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Sleep duration and incidence of hypertension in the Bandare-Kong cohort study: findings from PERSIAN cohort study

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Abstract

Background Hypertension remains a major global public health concern. Emerging evidence suggests that sleep duration may play a significant role as a modifiable risk factor; however, findings across studies have been inconsistent. This study aims to examine the association between sleep duration and hypertension in a cohort of adults residing in southern Iran.

Methods This population-based study was conducted among adults aged 35–70 years from the Bandare-Kong Non-Communicable Diseases (BKNCD) Cohort, part of the Prospective Epidemiological Research Studies in Iran (PERSIAN). Blood pressure was measured using a standardized protocol, and hypertension was defined as systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg or the use of antihypertensive medication. Sleep duration was self-reported through a structured questionnaire. Logistic and Cox regression models were applied to assess the association between sleep duration and hypertension. Analyses were performed using R software, with p-values < 0.05 considered statistically significant.

Results A total of 3,737 participants were included in the analyses, among whom 1,332 individuals without hypertension at baseline were followed for six years. At baseline, logistic regression analysis showed that women under the age of 50 who reported sleeping less than 7 h per night had 1.82 times higher odds of having hypertension compared to those who slept 7–8 h per night (OR = 1.82; 95% CI: 1.31–2.15; $p = 0.042$). Among participants aged 50 years and older, both women and men with short sleep duration (< 7 h/night) had significantly increased odds of hypertension compared to their counterparts who slept 7–8 h per night (women: OR = 2.68; 95% CI: 1.52–3.88; $p < 0.001$; men: OR = 2.35; 95% CI: 1.45–4.00; $p = 0.003$). Furthermore, Cox proportional hazards modeling indicated that individuals with either short (< 7 h) or long (> 8 h) sleep duration had a 1.91-fold increased risk of developing hypertension over the study period compared to those with normal sleep duration (7–8 h/night) (HR = 1.91; 95% CI: 1.84–3.50; $p = 0.041$).

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Conclusions Both short sleep duration (< 7 h/night) and long sleep duration (> 8 h/night) were associated with an increased risk of hypertension. These findings highlight the importance of adequate sleep as a potential modifiable factor in hypertension prevention. Future research is warranted to further explore the combined impact of sleep duration and sleep quality on the development of hypertension.

Keywords Sleep duration, Hypertension, Cohort study, Prospective epidemiological research studies in IrAN (PERSIAN)

Background

Hypertension, defined as systolic blood pressure (SBP) ≥ 140 mmHg, diastolic blood pressure (DBP) ≥ 90 mmHg, or the use of antihypertensive medications, is a growing global health concern. The global prevalence of hypertension among individuals aged 30–79 years is approximately 33%, while national data report a prevalence of 26.26% among Iranian children, adolescents, and adults [1, 2]. Hypertension is a major risk factor for a range of cardiovascular complications, including stroke, heart failure, myocardial infarction, and chronic kidney disease [3, 4]. These conditions contribute significantly to global mortality and morbidity [5]. According to the World Health Organization (WHO), high blood pressure affects over one billion adults and is responsible for more than nine million deaths annually [6]. Therefore, identifying effective strategies for hypertension prevention and management is essential. In particular, there is an urgent need to explore low-cost, accessible interventions, especially in developing and low-income countries [5].

Behavioral modifications, particularly lifestyle interventions such as adopting healthy dietary patterns and engaging in regular physical activity, are widely recommended as first-line strategies for managing and preventing hypertension [7]. While numerous physiological mechanisms involved in blood pressure regulation have been extensively investigated, the role of sleep as a modifiable risk factor in the pathophysiology of hypertension has only recently gained increasing scientific interest [8]. Adequate sleep, typically defined as 7 to 9 h per night, is essential for maintaining overall health and reducing the risk of chronic diseases [9]. Both short sleep duration (< 7 h) and long sleep duration (>9 h) have been associated with adverse health outcomes, including increased risk of cardiovascular disease and diabetes [10]. Additionally, prolonged sleep (>8 h) and poor sleep quality have been linked to obesity [11, 12], a key component of metabolic syndrome and a known risk factor for type 2 diabetes. Individuals with metabolic syndrome or diabetes are, in turn, at higher risk of developing hypertension [13], highlighting the potential indirect pathway through which suboptimal sleep may influence blood pressure regulation.

Previous studies investigating the association between sleep and hypertension have yielded inconsistent findings. Some studies have identified significant associations between hypertension and sleep apnea [14, 15] as well as both short and long sleep durations [16, 17]. In contrast, a longitudinal analysis and a cross-sectional study have found no significant associations between hypertension and sleep duration [18, 19]. Some evidence also suggests that geographic variation may influence the relationship between sleep and blood pressure, suggesting that cultural, environmental, or genetic factors may play a role [20]. Notably, a study identified a significant association between sleep duration and metabolic syndrome among residents of Bandare Kong city, a coastal region in southern Iran [21].

Given the inconsistencies in previous research findings and the rising prevalence of hypertension in Iran and globally, it is essential to investigate the potential impact of sleep duration on blood pressure as a way of informing public health interventions. Notably, no prior study has examined the association between sleep duration and blood pressure within the southern Iranian population. Using data from the Bandare-Kong Non-Communicable Diseases (BKNCD) cohort, this study aims to explore the association between sleep duration and hypertension risk among adults in southern Iran.

Methods

Study design and sampling

The present study is embedded within the Bandare-Kong Non-Communicable Diseases (BKNCD) cohort, one of the regional branches of the Prospective Epidemiological Research Studies in Iran (PERSIAN), which was launched in 2016. The BKNCD cohort is a population-based prospective study designed to investigate the prevalence, incidence, and risk factors of non-communicable chronic diseases in southern regions of Iran. The full methodology and rationale of the BKNCD cohort have previously been described in detail [22].

In the BKNCD cohort, 4,056 adults aged 35–70 years residing in Bandar-e-Kong city were initially enrolled. After applying inclusion and exclusion criteria, 3,737 individuals were eligible for the present analyses. Among these, 2,676 participants were free of hypertension at baseline. For prospective analyses, a subset of 1,332 non-hypertensive individuals was selected for follow-up over a six-year period (2,238 days) to identify incident cases of hypertension. Figure 1 illustrates the stepwise participant

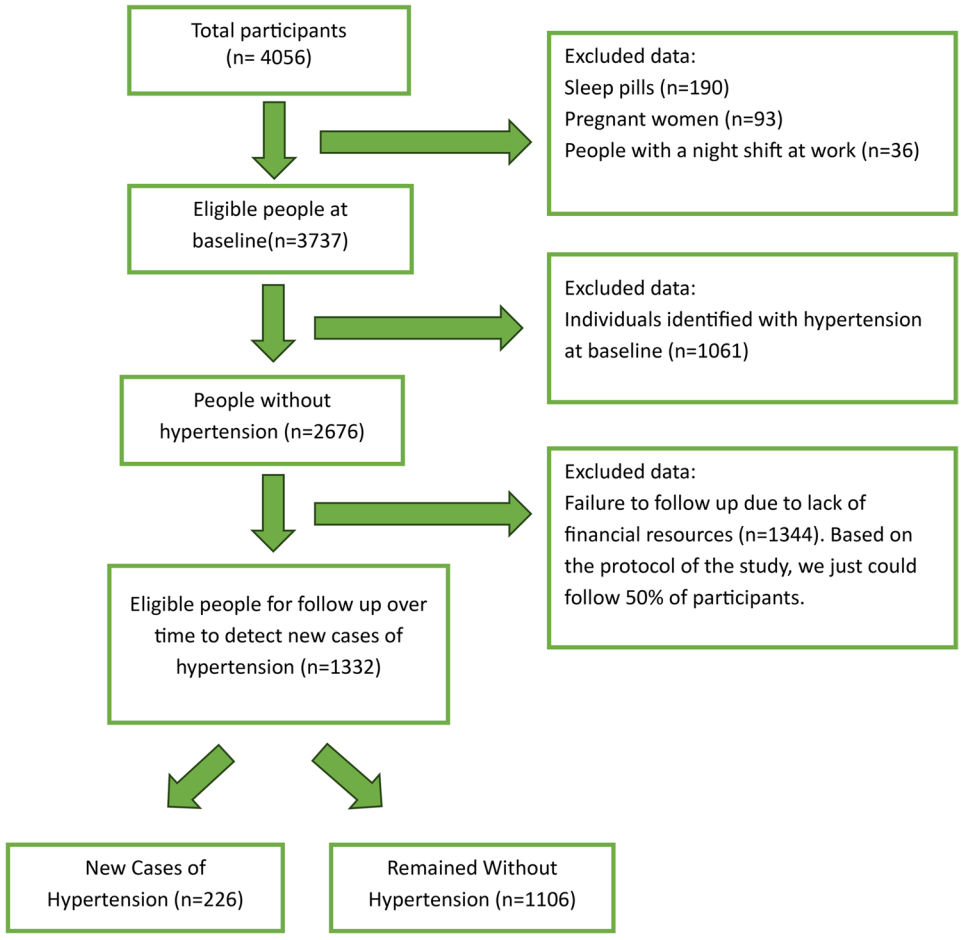

Fig. 1 Sampling flowchart

selection process, including exclusion criteria applied at each stage.

Baseline data collection

Baseline data were collected through face-to-face interviews conducted by trained personnel using standardized questionnaires that covered a wide range of variables, including sociodemographic characteristics, lifestyle behaviors, dietary patterns, physical activity, sleep habits, and medical history.

Follow-Up data collection

Participants were followed annually over six years through structured telephone interviews conducted by trained staff. During each follow-up call, participants were asked about any newly diagnosed medical conditions, including hypertension, as well as any hospitalizations or major health events. For self-reported cases of hypertension, if the participant possessed relevant medical records, the study team collected supporting documentation. Two internists independently reviewed these documents to confirm the diagnosis, with a third physician resolving any discrepancies. Therefore, incident hypertension cases included only those that were self-reported and subsequently confirmed through medical records and physician review.

Blood pressure measurement

Blood pressure was measured by trained personnel using a standard mercury sphygmomanometer with an appropriately sized cuff. Measurements were taken in a seated position, with the participant's feet flat on the floor and the arm supported at heart level, following at least five minutes of rest. Two blood pressure readings were obtained at least five minutes apart, and their average was used for analysis. Hypertension was defined as a SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg or current use of antihypertensive medications.

Assessment of sleep duration

Sleep duration data were collected through face-to-face interviews using a series of structured questions, including: "What time do you usually fall asleep?", "On average, how long after going to bed do you actually fall asleep?",

"What time do you usually wake up in the morning?", "Have you had night shifts at work?", and "Do you regularly use sleeping pills?". Based on participants' responses and in accordance with previous research [23, 24], sleep duration was categorized into three groups: short sleep (< 7 h per night), normal sleep (7–8 h per night), and long sleep (>8 h per night).

Statistical analysis

The dataset was screened for extreme values. Sleep duration measurements exceeding Q3 + 3×IQR or falling below Q1 – 3×IQR were flagged as outliers and subsequently excluded (Q1: first quartile, Q3: third quartile, IQR: interquartile range). After cleaning, the minimum and maximum sleep duration values were 2 and 12 h, respectively. Baseline characteristics of participants were reported using frequency and percentage, stratified by sleep duration categories (< 7 h, 7–8 h, > 8 h) and hypertension status (yes/no). The chi-square test was employed to examine associations between categorical variables. Logistic regression analysis was conducted to assess the cross-sectional association between sleep duration and hypertension at baseline. To evaluate the longitudinal relationship between sleep duration and incident hypertension, Cox proportional hazards models were applied after verifying the proportional hazards assumption using Schoenfeld residuals. Variables with a P-value < 0.25 in univariable models were included in the multivariable analysis. We reported adjusted odds ratios (aOR) from logistic regression model and adjusted hazard ratios (aHR) from Cox regression models. All statistical analyses were performed using R software (version 4.4.1). A p-value of < 0.05 was considered statistically significant.

Results

The mean age of participants at baseline was 48.61 ± 8.51 years. Baseline characteristics of the 3,737 participants, stratified by sleep duration and hypertension status, are presented in Table 1. Among the 1,061 individuals with hypertension (28.4% of the total sample), 770 (20.6%) participants reported short sleep duration (< 7 h/night), 243 (6.5%) had normal sleep duration (7–8 h/night), 45 (1.2%) reported long sleep duration (> 8 h/night), and 3(0.1%) hypertensive cases had missing sleep duration data.

Adjusted for education, marital status, occupation, Socio-economic status, BMI, smoking, hookah use, salt intake, and physical exercise. The Logistic regression model was used.

As shown in Table 2, after adjusting for potential confounders, short sleep duration (< 7 h/night) was significantly associated with increased odds of hypertension across several subgroups. Among men aged 50 years and older, those with short sleep had 2.35 times higher odds of hypertension compared to those sleeping 7–8 h (OR = 2.35; 95% CI: 1.45–4.00; $p = 0.003$). For women under 50 years, short sleep duration was associated with 1.82 times greater odds of hypertension (OR = 1.82; 95% CI: 1.31–2.15; $p = 0.042$). Similarly, in women aged 50 years and older, short sleep was linked to a 2.68-fold increase in the odds of hypertension (OR = 2.68; 95% CI: 1.52–3.88; $p < 0.001$).

During the 6-year follow-up period (the median follow-up time was 1738 days, range: 219–2238 days), among 1,332 participants who were normotensive at baseline, who did not work night shifts, were not pregnant, and did not regularly use sleeping pills, 226 individuals developed hypertension, yielding a cumulative incidence of 17.0% (95% CI: 15.0%–19.0%).

Table 3 presents the baseline characteristics of participants included in the longitudinal analysis. Both the mean and median (50th percentile) sleep durations were lower among those who developed hypertension compared to those who remained normotensive; however, this difference was not statistically significant ($p > 0.05$).

Adjusted for age, sex, education, marital status, occupation, Socio-economic status, BMI, smoking, hookah use, salt intake, and physical exercise. Mean (SE): mean for survival time (Standard Error). Given that the frequency of people with hypertension who slept more than 8 h per night was low (only 8 cases), sleep duration was divided into two groups (< 7 or > 8 h; 7–8 h) instead of three groups.

As shown in Fig. 2, participants with normal sleep duration (7–8 h/night) demonstrated the highest hypertension-free survival probability throughout the follow-up period. The mean survival time and the results of the multivariable Cox proportional hazards model are presented in Table 4. After adjusting for potential confounders, individuals with short (< 7 h) or long (> 8 h) sleep duration had a 1.91-fold higher hazard of developing hypertension over the study period compared to those with normal sleep duration (7–8 h) (HR = 1.91; 95% CI: 1.02–3.58; $p = 0.041$).

According to Table 5, after accounting for potential confounders, the protective effect of sleep on hypertension risk varied by age and gender. In men aged 50 and older, each additional hour of sleep was linked to a 27% lower risk of developing hypertension (HR = 0.73; 95% CI: 0.57–0.92; $p = 0.004$), whereas no significant effect was observed in younger men. For women, increased sleep was beneficial across both age groups, though the magnitude differed. Women under 50 experienced a 24% reduction in hypertension risk per extra hour of sleep (HR = 0.76; 95% CI: 0.57–0.94; $p = 0.008$), while those aged 50 and above saw the greatest benefit, with a 32% risk reduction (HR = 0.68; 95% CI: 0.59–0.90; $p < 0.001$).

Table 1 Participants' characteristics at baseline stratified according to hypertension status and sleep duration

Characteristic	Group	Total (n=3737)	Without Hypertension (n=2676;71.6%)				Hypertension (n=1061;28.4%)			
			Sleep duration (n=2662;71.2%, missing data=14;0.4%)				Sleep duration (n=1058;28.3%, missing data=3;0.1%)			
			<7 hours/night (n=1913;51.2%)	7–8 hours/night (n=599;16.0%)	>8 hours/night (n=150;4.0%)	P value	<7 hours/night (n=770;20.6%)	7–8 hours/night (n=243;6.5%)	>8 hours/night (n=45;1.2%)	*P* value
Sex	Male	1499(40.1%)	784(21.0%)	245(6.5%)	42(1.1%)	0.007	320(8.6%)	90(2.4%)	12(0.3%)	0.082
	Female	2238(59.9%)	1129(30.2%)	354(9.5%)	108(2.9%)		450(12.0%)	153(4.1%)	33(0.9%)	
Age	<50	2155(57.7%)	1271(34.0%)	423(11.3%)	118(3.1%)	0.003	234(6.3%)	80(2.1%)	17(0.5%)	0.479
	≥50	1582(42.3%)	642(17.2%)	176(4.7%)	32(0.9%)		536(14.3%)	163(4.4%)	28(0.7%)	
Education	<6	2571(68.8%)	1214(32.5%)	389(10.4%)	101(2.7%)	0.871	614(16.4%)	206(5.5%)	36(0.97%)	0.478
	6-12	859(23.0%)	505(13.5%)	153(4.1%)	35(0.9%)		125(3.4%)	30(0.8%)	8(0.2%)	
	>12	307(8.2%)	194(5.2%)	57(1.5%)	14(0.4%)		31(0.8%)	7(0.2%)	1(0.03%)	
Marital status	Single	410(11.0%)	197(5.3%)	49(1.3%)	29(0.8%)	<0.001	93(2.5%)	29(0.8%)	11(0.3%)	0.049
	Married	3327(89.0%)	1716(45.9%)	550(14.7%)	121(3.2%)		677(18.1%)	214(5.7%)	34(0.9%)	
Occupation	No	2155(57.7%)	1000(26.8%)	329(8.8%)	93(2.5%)	0.051	521(13.9%)	161(4.3%)	35(0.9%)	0.313
	Yes	1582(42.3%)	913(24.4%)	270(7.2%)	57(1.5%)		249(6.7%)	82(2.2%)	10(0.3%)	
Socio-economic status	Poor	1427(38.2%)	679(18.2%)	230(6.17%)	78(2.1%)	<0.001	310(8.3%)	108(2.9%)	22(0.6%)	0.566
	Middle	779(20.8%)	392(10.5%)	151(4.0%)	26(0.7%)		158(4.2%)	46(1.2%)	6(0.2%)	
	Rich	1513(40.5%)	840(22.45%)	217(5.8%)	46(1.2%)		302(8.1%)	89(2.4%)	17(0.4%)	
	Missing data	18(0.5%)	2(0.05%)	1(0.03%)	0(0.0%)		0(0.0%)	0(0.0%)	0(0.0%)	
Smoking	No	3277(87.7%)	1692(45.3%)	517(13.8%)	133(3.5%)	0.360	662(17.7%)	215(5.8%)	43(1.15%)	0.130
	Yes	460(12.3%)	221(5.9%)	82(2.2%)	17(0.5%)		108(2.9%)	28(0.7%)	2(0.05%)	
Hookah Use	No	3116(83.4%)	1616(43.2%)	511(13.7%)	121(3.2%)	0.373	633(16.9%)	185(4.9%)	36(1.0%)	0.111
	Yes	621(16.6%)	297(8.0%)	88(2.3%)	29(0.8%)		137(3.7%)	58(1.6%)	9(0.2%)	
Salt intake	Light (<2.99 gr/day)	663(17.7%)	291(7.8%)	92(2.5%)	26(0.7%)	0.427	173(4.6%)	68(1.8%)	12(0.3%)	0.375
	Moderate (3 gr/day)	2223(59.6%)	1179(31.6%)	352(9.4%)	84(2.2%)		446(12.0%)	136(3.64%)	25(0.7%)	
	High (>3 gr/day)	831(22.2%)	438(11.7%)	155(4.1%)	40(1.1%)		151(4.0%)	38(1.03%)	8(0.2%)	
	Missing data	20(0.5%)	5(0.1%)	0(0.0%)	0(0.0%)		0(0.0%)	1(0.03%)	0(0.0%)	
Physical activity (METs/day)	Low (25-36.44)	926(24.8%)	382(10.2%)	148(4.0%)	56(1.5%)	<0.001	239(6.4%)	79(2.1%)	21(0.6%)	0.020
	Moderate (36.45-42.74)	1857(49.7%)	969(25.9%)	312(8.3%)	68(1.8%)		362(9.7%)	129(3.47%)	17(0.4%)	
	Vigorous (>= 42.75)	930(24.9%)	558(15.0%)	139(3.7%)	25(0.67%)		167(4.45%)	34(0.9%)	7(0.2%)	
	Missing data	24(0.6%)	4(0.1%)	0(0.0%)	1(0.03%)		2(0.05%)	1(0.03%)	0(0.0%)	

Table 1 (continued)

Characteristic	Group	Total (n=3737)	Without Hypertension (n=2676;71.6%)				Hypertension (n=1061;28.4%)			
			Sleep duration (n=2662;71.2%, missing data=14;0.4%)				Sleep duration (n=1058;28.3%, missing data=3;0.1%)			
			<7 hours/night (n=1913;51.2%)	7–8 hours/night (n=599;16.0%)	>8 hours/night (n=150;4.0%)	P value	<7 hours/night (n=770;20.6%)	7–8 hours/night (n=243;6.5%)	>8 hours/night (n=45;1.2%)	*P* value
Body mass index (BMI)	<25	1340(35.8%)	753(20.2%)	244(6.5%)	68(1.8%)	0.355	194(5.2%)	66(1.8%)	10(0.3%)	0.731
	≥25	2383(63.8%)	1151(30.8%)	354(9.47%)	82(2.2%)		574(15.35%)	177(4.7%)	35(0.9%)	
	Missing data	14(0.4%)	9(0.2%)	1(0.03%)	0(0.0%)		2(0.05%)	0(0.0%)	0(0.0%)	

Missing sleep data: 17 people (14 people in the normotensive group + 3 people in the hypertensive group)

In all cells, % of total (n=3737) has been reported

P-values were calculated using the Chi-square test

Table 2 The relationship between hypertension and categorical sleep duration at baseline based on sex and age

Dependent Variable: Hypertension (Yes/No)	Male		Female	
	Odds Ratio (95%CI)	P value	Odds Ratio (95%CI)	*P* value
Age<50				
Sleep duration				
<7 hours/night	1.67(0.84,3.35)	0.145	1.82(1.31,2.15)	0.042
7–8 hours/night	Reference Category		Reference Category	
>8 hours/night	1.03(0.42,2.52)	0.469	1.40(0.73,2.70)	0.308
Age≥50				
Sleep Duration				
<7 hours/night	2.35(1.45,4.00)	0.003	2.68(1.52,3.88)	<0.001
7–8 hours/night	Reference Category		Reference Category	
>8 hours/night	1.23(0.43,3.52)	0.693	1.28(0.68,3.61)	0.682

Adjusted for education, marital status, occupation, Socio-economic status, BMI, smoking, hookah use, salt intake, and physical exercise. The Logistic regression model was used

The Logistic regression model was used

Discussion

The findings of this cohort study contribute essential evidence to the growing body of literature investigating the association between sleep duration and hypertension. Our results indicate that short sleep duration (< 7 h per night) is significantly associated with a higher prevalence of hypertension, particularly among men aged 50 and older, as well as among women across age groups. These findings are consistent with previous studies suggesting that insufficient sleep is a modifiable risk factor for hypertension [10]. For instance, a cross-sectional study reported a 25% increase in the odds of hypertension among individuals sleeping fewer than 6 h per night [25]. Similarly, a systematic review and meta-analysis of 13 cross-sectional and longitudinal studies involving over 225,000 participants identified a significant association between short sleep duration and elevated blood pressure [26]. Another meta-analysis confirmed that sleep duration of less than 7 h per night is associated with an increased risk of hypertension [27]. Feng et al. reported a significant association between short sleep duration and diastolic blood pressure; however, unlike our findings and previous studies, this study did not find an association between sleep duration and systolic blood pressure. This discrepancy could be due to the differences in the blood pressure measurement or the sample's demographic variables [28].

Several physiological mechanisms may underlie the relationship between short sleep and elevated blood pressure. These include autonomic dysregulation, hormonal imbalances (e.g., increased ghrelin and decreased leptin), and elevated inflammatory and oxidative stress markers. These biological disruptions may contribute to obesity, insulin resistance, metabolic syndrome, and type 2 diabetes, all of which are established risk factors for hypertension [13]. Additionally, individuals with insufficient sleep are more likely to engage in unhealthy behaviors, including physical inactivity, poor dietary habits, smoking, and alcohol consumption, which further elevate the risk of hypertension [29].

Further subgroup analyses revealed that short sleep duration was associated with a higher risk of prevalent hypertension among women across all age groups. This sex-specific association aligns with findings from a large-scale analysis involving over 700,000 adults, which reported a stronger association between short sleep duration and hypertension in women compared to men [30]. Similarly, a systematic review and meta-analysis of sixteen cohort studies concluded that women were more susceptible to developing hypertension in response to insufficient sleep than men [31]. Consistent with these observations, our findings indicated that each additional hour of sleep was associated with a significantly lower risk of developing hypertension in women across both age groups, while no significant association was observed in younger men. These sex- and age-specific differences may reflect the protective cardiovascular, autonomic, and anti-inflammatory effects of estrogen. In the context of estrogen decline after menopause, longer sleep may

Table 3 Participants' characteristics who were followed over time ($n = 1332$)

Characteristic	Group	Total (Valid Percent)	Without Hypertension ($n = 1106$;83.0%)	New cases of Hypertension ($n = 226$;17.0%)	*P* value
Sex	Male	562(42.5%)	486(36.8%)	76(5.7%)	0.020
	Female	759(57.5%)	620(47.0%)	139(10.5%)	
Age	< 50	865(65.5%)	762(57.7%)	103(7.8%)	< 0.001
	≥50	456(34.5%)	344(26.0%)	112(8.5%)	
Education	< 6	851(64.4%)	692(52.4%)	159(12.0%)	0.006
	6–12	344(26.1%)	304(23.1%)	40(3.0%)	
	>12	126(9.5%)	110(8.3%)	16(1.2%)	
Marital status	Single	125(9.5%)	101(7.7%)	24(1.8%)	0.372
	Married	1196(90.5%)	1005(76.1%)	191(14.4%)	
Occupation	No	699(52.9%)	563(42.6%)	136(10.3%)	< 0.001
	Yes	622(47.1%)	543(41.1%)	79(6.0%)	
Socio-economic status	Poor	481(36.5%)	413(31.4%)	68(5.1%)	0.110
	Middle	286(21.7%)	229(17.4%)	57(4.3%)	
	Rich	550(41.8%)	460(35.0%)	90(6.8%)	
Residence type	Urban	1154(87.4%)	979(74.2%)	175(13.2%)	0.007
	Rural	167(12.6%)	127(9.6%)	40(3.0%)	
Menopausal women	No	541(71.6%)	457(60.5%)	84(11.1%)	0.002
	Yes	215(28.4%)	160(21.2%)	55(7.2%)	
Smoking	No	1171(88.6%)	982(74.3%)	189(14.3%)	0.725
	Yes	150(11.4%)	124(9.4%)	26(2.0%)	
Hookah Use	No	1126(85.2%)	945(71.5%)	181(13.7%)	0.674
	Yes	195(14.8%)	161(12.2%)	34(2.6%)	
Salt intake	Light (< 2.99 gr/day)	190(14.4%)	152(11.5%)	38(2.9%)	0.125
	Moderate (3 gr/day)	808(61.4%)	673(51.2%)	135(10.2%)	
	High (> 3 gr/day)	317(24.2%)	275(21.0%)	42(3.2%)	
Physical activity (METs/day)	Low (25–36.44.44)	269(20.4%)	227(17.2%)	42(3.2%)	0.936
	Moderate (36.45–42.74)	664(50.5%)	554(42.1%)	110(8.4%)	
	Vigorous (> = 42.75)	383(29.1%)	320(24.3%)	63(4.8%)	
Body mass index (BMI)	<25	550(41.7%)	492(37.3%)	58(4.4%)	< 0.001
	≥25	768(58.3%)	612(46.5%)	156(11.8%)	
Sleep duration	< 7 h/night	944(71.6%)	784(59.5%)	160(12.1%)	0.040
	7–8 h/night	302(22.9%)	255(19.3%)	47(3.6%)	
	> 8 h/night	72(5.5%)	64(4.9%)	8(0.6%)	
	Mean (Standard Deviation)		5.93(1.49)	5.65(1.43)	0.229
	Max-Min		12.0–2.0	11.0–2.0	-
	Percentiles 25–50-75		5.0–6.0.0.0-7.0	4.30–5.30.30.30-7.0	-

P-values were calculated using the Chi-square test for categorical variables and t-test for continuous variables

partly compensate for increased sympathetic activity and endothelial dysfunction, thereby attenuating hypertension risk [32, 33].

Our findings showed that long sleep duration was not associated with the prevalence of hypertension, a result consistent with previous cross-sectional studies [10] as well as systematic reviews and meta-analyses [18, 26, 31]. In contrast, other systematic and meta-analysis studies revealed a significant association between long sleep duration and hypertension [34, 35]. However, our longitudinal analysis confirmed a significant association between long sleep duration and incident hypertension over time. Similarly, a longitudinal study conducted in China reported that individuals sleeping ≥ 10 h per day had a 1.56-fold increased risk of developing hypertension [36]. Objective sleep assessment using actigraphy in a U.S.-based cohort study also revealed that frequent napping was associated with an 11.6% higher incidence of

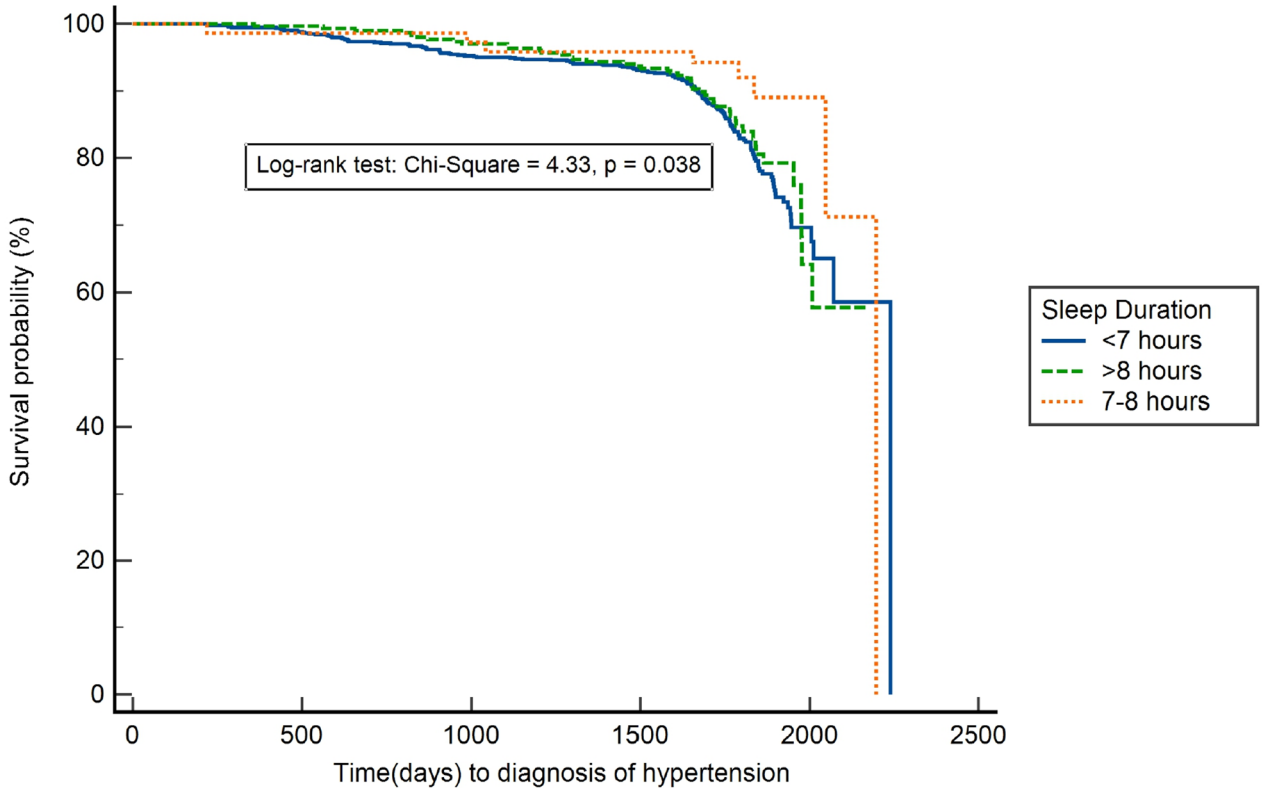

Fig. 2 Overall Kaplan-Meier curve for hypertension incidence by sleep duration

Table 4 The relationship between the incidence of hypertension and categorical sleep duration based on the Cox regression model

Dependent Variable: Time from study entry to diagnosis of hypertension	Hazard Ratio (95%CI)	*P* value	Mean (SE)*	95%CI for mean
Sleep Duration				
< 7 or > 8 h/night	1.91(1.02,3.58)	0.041	2027.74(18.47)	1991.53,2063.96
7–8 h/night	Reference Category		2086.05(42.28)	2003.18,2168.92

Adjusted for age, sex, education, marital status, occupation, Socio-economic status, BMI, smoking, hookah use, salt intake, and physical exercise

Mean (*SE*) mean for survival time (Standard Error)

Given that the frequency of people with hypertension who slept more than 8 hours per night was low (only 8 cases), sleep duration was divided into two groups (<7 or >8 hours; 7–8 hours) instead of three groups

Table 5 The relationship between the incidence of hypertension and sleep duration based on sex and age

Dependent Variable: Time from study entry to diagnosis of hypertension	Male		Female	
	Hazard Ratio (95%CI)	*P* value	Hazard Ratio (95%CI)	*P* value
Age < 50				
Sleep duration	0.83(0.64,1.06)	0.138	0.76(0.57,0.94)	0.008
Age ≥ 50				
Sleep Duration	0.73(0.57,0.92)	0.004	0.68(0.59,0.90)	< 0.001

Adjusted for education, marital status, occupation, Socio-economic status, BMI, smoking, hookah use, salt intake, and physical exercise. Individuals sleeping >8 hours/night were excluded. Only those with short (<7 hours) or normal (7-8 hours) sleep duration were included

Sleep duration was considered as a continuous variable. The Cox regression model was used

hypertension [37]. Additionally, a meta-analysis involving studies with sample sizes greater than 40,000 participants found a significant association between long sleep duration and the risk of hypertension in young and middle-aged adults (OR = 1.133; 95% CI: 1.064–1.206) [27]. Contrary to our finding, the pooled results from the cross-sectional studies of a systematic and meta-analysis study showed that long sleep duration also increased the

risk of hypertension; however, the pooled analysis from the longitudinal studies did not indicate a significant association between long sleep duration and hypertension [38].

The variability in findings across studies regarding long sleep duration and hypertension may be attributed to methodological differences, including how sleep duration is categorized, whether daytime naps are included, sample size variations, differing approaches to covariate adjustment, and variations in participant characteristics.

This study has several notable strengths. It was based on a population-based cohort with a 6-year follow-up period, which enabled us to assess both the prevalence and incidence of hypertension. The comprehensive data collection and availability of detailed demographic and clinical information enabled robust adjustment for potential confounders. Moreover, stratified analyses by sex and age groups improved the interpretability of the findings, providing nuanced insights relevant to public health interventions.

Despite these strengths, the study also has some important limitations. First, sleep duration was assessed through self-reported data, which may introduce recall bias and measurement inaccuracies. Although subjective reporting is a common and practical approach in large-scale epidemiological studies, it does not capture objective aspects of sleep, such as those measured by actigraphy or polysomnography. Second, our analysis focused solely on nighttime sleep duration. We did not account for daytime napping or sleep quality, both of which may influence blood pressure and overall cardiovascular health. Future research should aim to incorporate these additional dimensions of sleep to elucidate their relationship with hypertension better.

Conclusions

This study demonstrates that both short (< 7 h/night) and long (> 8 h/night) sleep durations are associated with an increased risk of hypertension among women under 50, women aged 50 and older, and men over 50 years in the Iranian cohort. These findings suggest that maintaining an adequate sleep duration may play a preventive role in the development of hypertension. The results highlight the potential importance of sleep hygiene as a modifiable factor in hypertension prevention strategies and may inform future clinical and public health interventions.

Abbreviations

PERSIAN	Prospective Epidemiological Research Studies in IrAN
BKNCD	Bandare-Kong Non-Communicable Diseases
BMI	Body mass index; CVD: Cardiovascular disease
DBP	Diastolic blood pressure
SBP	Systolic blood pressure
WHO	World Health Organization
OR	Odds ratio
HR	Hazard ratio
CI	Confidence interval

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Authors' contributions

SR and MK designed and supervised the study. MS collected the data. SR performed the statistical analysis. FR and AA wrote the manuscript. SA was consulted on the possible associated factors to be taken into account. MK and YM revised the manuscript. All authors read and approved the final manuscript.

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Data availability

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

Declarations

Ethics approval and consent to participate

The study obtained informed consent from all subjects. For the vulnerable population, informed consent was obtained from parents or legal guardians. The study protocol underwent thorough evaluation and received approval from the Ethics Committee of Hormozgan University of Medical Sciences (Ethics Code: IR.HUMS.REC.1402.338), aligning with the ethical principles outlined in the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Clinical trial number

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