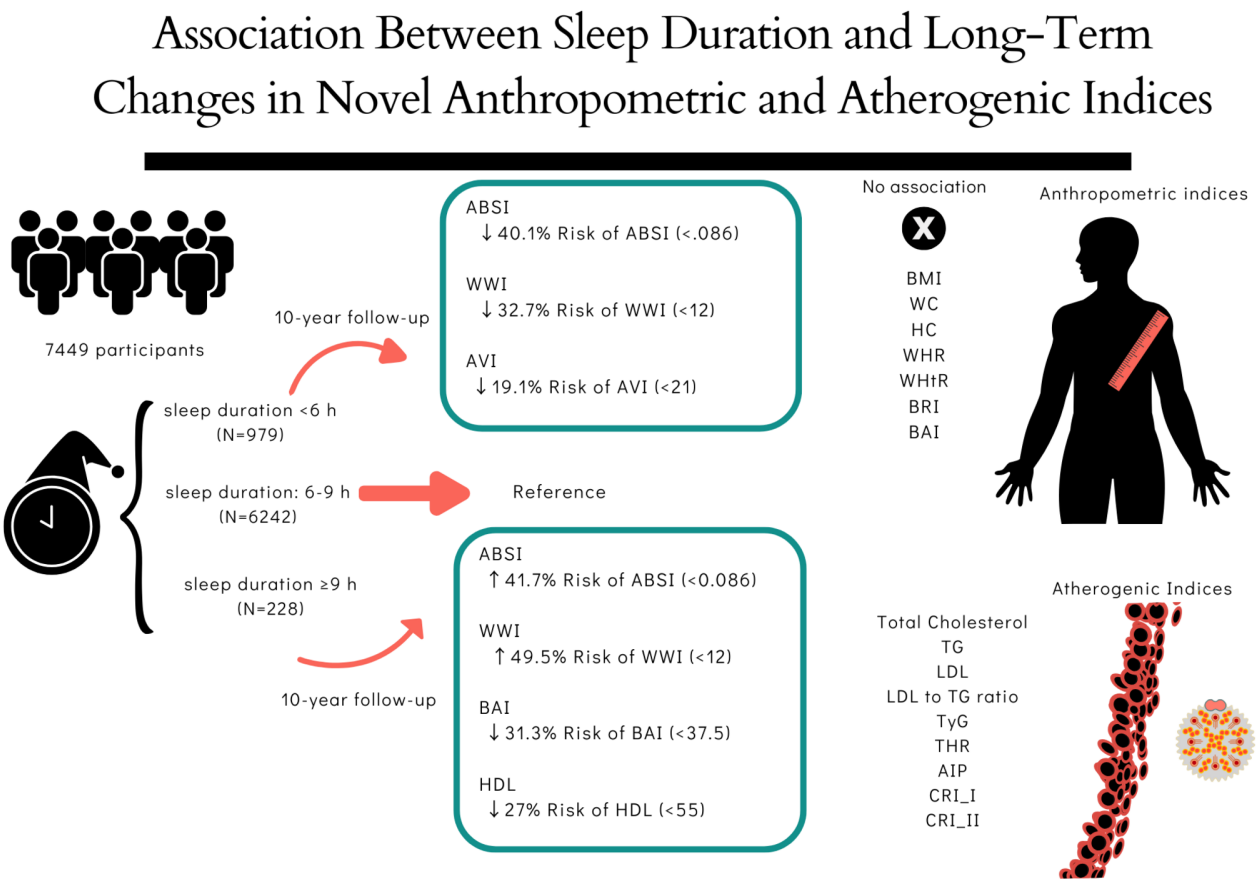
Graphical Abstract



Keywords Sleep duration, Anthropometric indices, Atherogenic index, Cohort study, 10-year follou-up

Introduction

Sleep is vital for human health, with age-specific recommendations for adequate duration [1]. Despite guidelines suggesting 7–9 h for adults aged 26–64 and 7–8 h for those 65+, a global decline in sleep duration is concerning [2]. Insufficient sleep has diverse health impacts, notably in retired Iranian elders [3]. Research highlights variations in sleep patterns among different demographic groups in Iran [4]. This trend raises significant public health concerns, affecting community well-being and healthcare systems [4–6].

In addition to its immediate effects on alertness and cognitive function, sleep duration significantly influences various health outcomes, including lipid profiles and weight management [7]. Research has shown that longer sleep duration is linked to elevated levels of high-density lipoprotein (HDL) cholesterol and can affect other lipid components [8–10]. Atherogenic indices derived from lipid profiles are thought to detect dyslipidemias better, even when individual lipid parameters show only minor changes [11]. These indices include the Atherogenic Index of Plasma (AIP), Atherogenic Coefficient (AC), Castelli risk index (CRI-I, CRI-II), Triglyceride-glucose index (TyG) and Triglyceride to HDL-C Ratio (THR) [12–14]. A meta-analysis indicated that AIP levels were significantly higher in patients with obstructive sleep apnea (OSA) [15]. However, to our knowledge, limited studies have investigated the relationship between sleep duration and atherogenic indices [16].

Inadequate sleep has been shown to impede fat loss during dietary interventions, with each additional hour of sleep linked to increased fat loss percentages [17]. A recent meta-analysis by Grimaldi et al. indicated that many longitudinal studies assessing baseline sleep duration and its impact on anthropometric changes utilized body mass index (BMI), fat percentage, or obesity risk as measures [18]. Other studies examined other traditional anthropometric indexes such as waist circumference (WC), waist-to-hip Ratio (WHR), and waist-to-height ratio (WHtR) [19, 20]. However, it has been observed that

these central obesity indices provide only limited insight into fat distribution. BMI, for example, is a rough indicator of obesity, as individuals with similar BMI values can exhibit varying degrees of fatness [21]. Additionally, the implications of WC can be unclear depending on overall body size [22]. Consequently, novel anthropometric indices have been developed, including the body shape index (ABSI) [23], weight-adjusted-waist index (WWI) [24], body round index (BRI) [25], body adiposity index (BAI) [26], and abdominal volume index (AVI) [27].

Despite the significant body of research examining the relationship between sleep status with lipid profiles and traditional anthropometric indices, a gap remains in our understanding of how sleep duration affects atherogenic and novel anthropometric indices. This prospective cohort study aims to bridge this gap by exploring the intricate connections between sleep duration and these parameters, offering valuable insights into the broader implications of sleep on overall health and well-being. By elucidating these relationships, this study seeks to contribute to the expanding body of knowledge in sleep research and inform strategies for promoting optimal health through improved sleep practices.

Methods

Study population

The research was conducted on individuals who participated in the ongoing Mashhad stroke and heart atherosclerotic disorder (MASHAD) study, which commenced in 2010 and is scheduled to continue until 2020. The research involved 9,704 participants from various districts of Mashhad, a major city in the northeastern Iran region. The MASHAD study and its methodologies were extensively discussed in the recent paper [28]. All healthy individuals between 35 and 65 years old were included in the MASHAD study. Individuals with cardiovascular disease, cancer, and auto-immune diseases or who migrated were excluded from the study. This study was approved by the ethics committee of Mashhad University of Medical Sciences (IR.MUMS.MEDICAL.REC.1403.005). Also, written informed consent was provided from study participants.

The socio-economic status and demographic information of the 7449 included a self-administered questionnaire completed by participants. This information included age, sex, educational status, employment status, and marital status. Hypertension was defined as a systolic blood pressure (SBP) of 140 mmHg or higher, a diastolic blood pressure (DBP) of 90 mmHg or higher, or the use of antihypertensive medications. Type 2 diabetes mellitus (T2DM) was defined as a fasting blood glucose (FBG) of 126 mg/dl or higher, or treatment with oral hypoglycemic medications or insulin [28].

Sleep measurement

A self-administered questionnaire was used to measure sleep duration. Night shift workers were excluded from this study [29]. The participants were categorized into three subgroups according to their nocturnal sleep duration: less than 6 h, 6 to 9 h, and more than or equal to 9 h [30]. These groups consisted of 979, 6242, and 228 individuals, respectively.

Atherogenic indices calculation

A decade later, the same individuals were requested to return and provide blood and urine specimens. Blood samples were collected between 8 AM and 10 AM. Collecting blood from the elbow vein after refraining from eating for 14 h. Vacuum tubes (20 ml) were used to collect samples from individuals who were sitting down, following a specified procedure. We centrifuged the blood samples at room temperature for 30–45 min to divide the serum and plasma into six small containers (0.5 ml each). We dispatched them to the Bu Ali Research Institute in Mashhad. Additionally, the serum samples were stored at -80 degrees Celsius for potential testing. Low-density lipoprotein cholesterol (LDL-C) was calculated from the serum total cholesterol (TC), triglycerides (TGs), and HDL-C concentrations expressed in mg/dl using Friedewald's formula [31] if serum TGs concentrations were lower than 400 mg/dl [28].

Then we calculated AIP, AC, CRI-I, CRI-II, TyG, and THR [12–14].

$$\text{AIP} = \log\left(\frac{TG\left(\frac{mg}{dl}\right)}{HDL-C\left(\frac{mg}{dl}\right)}\right)$$

$$\text{AC} = \text{TC-}\ \frac{HDL-C\ \left(\frac{mg}{dl}\right)}{LDL-C\ \left(\frac{mg}{dl}\right)}$$

$$\text{CRI-I} = \frac{TC\left(\frac{mg}{dl}\right)}{HDL-C\left(\frac{mg}{dl}\right)}$$

$$\text{CRI-II} = \frac{LDL-C\left(\frac{mg}{dl}\right)}{HDL-C\left(\frac{mg}{dl}\right)}$$

$$\text{TyG} = \log\left(\frac{\text{TG}\left(\frac{mg}{dl}\right)\times fasting\ Glucose\left(\frac{mg}{dl}\right)}{2}\right)$$

$$\text{THR} = \frac{TG\ \left(\frac{mg}{dl}\right)}{HDL-C\ \left(\frac{mg}{dl}\right)}$$

Novel anthropometric indices calculation

Height, WC, and hip circumference (HC) were measured in centimeters to the nearest 0.1 cm using a stadiometer (SECA 217, Hamburg, Germany). Additionally, a digital scale was utilized for weight measurement. Participants refrained from food consumption for 14 h and avoided vigorous exercise for the preceding 12 h. WC measurement was taken at the midpoint between the bottom of the ribcage and the top of the hip bones, and it was recorded after a normal exhalation. At the same time, the individual stood with their feet close together and arms hanging relaxed at their sides. HC was measured at the

widest part of the buttocks without tight underwear, ensuring no pressure was applied to the skin.

Using these measurements, we calculated BMI, WHR, and WHtR, and novel anthropometric indices included ABSI [23], WWI [24], BRI [25], BAI [26], and AVI [27].

$$\text{BMI}= \frac{\text{weight (kg)}}{\text{height (m)}^2}$$

$$\text{WHR}= \frac{\text{WC (cm)}}{\text{HC (cm)}}$$

$$\text{WHtR}= \frac{\text{WC (cm)}}{\text{Height (cm)}}$$

$$\text{ABSI}= \frac{WC\ (\text{cm})}{BMI^{\frac{2}{3}} * Height\ (m)^{1/2}}$$

$$\text{WWI}= \frac{\text{WC (cm)}}{\sqrt{weight\ (kg)}}$$

$$\text{BRI} = 364.2 - (365.5 \times \sqrt{1 - (\frac{(WC\ (\text{cm})/2\pi)^2}{(0.5*\text{Height (m)})^2})}$$

$$\text{BAI}= \frac{HC\ (\text{cm})}{height\ (m)^{1.5}} - 18$$

$$\text{AVI}= \frac{\left(2 * WC\ (\text{cm})^2\right) + \left(0.7 * \left(\frac{WC\ (\text{cm})}{HC\ (\text{cm})}\right)^2\right)}{1000}$$

Statistical analysis

All statistical analyses were conducted using SPSS version 26 (IBM Corp. Released 2019. IBM SPSS Statistics for Windows, Version 26.0. Armonk, NY: IBM Corp). Graphical representation in this manuscript were created using GraphPad Prism version 10.4.1 (Windows). Descriptive statistics were used to summarize the demographic characteristics of the study participants, including mean and standard deviation (SD) for continuous variables and frequency and percentages for categorical variables. The Chi-Square and Fisher's exact tests were used for statistical analysis to compare qualitative variables. One-way ANOVA was applied to compare the mean of quantitative variables between three sleep duration groups. Anthropometric and atherogenic indices cutoffs were determined based on the 75th percentile. Sleep duration was treated as the independent variable, while the indices were treated as dependent variables. A multivariable logistic regression model was constructed to investigate the relationship between sleep duration and anthropometric/atherogenic indices, adjusted for potential confounding variables, including age, sex, marital, job, educational status, T2DM, and hypertension. *P*-value < 0.05 is considered significant.

Table 1 Demographic characteristics according to sleep duration

Variables	Sleep duration			*P* value
	< 6 h (*N* = 979)	6–9 h (*N* = 6242)	> 9 h (*N* = 228)	
Age	49.9 ± 7.8	47.2 ± 7.9	46.83 ± 7.95	**< 0.001**
Sex				
Male	389 (39.8)	2525 (40.4)	65 (28.5)	**0.001**
Female	590 (60.2)	3717 (59.6)	163 (71.5)	
Job status				
Employee	349 (35.6)	2423 (38.8)	51 (22.5)	**< 0.001**
Unemployed	513 (52.4)	3236 (51.9)	164 (72.2)	
Retired	117 (12)	581 (9.3)	12 (5.3)	
Marital status				
Single	3 (0.3)	38 (1.2)	0	**0.001**
Married	903 (92.3)	5898 (93.9)	207 (90.8)	
Divorced	13 (1.3)	71 (1.1)	4 (1.8)	
Widow	60 (6.1)	235 (3.8)	17 (7.4)	
Education level				
low	551 (56.3)	3243 (52)	154 (67.8)	**< 0.001**
Moderate	331 (33.9)	2246 (36)	63 (27.8)	
High	96 (9.8)	749 (12)	10 (4.4)	

Data presented as mean ± SD or number (%); One Way ANOVA or Chi-Square tests have been done

Results

A total of 7,455 individuals participated in the study. As outlined in Table 1 and 979 participants (4.3% male) reported sleeping less than 6 h, 6,242 participants (35.4%) slept between 6 and 9 h, and 228 participants (60.3%) reported sleeping more than 9 h. Notably, the majority of those who slept longer were males ($P = 0.005$). Significant differences were observed in sex distribution, marital status ($P = 0.001$), employment, and educational status ($P < 0.001$).

Table 2 presents the results of the ANOVA test. Participants who slept less than 6 h had a higher mean age (49.9 ± 7.8, $P < 0.001$). Those who slept more than 9 h exhibited higher means for WHR (0.93 ± 0.09, $P = 0.001$), WHtR (0.6 ± 0.08, $P = 0.013$), ABSI (0.84 ± 0.06, $P < 0.001$), WWI (11.59 ± 1.06, $P < 0.001$), BRI (5.77 ± 2.03, $P = 0.009$), and AVI (19.03 ± 4.92, $P = 0.029$). No significant differences were identified in BMI, WC, HC, BAI, TC, TG, LDL, HDL, TyG, THR, AIP, CRI I and II, or AC.

The results of the logistic regression are presented in Fig. 1. Participants who slept more than 9 h exhibited a 41.7% greater likelihood of having a reduced ABSI (ABSI < 0.086) with an odds ratio (OR) of 1.417 (95% CI: 1.053–1.907, $P = 0.021$). Additionally, they had a 49.5% increased likelihood of maintaining a lower WWI (WWI < 12) with an OR of 1.495 (95% CI: 1.097–2.036, $P = 0.011$) compared to individuals who slept the recommended amount. Conversely, sleeping less than 6 h was associated with a 40.1% decrease in the odds of having a reduced ABSI (ABSI < 0.086) (OR: 0.599 (95% CI: 0.498–0.721), $P < 0.001$) and a 32.7% lower likelihood of achieving a reduced WWI (WWI < 12) (OR: 0.673 (95% CI: 0.560–0.808), $P < 0.001$). Furthermore, participants sleeping over 9 h had a 31.3% lower likelihood of having a lower BAI (BAI < 37.5) (OR: 0.687 (95% CI: 0.493–0.958), $P = 0.027$) and a 27% reduced likelihood of having lower HDL (HDL < 55 mg/dl) (OR: 0.730 (95% CI: 0.544–0.981), $P = 0.037$). Additionally, sleeping fewer than 6 h increased

the odds of having a lower AVI (AVI < 21) by 19.1% (OR: 0.809 (95% CI: 0.688–0.952), $P = 0.010$).

Discussion

The current study examined the relationships between both prolonged and short sleep duration over a 10-year follow-up period and various anthropometric and atherogenic indices. Compared to participants between 6 and 9 h, those with shorter sleep durations showed a reduced likelihood of experiencing decreases in ABSI, WWI, and AVI. Conversely, individuals who slept for more than 9 h had a higher chance of reducing their ABSI and WWI, but were less likely to see a decrease in their BAI. Notably, AVI was only influenced by shortened sleep duration, while BAI was affected solely by longer sleep duration. Among the atherogenic and lipid indices, participants who slept for over 9 h were less likely to see a reduction in HDL levels.

Generally, there exists a dual relationship between HDL values and sleep duration. A study by Chen et al. indicated that baseline sleep duration had a significant association with HDL values in participants with a normal BMI after four years of follow-up. Conversely, baseline HDL values were not linked to sleep duration during the same follow-up period [32]. The impact of sleep duration on HDL is notable; in this study involving 3,492 participants with normal BMI, sleep duration only influenced HDL and not any other lipid components [32]. Similarly, our current study discovered that sleep duration was also exclusively associated with HDL after a ten-year follow-up. In our study, although direct comparisons of mean HDL levels across groups showed no significant differences, regression analysis indicated that sleep durations over 8 h reduced the risk of low HDL compared to 6 to 9 h. In contrast, shortened sleep duration did not correlate significantly with HDL values. Supporting these findings, a cohort study from China revealed that only sleep durations exceeding 8 h were associated with reduced HDL compared to 7-hour sleep durations. In line with our results, shortened sleep duration did not affect HDL values [33]. Additionally, a meta-analysis indicated that long sleep durations, rather than short ones, were associated with low HDL levels in children and adolescents [34]. Some research, however, has suggested a U-shaped association between nighttime sleep duration and low HDL [35, 36]. This discrepancy may be due to studies that focused specifically on elderly populations [35] or females [36], where inadequate sleep might have more pronounced negative effects on health.

We found no significant relationship between sleep duration and atherogenic indices. This may be attributed to the weak or absent direct connection between sleep duration and specific lipid profile components. A meta-analysis involving 30,033 participants failed to provide supporting evidence for a relationship between sleep duration and the development of dyslipidemia [37]. For instance, in line with our findings, Petrov et al. also reported no association between sleep duration and the CRI-I (total cholesterol/HDL ratio) [16].

Night sleep duration is linked to general and visceral obesity, even after accounting for variations in sleep quality [38]. Grimaldi et al. conducted a meta-analysis showing a significant effect size for sleep variables at baseline and their association with anthropometric indices at follow-up. However, the reverse association was not statistically significant [18]. A recent meta-analysis examining the longitudinal effects of sleep duration on body composition found that shorter sleep duration is associated with an 8% higher risk of abdominal obesity [39]. However, all studies included in this analysis utilized WC, WHR, WHtR, or percentage body fat to evaluate abdominal obesity [39]. WWI is calculated by dividing WC by the square root of weight, offering a weight-independent measure of abdominal obesity [24]. Additionally, ABSI is derived from WC, BMI, and height, aiming to assess abdominal obesity accurately [23]. The current findings

Table 2 Anthropometric and atherogenic indices according to sleep duration

Variables	Sleep duration			*P*-value
	< 6 h (*N* = 979)	6–9 h (*N* = 6242)	> 9 h (*N* = 228)	
BMI, kg/m^2	28.27 ± 4.62	28.46 ± 4.89	28.45 ± 4.72	0.56
WC, cm	92.66 ± 10.96	91.88 ± 10.73	92.23 ± 10.69	0.1
HC, cm	101.53 ± 10.20	101.96 ± 11.3	101.34 ± 9.3	0.4
WHR	0.91 ± 0.08	0.91 ± 0.07	0.93 ± 0.09	**0.001**
WHtR	0.59 ± 0.07	0.59 ± 0.08	0.6 ± 0.08	**0.013**
ABSI	0.81 ± 0.06	0.82 ± 0.07	0.84 ± 0.06	**< 0.001**
WWI	11.2 ± 1.01	11.25 ± 1.03	11.59 ± 1.06	**< 0.001**
BRI	5.41 ± 1.79	5.39 ± 1.87	5.77 ± 2.03	**0.009**
BAI	33.53 ± 6.3	33.17 ± 6.35	33.67 ± 6.13	0.14
AVI	18.17 ± 4.35	18.44 ± 4.54	19.03 ± 4.92	**0.029**
TC, mg/dl	205.43 ± 47.07	205.73 ± 44.88	212.08 ± 42.9	0.11
TG, mg/dl	145.3 ± 81.19	148.08 ± 63.32	155.71 ± 89.08	0.25
HDL, mg/dl	48.43 ± 10.65	48.34 ± 10.5	49.74 ± 10.67	0.14
LDL, mg/dl	115.37 ± 35.65	115.86 ± 33.98	118.02 ± 33.19	0.58
LDL TG ratio	0.95 ± 0.44	0.95 ± 0.45	0.93 ± 0.44	0.75
TyG	8.88 ± 0.61	8.86 ± 0.61	8.9 ± 0.63	0.49
THR	3.23 ± 2.31	3.3 ± 2.4	3.42 ± 2.55	0.57
AIP	0.43 ± 0.24	0.44 ± 0.25	0.44 ± 0.26	0.75
CRI_I	4.65 ± 1.13	4.61 ± 1.13	4.63 ± 1.15	0.58
CRI_II	2.45 ± 0.79	2.47 ± 0.78	2.45 ± 0.78	0.82
AC	3.65 ± 1.1	3.61 ± 1.13	3.63 ± 1.15	0.58

BMI: Body Mass Index, WC: Waist Circumference, HC: Hip Circumference, WHR: Waist-to-Hip Ratio, WHtR: Waist-to-Height Ratio, BAI: Body Adiposity Index, BRI: Body Round Index, ABSI: A Body Shape Index, AVI: Abdominal Volume Index, WWI: Weight-Adjusted-Waist Index, TC: Total cholesterol, TG: Triglyceride, HDL: High-density lipoprotein, LDL: low-density lipoprotein, TyG: Triglyceride-Glucose Index, THR: Triglyceride: HDL Ratio, AIP: Atherogenic Index of plasma, CRI_I: Castelli Risk Index I, CRI_II: Castelli Risk Index II, AC: Atherogenic Coefficient

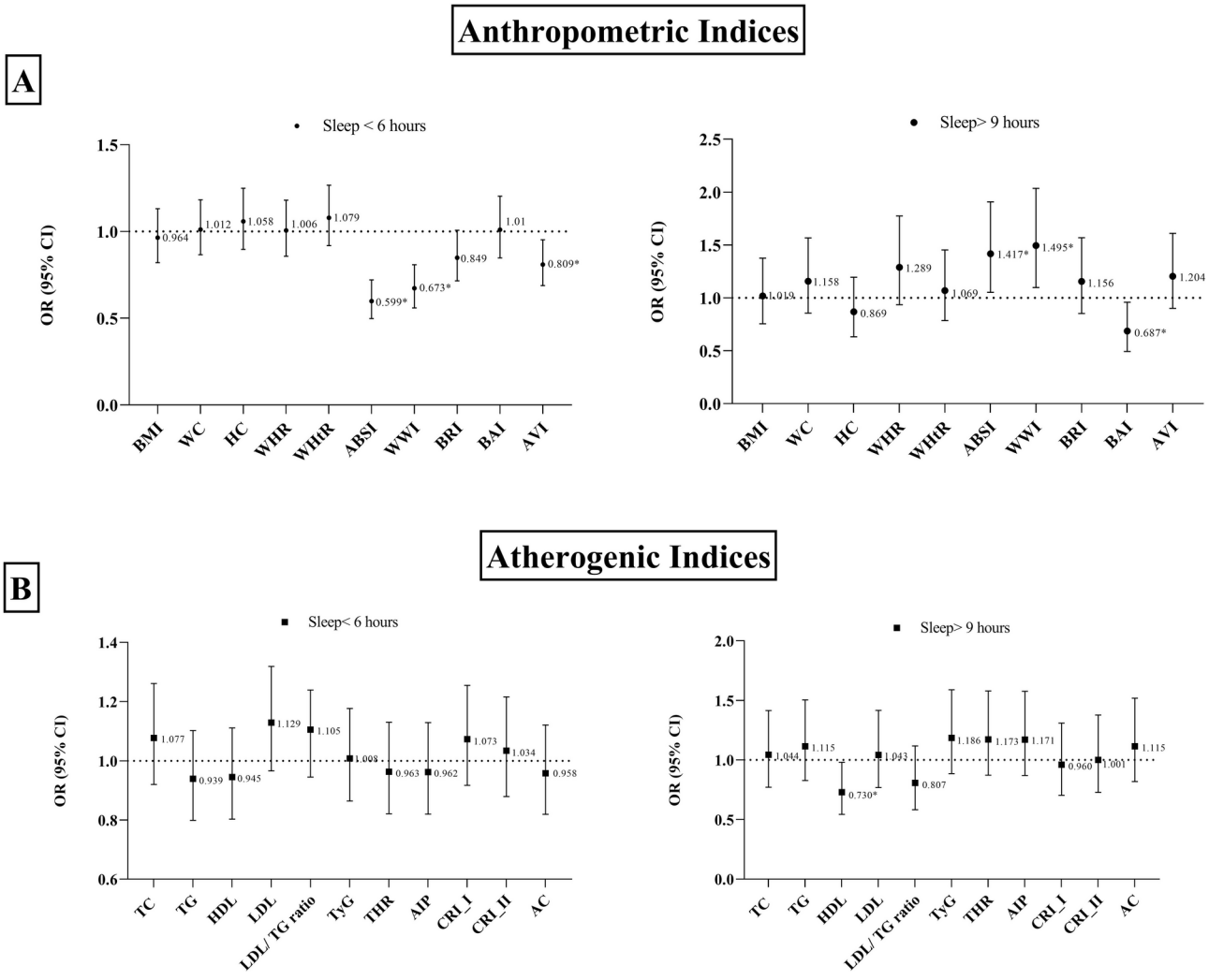


Fig. 1 Impact of Baseline Sleep Duration on anthropometric indices (**A**) and atherogenic indices (**B**); *$p < 0.05$; Abbreviations: BMI: Body Mass Index, WC: Waist Circumference, HC: Hip Circumference, WHR: Waist-to-Hip Ratio, WHtR: Waist-to-Height Ratio, BAI: Body Adiposity Index, BRI: Body Round Index, ABSI: A Body Shape Index, AVI: Abdominal Volume Index, WWI: Weight-Adjusted-Waist Index, TG: Triglyceride, TC: Total cholesterol, HDL: High-density lipoprotein, LDL: low-density lipoprotein, TyG: Triglyceride-Glucose Index, THR: Triglyceride HDL Ratio, AIP: Atherogenic Index of plasma, CRI_I: Castelli Risk Index I, CRI_II: Castelli Risk Index II, AC: Atherogenic Coefficient

indicate that, among various anthropometric indices, the odds of reduction in ABSI and WWI increase with longer sleep duration. This study is the first to investigate the long-term effects of sleep duration on these novel anthropometric indices. Nonetheless, analyses of the US National Health and Nutrition Examination Survey (NHANES) revealed that ABSI and WWI are positively associated with the risk of obstructive sleep apnea (OSA) [40–42]. Furthermore, another study identified a connection between WWI and Circadian syndrome, of which sleep deprivation is a defining component [43].

AVI is another index that provides a more accurate measurement of abdominal obesity than WC. For instance, a study found that AVI was superior to WC in predicting metabolic syndrome [44]. In the current research, it was discovered for the first time that short sleep duration is a risk factor for increased AVI levels. Interestingly, sleep durations exceeding nine hours did not significantly influence this index. This suggests that AVI is more affected by short sleep durations than longer ones. While AVI, WWI, and ABSI measure abdominal obesity, AVI specifically assesses abdominal volume relative to body surface area [27]. This focus on abdominal volume may explain why sleep patterns less influence AVI, showing minimal impact from longer sleep durations. Additionally, the strength of its relationship with sleep duration appears to be weaker than that of the other two abdominal obesity indicators.

BAI demonstrates superior sensitivity in identifying and categorizing obesity compared to BMI when utilizing dual-energy X-ray absorptiometry (DXA) as the reference method [26]. This index is calculated using HC

and height, allowing for an estimation of overall body fat percentage without incorporating weight [26]. While prior studies have investigated the inverse relationship between HC and sleep duration [19], research examining the effects of sleep on BAI remains limited. Evidence indicates that in addition to short sleep duration being a risk factor for excessive abdominal adiposity, individuals who sleep excessively long are twice as likely as those with normal sleep patterns to exhibit abdominal fat [20]. Our current study corroborates that prolonged sleep duration is a risk factor for BAI among all anthropometric indices analyzed. Furthermore, a cross-sectional study involving 10,619 UK adolescents aged 13–15 revealed a U-shaped relationship between sleep duration and overweight/obesity [44]. Additional findings also indicate a U-shaped association between sleep duration and body weight [45] or weight gain [46] in adults.

Insufficient sleep can adversely affect body composition through several mechanisms. One key factor is hormonal dysregulation, which results in elevated ghrelin and reduced leptin levels, increasing appetite and hunger, thereby promoting weight gain [47, 48]. Lack of sleep is also linked to heightened cortisol production, contributing to weight accumulation, particularly around the abdomen [49–52]. Additionally, fatigue from inadequate sleep often leads to decreased physical activity, as research indicates reduced spontaneous movement following sleep restriction [53, 54] Moreover, short sleep durations may activate inflammatory pathways and influence the expression of genes related to metabolism and oxidative stress [55, 56]. Insufficient sleep is associated with impaired decision-making, leading to higher caloric intake from snacks, especially among adolescents, who have a greater risk of consuming excess calories and fast food [57–59]. Overall, inadequate sleep initiates physiological and behavioral changes predisposing individuals to obesity and related body composition changes.

In addition to insufficient sleep, excessive sleep can lead to adverse health outcomes [60]. Prolonged sleep duration may be associated with a reduced metabolic rate, as energy expenditure decreases during sleep [61]. Individuals who sleep for extended periods often tend to skip breakfast, which can contribute to weight gain and abdominal obesity [62]. Therefore, it is reasonable to understand why higher sleep duration in the current study increased the BAI.

Strength and limitations

The present study marks a pioneering effort in conducting a prospective cohort analysis to elucidate the influence of sleep on specific parameters. The study's robust sample size and prospective methodology contribute significantly to its scientific merit. Nevertheless, the reliance on self-reported questionnaires for nocturnal sleep duration data introduces a potential source of bias. Evidence indicates that, in a community sample, sleep duration assessed through retrospective questionnaires tends to be about 20–30 min longer than measurements obtained from polysomnography, which is a more accurate tool [63]. Our inability to access more precise measures of sleep duration may have influenced the results.

Conclusion

This prospective cohort study, conducted within the MASHAD study population in northeast Iran, aimed to investigate the impact of sleep duration on long-term changes in novel anthropometric and atherogenic indices over a 10-year follow-up period. The findings revealed that those who slept less than 6 h were likelier to experience an increase in ABSI, WWI, and AVI compared to participants who slept between 6 and 9 h. In contrast, individuals with over 9 h of sleep showed a greater tendency to decrease their ABSI and WWI, although they had an increase in their BAI. Additionally, among the atherogenic and lipid indices, participants who slept for more than 9 h exhibited elevated levels of HDL. This study suggests that sleep duration has a pronounced effect on novel anthropometric indices as observed through a longitudinal approach.

Acknowledgements

We gratefully acknowledge Mashhad University of Medical Sciences and the participants who collaborated in this study.

Author contributions

F.K. was responsible for conceptualization, writing the original draft, and designing the graphical abstract. M.I. contributed to writing the original draft. S.D. (corresponding author) performed data analysis and provided supervision. M.G.-M. oversaw the study design. M.M. provided scientific consulting. M.J., V.M., and M.M.B. were involved in data gathering. All authors reviewed and approved the manuscript.

Funding

This work was supported by the Mashhad University of Medical Sciences [grant number: 4021664].

Data availability

Data and materials are available from the corresponding author, Dr. Susan Darroudi.

Declarations

Ethical approval and consent to participate

All individuals were well informed and their written consent was drawn. Accordingly, the study protocol was validated by the Ethics Committee of the Mashhad University of Medical Sciences (MUMS) and the Institutional Review Board of Mashhad University Medical Center (approval number IR.MUMS.MEDICAL.REC.1398.228).

Consent for publication

It does not apply to the Consent of Image Publication for this manuscript. The figure was designed only in this manuscript for presenting the results of the current paper.

Competing interests

The authors declare no competing interests.

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Received: 23 April 2024 / Accepted: 21 April 2025
Published online: 01 May 2025

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