

Population-based cross-sectional study of sex-specific dose-response associations between night sleep duration and hypertension in Islamic Republic of Iran

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Abstract

Background: Several studies have suggested that sleep disorders have adverse effects on blood pressure. However, the findings remain controversial and only a few studies have investigated the association between sleep duration and hypertension among all age and sex subgroups.

Aim: To evaluate the dose-response association between sleep duration and blood pressure in the Iranian population using the Ravansar non-communicable disease cohort study.

Methods: This was a cross-sectional study of 9865 participants aged 35–65 years from the 2014–2017 Ravansar non-communicable disease cohort study. Night sleep duration was classified as ≤ 5 hours, 6 hours, 7 hours, 8 hours, 9 hours, and ≥ 10 hours. The association between self-reported sleep duration and hypertension was examined using multivariable logistic regression in STATA version 14. Restricted cubic spline analysis showed the dose-response association between sleep duration and hypertension.

Results: The age-adjusted prevalence of hypertension was 16.50% among men, 24.20% among women and 20.50% in the total population. Compared with reference sleep duration (7 hours) in the total population, the multivariable odds ratio [OR (95% CI)] for hypertension was 0.70 (0.55–0.88) for the group with 9 hours sleep duration and 0.90 (0.74–1.09) for the group with ≤ 5 hours sleep duration. Among pre-menopausal women, we observed an inverse association between 9 hours sleep duration and hypertension [0.62 (0.42–0.90)]. The age-adjusted cubic spline suggested a linear inverse association between sleep duration and prevalence of hypertension among men and the total population and a non-linear association among women.

Conclusion: Longer sleep duration (from 9 hours) had a negative association with hypertension. Further studies are needed to identify the risk factors associated with sleep duration and hypertension among the general population in the Islamic Republic of Iran.

Keywords: sleep, sleep disorder, high blood pressure, hypertension, Ravansar cohort study, Iran

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Introduction

In the Islamic Republic of Iran, hypertension is a serious public health concern that significantly increases the risk of cardiovascular and cerebrovascular events (1). Analysis of national data showed that the prevalence of pre-hypertension and hypertension is 25.6% and 39.8%, respectively, among Iranian adults aged 25–70 years (2); the related prevalence among Iranians not taking anti-hypertensive medication is 12.6% (3).

In addition to metabolic risk factors, behaviours such as low physical activity, unhealthy diet and smoking (4,5) have been proven to be the main risk factors for incident hypertension (6). Recent studies suggest that sleep disorders have a negative effect on health (7–9). A meta-analysis reported a dose–response association between

sleep duration and hypertension; it demonstrated that sleeping fewer than 6 hours increases the risk of hypertension by 10% compared to sleeping 7 hours, and by 20–30% when sleeping fewer than 5 hours a day (10). While the Rotterdam study found no association among the elderly (11), the Whitehall II study indicates an association only among women (12).

A study of the Chinese population found that longer (≥ 10 hours) or shorter (< 5 hours) sleep duration than 7 hours of sleep was associated with hypertension (13). A US National Health Interview Survey data reveals a U-shaped association between sleep length and hypertension among all age and sex subgroups (14).

Several studies in the Islamic Republic of Iran have assessed the association between sleep disorders and

blood pressure (15,16); however, only a few (12,17,18) have investigated this association in terms of the sex differences.

Objective

In this study, we examined the sex-specific dose-response association between sleep duration and blood pressure in a large general population using the Ravansar Non-Communicable Disease (RaNCD) cohort study.

Methodology

Study population and design

This cross-sectional study used data from the RaNCD cohort study, which is part of the national cohort study of the Prospective Epidemiologic Research Studies of Iranian Adults that determines trends in the prevalence, incidence, risk factors and outcomes of non-communicable diseases. The cohort study was designed to be conducted over at least 15 years on a triennial basis.

The RaNCD study included 10 065 rural and urban individuals with the following inclusion criteria: 35–65 years of age; minimum residence of 1 year in Ravansar District, with a minimum stay of 9 months per year; likely to stay in Ravansar for the foreseeable future together with a willingness to participate; provision of written informed consent; capable of communicating with the research team; and having Iranian citizenship (according to national ID card and birth certificate) from November 2014 to February 2017 (19).

For our study of 10 065 individuals from the RaNCD study, we excluded those with missing data for body mass index (BMI), waist circumference (WC), creatinine (Cr), fasting plasma glucose (FPG), education, marital status, alcohol use, systolic blood pressure (SBP), diastolic blood pressure (DBP), smoking status, physical activity, total cholesterol (TC), triglycerides (TG), high-density

lipoprotein cholesterol (HDL-C) and night sleep duration (n=200, considering overlaps between missing values). In all, 9865 individuals were eligible for our study (see Figure 1).

We confirm that the original cohort received appropriate ethical approval and informed consent was obtained from all participants. Any further data analysis in our study was in line with the terms of that approval and consent; the study protocol was approved by the Ethics Committee of the Kermanshah University of Medical Sciences. We used the STROBE checklist as a reporting guideline for this study.

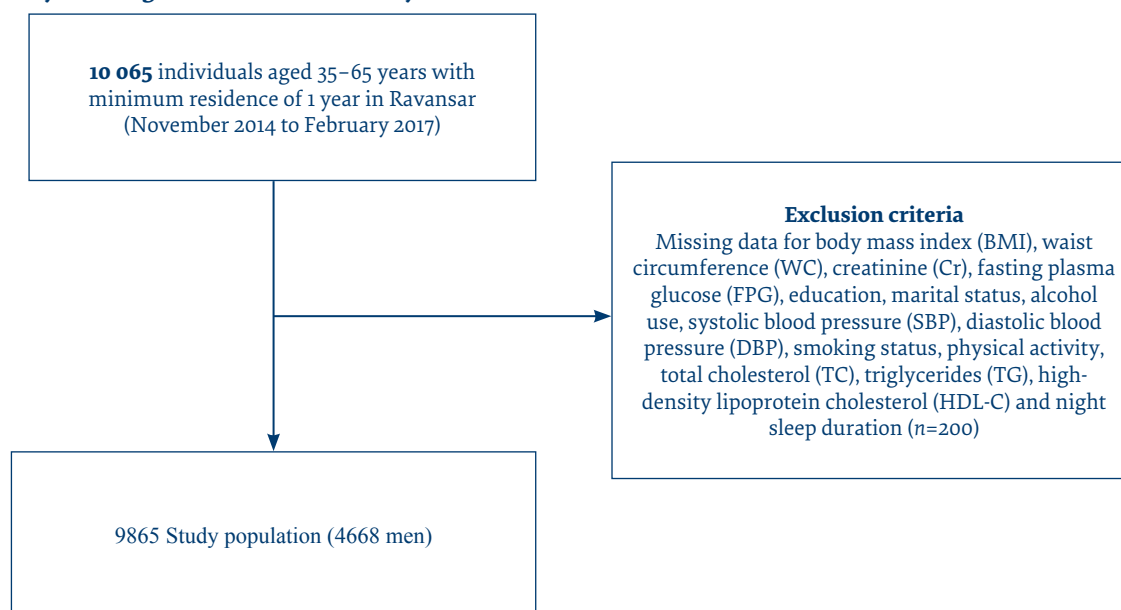
Clinical and laboratory measurements

Information on demographic data, educational level, night sleep duration, alcohol use, smoking status and medication history (during the past 12 months) was obtained by a trained interviewer using a standard questionnaire. To address the potential recall bias on medications, participants were requested to provide relevant medical records to the follow-up team.

Anthropometric measurements (height and weight) were recorded with shoes removed and wearing lightweight clothes. Weight was measured to the nearest 100 grams. Height was measured in a standing position using a tape measure while shoulders were in normal alignment. WC was measured with lightweight clothing, at the level of the umbilicus. Questions on physical activity had 3 components: during work time, during leisure time and while doing sports. Metabolic equivalents were measured based on weekly physical activity time.

Based on the RaNCD study design (19), SBP and DBP were measured 2 times in the right arm after a 10-minute rest in a sitting position. Blood pressure was determined by averaging 2 readings. A blood sample was taken from all participants between 07:00 and 09:00 after 8–12 hours

Figure 1 Study flow diagram: RaNCD cohort study



of overnight fasting, using standard blood-drawing vacationer techniques. FPG was measured by the enzymatic colorimetric method using glucose oxidase; their inter- and intra-assay coefficients of variation at baseline and follow-up phases were <2.3% and 2.3%, respectively.

Total cholesterol was assayed using the enzymatic colorimetric method with cholesterol esterase and cholesterol oxidase; the inter- and intra-assay coefficients of variation at baseline and follow-up phases were both 1.9%. Analyses were conducted using related kits and a Selectra 2 autoanalyser (Vital Scientific). Additional details for laboratory measurements, including FPG, TC, TG, HDL-C and Cr, had been addressed previously (19).

Definition of variables

BMI was calculated as weight (kg) divided by the square of body height (m). The estimated glomerular filtration rate (eGFR; mL/min per 1.73 m²) was estimated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) (20).

Categorical variables were defined as education (<6 years, 6–12 years and >12 years); marital status (single, married and widowed/divorced); smoking status (current, former and never); CKD (eGFR of less than 60 mL/min per 1.73 m² for longer than 3 months); diabetes (FPG≥7 mmol/L or using anti-diabetic medications); hypertension (SBP≥140 mm Hg or DBP≥90 mm Hg or using anti-hypertensive medications or had been previously diagnosed by a doctor); pre-hypertension (130≤SBP<140 mm Hg or 80≤DBP<90 mm Hg without using anti-hypertensive medications (21)); and dyslipidaemia (LDL cholesterol (mg/dL)≥160 and/or total cholesterol (mg/dL)≥240 and/or HDL cholesterol (mg/dL)<40 and/or triglycerides (mg/dL)≥200 and/or having a history of medication for dyslipidaemia.

Menopausal status was defined as the absence of natural menstrual bleeding for more than 12 consecutive months and the exclusion of other pathological or physiological reasons.

Assessment of sleep duration

Total sleep duration was measured by asking the participant when they usually go to bed and wake up. As literature suggests (10, 12), we categorized sleep duration as ≤5 hours, 6 hours (>5 and ≤6 hours), 7 hours (reference, >6 and ≤7 hours), 8 hours (>7 and ≤8 hours), 9 hours (>8 and ≤9 hours) and ≥10 hours. Sleep duration of less than 1 hour is considered zero. We acknowledge that the use of a single question to measure sleep duration may introduce some degree of measurement error. However, this method has been used in previous studies (10,12).

Statistical analysis

Baseline characteristics were described as mean (standard deviation: SD) values for continuous variables, and frequencies (%) for categorical variables. For covariates with a skewed distribution (e.g. TG), the median (interquartile range: IQR) was reported. We compared

the baseline characteristics of men and women among those with and without hypertension using the Student's t-test for normally distributed continuous variables, the chi-squared test for categorical variables, and the Mann-Whitney U statistic for skewed and ordered variables.

Age-adjusted prevalence of hypertension was also presented for different night sleep duration groups. To be able to capture a potential dose-response association between the night sleep duration and hypertension, age-adjusted restricted cubic splines with 4 knots defined at the 5th, 25th, 75th and 95th percentiles were used, a method that enabled us to flexibly model across the groups of night sleep duration while analysing the dose-response association (22).

Potential risk factors suggested by previous studies were included in the univariable and multivariable logistic regression models to evaluate associations between hypertension and night sleep duration.

Confounding variables were adjusted in 5 models: model 1 was a univariable model that included only the night sleep duration variable; model 2 was a multivariable model adjusted for age and sex (only for the total population); model 3 was adjusted for other confounding covariates such as education, marital status, physical activity, current smoking, alcohol use and menopause status for women; model 4 was further adjusted with anthropometric covariates, including BMI and WC; and model 5 was a full model adjusted for the availability of diabetes, dyslipidaemia and CKD.

The multicollinearity of the variables was examined by the variance inflation factor (VIF) (23). We did not find evidence of collinearity (mean VIF=1.44).

The odds ratios (OR) and 95% confidence intervals (95% CI) were reported. However, there was no sex interaction with night sleep duration ($P=0.90$), and all analyses were done separately for men and women. The sex interaction with alcohol intake and BMI was checked, and since no interaction was observed ($P=0.90$ and $P=0.15$, respectively), the adjusted model was reported.

As a sensitivity analysis, we examined the association between night sleep duration and the prevalence of hypertension according to menopausal status. All analyses were conducted using STATA version 14 and a two-tailed $P<0.05$ was considered significant.

Results

The study population consisted of 4668 men and 5197 women at baseline with a mean (SD) age of 47.8 (8.0) and 48.3 (8.4) years, respectively (see Table 1). The result using t-test analysis showed that men and women with hypertension had higher BMI, WC, TC and HDL-c and lower levels of TG and eGFR. The chi-square test for both genders revealed a significant statistical difference between smoking status, physical activity and education levels among those with and without hypertension.

The mean age-adjusted SBP of men and women on hypertension medication was 130 mm Hg (95% CI:128.3–

Table 1 Baseline characteristics of study participants by gender and hypertension status: RaNCD cohort study

Variables	Total population (n=9865)	Men (n=4668)		P value	Women (n=5197)		P value
		Without hypertension (n=3898)	With hypertension (n=770)		Without hypertension (n=3940)	With hypertension (n=1257)	
Age, years, mean (SD)	48.1(8.2)	46.6(7.6)	53.5(7.6)	<0.0001	46.5(7.8)	54.1(7.5)	<0.0001
Body mass index, kg/m ² , mean (SD)	27.5(4.6)	26.08(4.0)	27.7(4.14)	<0.0001	28.2(4.8)	29.8(4.9)	<0.0001
Waist circumference, cm, mean (SD)	97.3(10.6)	95.5(9.3)	99.8(10.5)	<0.0001	97.3(11.2)	101.5(10.8)	<0.0001
Systolic blood pressure, mm Hg, mean (SD)	109.9(17.5)	103.2(12.6)	133.9(20.6)	<0.0001	106.8(12.1)	125.6(20.2)	<0.0001
Diastolic blood pressure, mm Hg, mean (SD)	70.7(10.2)	69.3(8.2)	82.8(11.1)	<0.0001	67.3(8.0)	78.6(11.8)	<0.0001
Marital status, n(%)				0.06			<0.0001
Single	413(4.19)	87(2.23)	7(0.91)		304(7.72)	15(1.19)	
Married	8904(90.26)	3779(96.95)	757(98.31)		3298(83.71)	1070(85.12)	
Divorced /widowed	548(5.55)	32(0.82)	6(0.78)		338(8.58)	172(13.68)	
Education, years, n(%)				<0.0001			<0.0001
<6	6209(62.94)	1528(39.20)	417(54.16)		3100(78.68)	1164(92.6)	
6-12	2885(29.24)	1831(46.97)	265(34.42)		706(17.92)	83(6.60)	
≥12	771(7.82)	539(13.83)	88(11.43)		134(3.40)	10(0.80)	
Smoking status, n(%)				<0.0001			<0.0001
Never	7904(80.12)	2498(64.08)	479(62.21)		3790(96.19)	1137(90.45)	
Current	1149(11.65)	918(23.55)	129(16.75)		63(1.60)	39(3.10)	
Former	812(8.23)	482(12.37)	162(21.04)		87(2.21)	81(6.44)	
Alcohol use, yes	622(6.31)	536(13.75)	82(10.65)	0.02	4(0.1)	-	0.260
Physical activity, n(%)				<0.0001			<0.0001
Light	2696(27.33)	1234(31.66)	307(39.87)		817(20.74)	338(26.89)	
Moderate	5084(51.54)	1323(33.94)	240(31.17)		2702(68.58)	819(65.16)	
Vigorous	2085(21.14)	1341(34.4)	223(28.96)		421(10.69)	100(7.96)	
Post-menopause women, n(%)	1370(26.36)	-	-		771(19.57)	599(47.65)	
Fasting plasma glucose, mmol/L, mean (SD)	5.4(1.7)	5.3(1.6)	5.8(2.1)	<0.0001	5.2(1.4)	5.9(2.2)	<0.0001
Total cholesterol, mmol/L, mean (SD)	4.8(1.0)	4.7(0.93)	4.8(1.0)	0.02	4.8(1.0)	5.1(1.03)	<0.0001
Triglycerides, mmol/L, median (IQR)	1.3(0.95–1.3)	1.37(0.97–1.98)	1.5(1.10–2.10)	<0.0001	1.21(0.88–1.68)	1.46(1.06–2.01)	<0.0001
High-density lipoprotein cholesterol, mmol/L, mean (SD)	1.2(0.3)	1.11(0.26)	1.08(0.25)	0.003	1.29(0.29)	1.26(0.30)	0.001
eGFR, ml/min/1.73 m ² , mean (SD)	70.0(15.2)	63.3(11.1)	58.4(12.0)	<0.0001	78.4(14.7)	71.7(15.1)	<0.0001
Low-density lipoprotein cholesterol, mmol/L, mean (SD)	2.6(0.66)	2.61(0.63)	2.7(0.68)	0.08	2.63(0.67)	2.8(0.69)	<0.0001
Anti-hypertensive drug, yes, n(%)	1136(11.52)	-	364(47.27)	-	-	772(61.42)	
Diabetes drug, yes, n(%)	451(4.57)	129(3.31)	61(7.92)	<0.0001	118(3.0)	143(11.38)	<0.0001
Lipid-lowering drug, yes, n(%)	398(4.03)	82(2.10)	67(8.7)	<0.0001	98(2.50)	151(12.01)	<0.0001
Diabetes, yes, n(%)	801(8.12)	249(6.39)	116(15.06)	<0.0001	217(5.51)	219(17.42)	<0.0001
Dyslipidaemia, yes, n(%)	4375(44.35)	2005(51.44)	473(61.43)	<0.0001	1296(32.89)	601(47.81)	<0.0001
Chronic kidney disease, yes, n(%)	2594(26.29)	1570(40.28)	425(55.19)	<0.0001	333(8.45)	266(21.16)	<0.0001
Night sleep duration, hour, mean (SD)	7.05(1.2)	7.0(1.2)	6.9(1.2)	0.28	7.2(1.2)	7.0(1.3)	<0.0001

eGFR: estimated glomerular filtration rate.

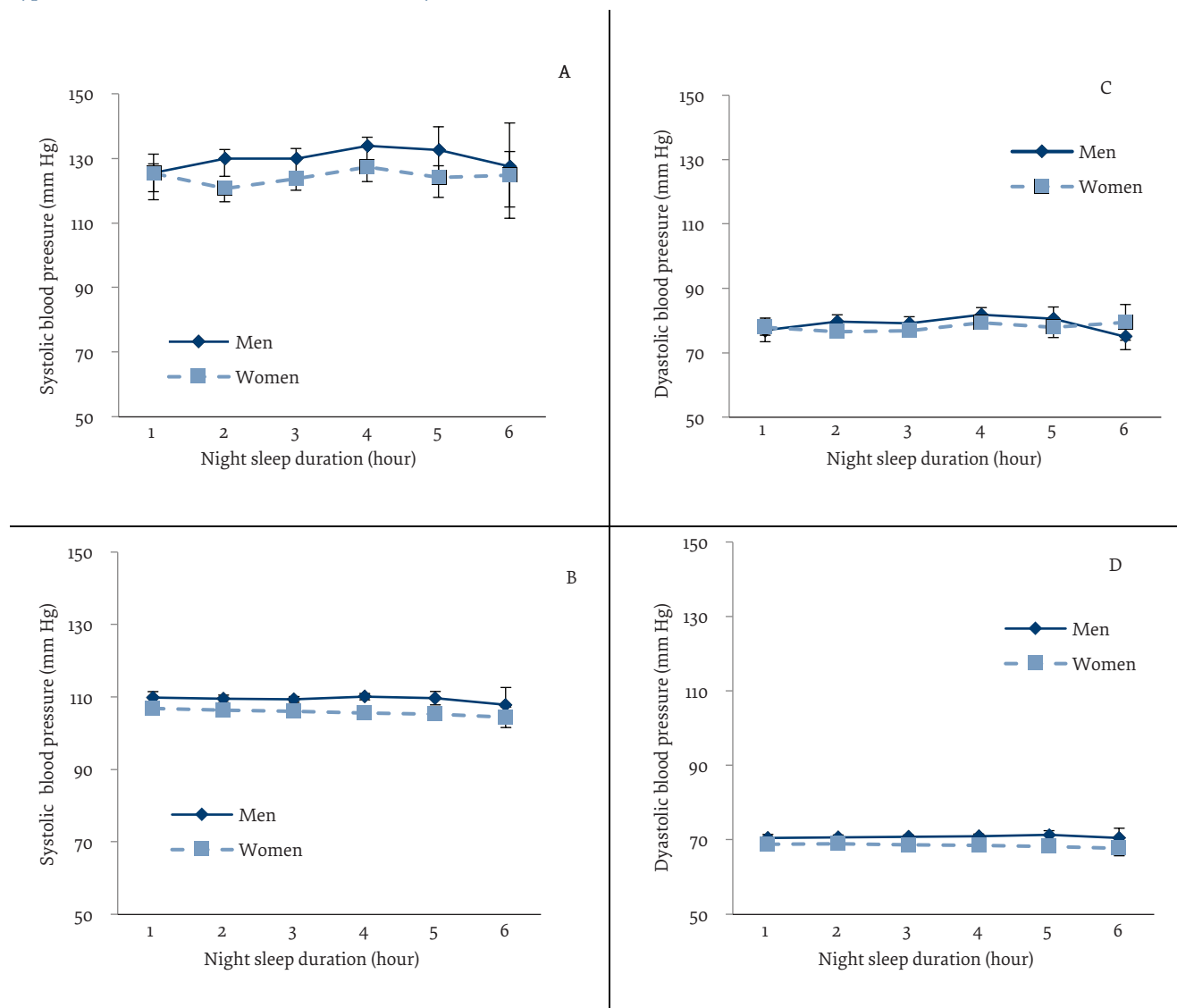
Values are shown as mean (SD) and n(%), for continuous and categorical variables, respectively; for TG, values are shown as median (interquartile range: IQR).

Comparison was done using the Student's t-test for normally distributed continuous variables, the chi-squared test for categorical variables, and the Mann-Whitney U statistic for skewed and ordered variables.

132.8) and 124.2 mm Hg (95% CI:122.8–125.6), respectively; the mean SBP was 109.6 (95% CI:109.2–110.1) among men and 105.9 (95% CI:105.5–106.4) among women not taking hypertension medication.

The mean age-adjusted DBP of men and women on hypertension medication was 79.8 mm Hg (95% CI:78.6–80.9) and 77.6 mm Hg (95% CI:76.8–78.5), respectively; the mean DBP was 70.8 (95% CI:70.6–71.1) among men

Figure 2 Mean values of systolic and diastolic blood pressure according to varied night sleep duration by gender and status of hypertension medication: RaNCD cohort study



A and C: On hypertension medications
B and D: Not on hypertension medications

Table 2 Age-adjusted prevalence^a of hypertension by gender: RaNCD cohort study^{*}

	Total population	Men	Women
Night sleep duration, hours			
≤5	20.5(18.3-22.8)	16.8(13.8-19.9)	23.8(20.5-27.1)
6	21.4(19.8-23.0)	16.4(14.3-18.6)	25.9(23.5-28.4)
7	21.5(20.2-22.8)	17.2(15.4-19.0)	25.4(23.4-27.3)
8	20.0(18.6-21.4)	16.1(14.1-18.0)	23.6(21.5-25.6)
9	16.2(13.7-18.6)	14.2(10.3-18.1)	18.4(15.2-21.7)
≥10	19.5(13.4-25.6)	14.9(5.5-24.3)	23.5(15.3-31.6)
P for trend	0.016	0.394	0.017

^aage and gender adjusted prevalence for total population and age adjusted prevalence for gender-specific prevalence
^{*}Data presented as: prevalence %(95% CI)

and 68.7 (95% CI:68.4–68.9) among women not taking hypertension medication (see Figure 2).

The age-adjusted hypertension prevalence was 16.5% among men, 24.2% among women (17% pre-menopause and 44.3% post-menopause) and 20.5% among the total population. As shown in Table 2, the age-adjusted prevalence of hypertension was 16.8% among men with ≤5-hour sleep duration and 14.9% in the group with ≥10-hour sleep duration (*P* for trend=0.39). However, among women, the age-adjusted prevalence of hypertension shows a significant decrease by increasing sleep hours: 23.8% in the group with ≤5-hour sleep duration and 23.5% in the group with ≥10-hour sleep duration (*P* for trend=0.02).

Table 3 shows the univariable and multivariable logistic regression of different sleep duration hours with the prevalence of hypertension. Among the total population, 9 hours of sleep (7 hours as reference) showed an inverse association with hypertension [OR (95% CI): 0.70 (0.55–0.88%)]. This association was also observed among women [OR (95% CI): 0.65 (0.48–0.86%)] but not men for 9 hours of sleep.

Figure 3 shows multivariable OR (95% CI) of hypertension per additional night sleep hour for men, women and the total population. When night sleep duration was adjusted with age in model 2, each additional night sleep hour was associated with 0.93 (0.87–0.99%) for women and 0.98 (0.972–1.05%) for men; this was significant for women. However, after further adjustment in multivariable model 5, the OR (95% CI) for each additional sleep hour reached 0.96 (0.90–1.01%) for women and 1.0 (0.94–1.07%) for men.

As a sensitivity analysis, we examined the association between night sleep duration and the prevalence of hypertension according to menopausal status. Among premenopausal women, in comparison with the 7-hour night sleep duration in model 5, women with a 9-hour night sleep duration had the age-adjusted OR (95% CI) of 0.62 (0.42–0.90%). Among postmenopausal women, no significant association was observed between night sleep duration and the prevalence of hypertension (see Table 4).

Table 3: Night sleep duration and hypertension status by gender: RaNCD cohort study

	Night sleep duration (hours)						P for trend
	≤5	6	7	8	9	≥10	
Men							
n /N [†]	89/471	172/1051	259/1572	202/1241	40/288	8/45	
Model 1	1.18(0.9–1.54)	1.0(0.8–1.22)	Reference	0.98(0.80–1.20)	0.82(0.57–1.17)	1.10(0.50–2.38)	0.448
Model 2	0.97(0.73–1.29)	0.94(0.75–1.18)	Reference	0.91(0.74–1.13)	0.78(0.53–1.14)	0.83(0.36–1.91)	0.193
Model 3	0.96(0.73–1.28)	0.93(0.75–1.17)	Reference	0.93(0.75–1.15)	0.80(0.55–1.18)	0.84(0.36–1.96)	0.263
Model 4	0.93(0.70–1.25)	0.90(0.72–1.13)	Reference	0.93(0.75–1.16)	0.85(0.57–1.26)	0.97(0.40–2.32)	0.385
Model 5	0.94(0.70–1.26)	0.89(0.71–1.12)	Reference	0.93(0.75–1.16)	0.83(0.56–1.23)	0.89(0.36–2.16)	0.315
Women							
n /N [†]	134/459	277/1012	417/1646	322/1452	86/534	21/94	
Model 1	1.21(0.96–1.53)	1.11(0.93–1.33)	Reference	0.84(0.71–0.99)	0.56(0.44–0.73)	0.85(0.51–1.39)	<0.0001
Model 2	0.91(0.71–1.16)	1.03(0.85–1.25)	Reference	0.89(0.74–1.07)	0.62(0.47–0.82)	0.88(0.51–1.53)	0.013
Model 3	0.91(0.71–1.17)	1.04(0.86–1.26)	Reference	0.91(0.76–1.08)	0.63(0.48–0.84)	0.91(0.52–1.58)	0.025
Model 4	0.86(0.67–1.12)	1.03(0.85–1.26)	Reference	0.93(0.77–1.11)	0.65(0.49–0.87)	1.11(0.63–1.95)	0.095
Model 5	0.89(0.68–1.15)	1.03(0.84–1.25)	Reference	0.94(0.78–1.13)	0.65(0.48–0.86)	1.05(0.59–1.86)	0.087
Total population							
n /N [†]	223/930	449/2063	676/3218	524/2693	126/822	29/139	
Model 1	1.19(1.0–1.41)	1.05(0.91–1.20)	Reference	0.91(0.80–1.03)	0.68(0.55–0.84)	0.99(0.65–1.50)	0.002
Model 2	0.93(0.77–1.15)	0.99(0.86–1.15)	Reference	0.90(0.78–1.03)	0.66(0.53–0.83)	0.86(0.55–1.36)	0.005
Model 3	0.93(0.77–1.12)	0.99(0.86–1.15)	Reference	0.91(0.79–1.04)	0.68(0.54–0.85)	0.88(0.56–1.40)	0.011
Model 4	0.89(0.73–1.07)	0.98(0.84–1.13)	Reference	0.92(0.80–1.06)	0.71(0.56–0.89)	1.07(0.67–1.08)	0.057
Model 5	0.90(0.74–1.09)	0.97(0.83–1.12)	Reference	0.92(0.80–1.07)	0.70(0.55–0.88)	0.99(0.61–1.60)	0.040

All data is presented as OR (95% CI).

[†] n: number of hypertension patients; N: number of population

Model 1: unadjusted

Model 2: adjusted for age and sex (for total population)

Model 3: model 2+education, marital status, physical activity, current smoking and alcohol use

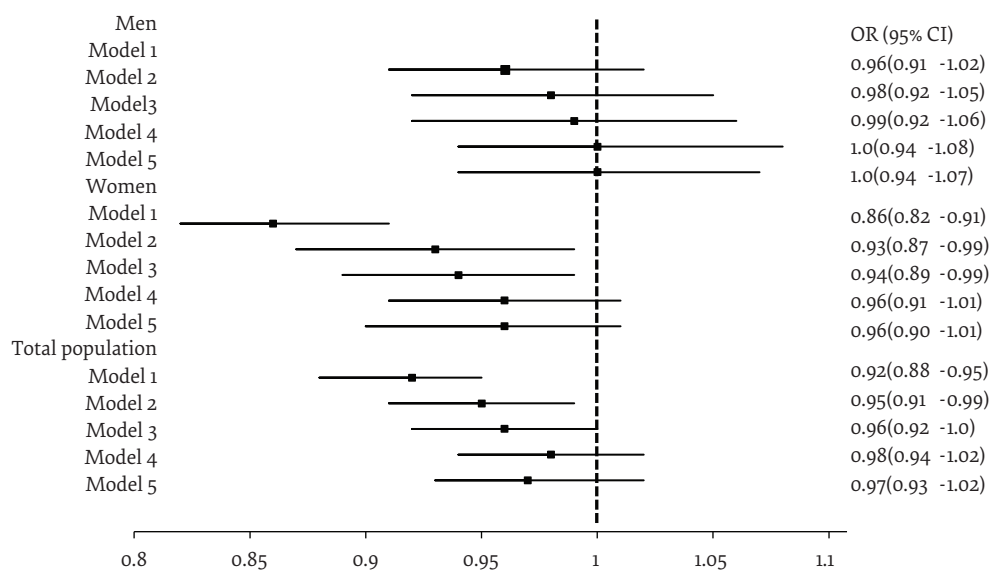
Model 4: model 3+BMI, WC

Model 5: model 4+ diabetes, dyslipidaemia, CKD

P value for gender interaction with night sleep duration is 0.90

BMI: Body mass index; WC: waist circumference; CKD: chronic kidney disease; OR: odds ratio; CI: confidence interval.

Figure 3 Odds ratios and 95% confidence intervals of the sleep duration (per hour as a continuous variable) on the prevalence of hypertension by gender: RaNCD cohort study



Model 1: unadjusted
 Model 2: adjusted for age and sex (for total population)
 Model 3: model 2+education, marital status, physical activity, current smoking, alcohol use and menopause status for women
 Model 4: model 3+BMI, WC
 Model 5: model 4+ diabetes, dyslipidaemia, CKD
 The P value for gender interaction with night sleep duration is 0.9.
 Pseudo-R-squared values for model 5 among the total population, men, and women are 0.30, 0.26 and 0.32, respectively.
 BMI: Body mass index; WC: waist circumference; CKD: chronic kidney disease.

Table 4 Night sleep duration and hypertension status among women stratified by menopausal state: RaNCD cohort study

	Night sleep duration (hour)						P for trend
	≤5	6	7	8	9	≥10	
Post-menopause							
n /N [†]	71/167	127/293	201/443	144/328	43/115	13/24	
Model 1	0.89(0.62–1.27)	0.92(0.68–1.24)	Reference	0.94(0.71–1.26)	0.72(0.47–1.10)	1.42(0.62–3.24)	0.469
Model 2	0.85(0.59–1.23)	0.91(0.67–1.24)	Reference	0.93(0.69–1.25)	0.65(0.42–1.00)	1.20(0.52–2.78)	0.286
Model 3	0.83(0.57–1.20)	0.90(0.66–1.22)	Reference	0.93(0.69–1.26)	0.66(0.43–1.01)	1.17(0.50–2.71)	0.302
Model 4	0.78(0.54–1.15)	0.92(0.67–1.25)	Reference	0.97(0.72–1.32)	0.66(0.73–1.03)	1.42(0.60–3.37)	0.518
Model 5	0.82(0.54–1.17)	0.92(0.67–1.26)	Reference	0.98(0.72–1.33)	0.68(0.43–1.06)	1.30(0.54–3.16)	0.533
Pre-menopause							
n /N [†]	216/1203	63/292	150/719	178/1124	43/419	8/70	
Model 1	1.26(0.92–1.72)	1.20(0.95–1.52)	Reference	0.86(0.69–1.07)	0.52(0.37–0.74)	0.59(0.28–1.25)	0.001
Model 2	0.95(0.68–1.32)	1.12(0.87–1.43)	Reference	0.87(0.69–1.09)	0.61(0.42–0.88)	0.71(0.33–1.56)	0.025
Model 3	0.96(0.68–1.34)	1.13(0.88–1.45)	Reference	0.88(0.70–1.11)	0.63(0.43–0.90)	0.74(0.34–1.63)	0.043
Model 4	0.91(0.64–1.28)	1.10(0.86–1.42)	Reference	0.89(0.71–1.13)	0.65(0.45–0.95)	0.89(0.40–1.97)	0.100
Model 5	0.93(0.66–1.31)	1.08(0.83–1.39)	Reference	0.90(0.71–1.14)	0.62(0.42–0.90)	0.86(0.39–1.90)	0.077

All data is presented as OR (95% CI).
[†] n: number of hypertension patients; N: number of population
 Model 1: unadjusted
 Model 2: adjusted for age
 Model 3: model 2+education, marital status, physical activity, current smoking and alcohol use
 Model 4: model 3+BMI, WC
 Model 5: model 4+ diabetes, dyslipidaemia, CKD
 BMI: Body mass index; WC: waist circumference; CKD: chronic kidney disease; OR: odds ratio; CI: confidence interval

Discussion

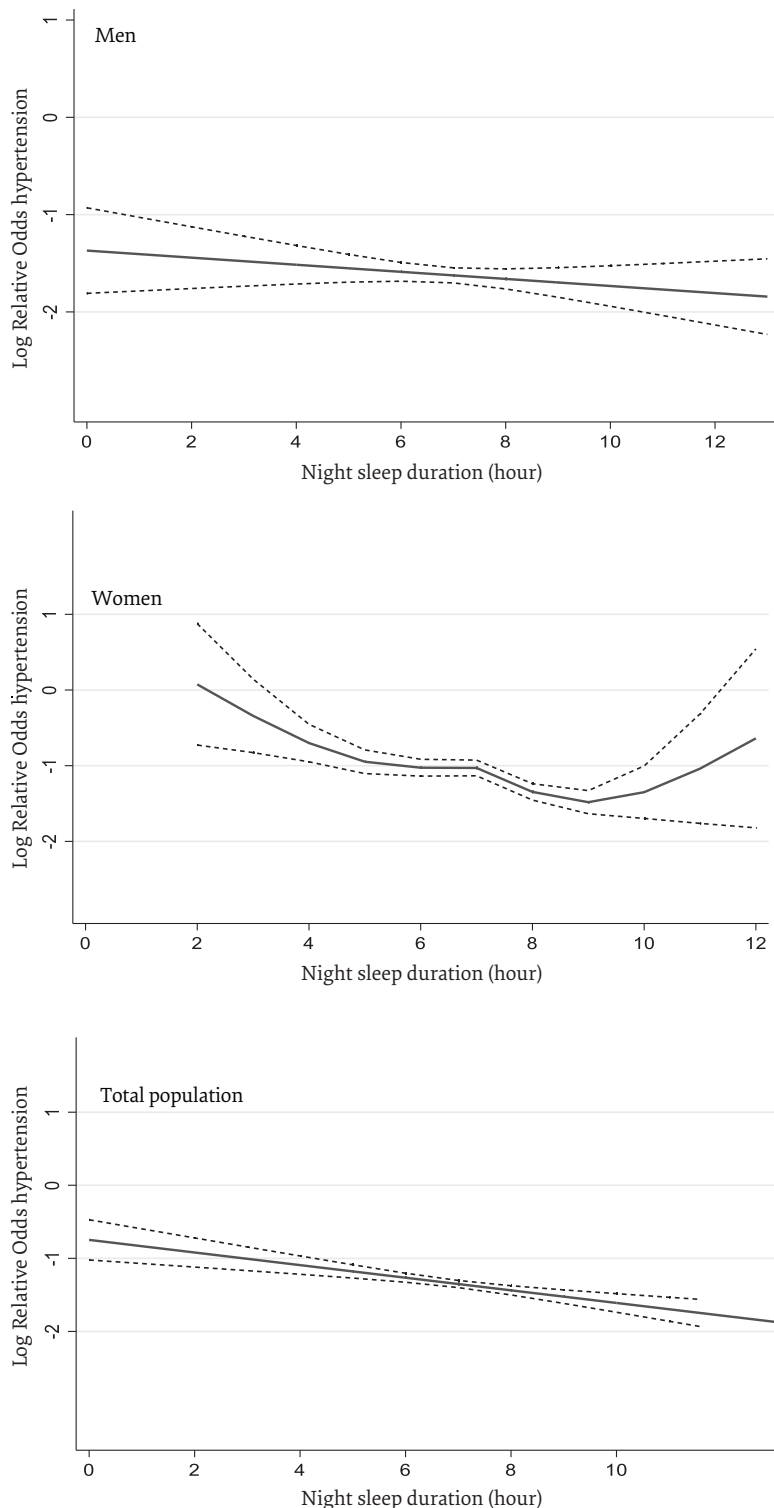
This study indicated that a long sleep duration of 9 hours is associated with a lower prevalence of hypertension in the Iranian community-based population. There

was a negative association between the prevalence of hypertension and progressively increasing sleep duration among the total population and women, while no significant trend was observed among men.

The association of sleep duration with hypertension persisted after adjustment for potential confounders, including age, education, marital status, physical activity, alcohol use, current smoking, BMI and waist circumference among the total population; the same results were observed only among women.

Analyses showed that the association between sleep duration and hypertension was not confounded by diabetes, dyslipidaemia or CKD. This study demonstrated a dose–response association between sleep duration and hypertension; however, this association was not linear among women.

Figure 4 Age-adjusted dose–response relationship between night sleep duration and log relative odds of hypertension hours RaNCD hours study



X-axis: Night sleep duration (hours)
Y-axis: Log relative odds of hypertension

This study indicates a sex-related difference in the association between sleep duration and hypertension (a U-shaped pattern for women vs a linear pattern for men). Obstructive sleep apnoea is more prevalent among men (24) while women experience more insomnia (25); both are independent risk factors for hypertension (26,27). Accordingly, they can influence the association between sleep duration and hypertension and must be considered in adjusted models.

In their review, Burgard et al. explained that women sleep more than men; we also indicated this in our study (28). This factor can influence the association between hypertension and sleep duration in terms of gender (a U-shaped pattern in women vs a linear decreasing pattern of hypertension odds with sleep duration among men) (see Figure 4).

It has been shown that the association of sleep disorders with hypertension is more prevalent among women (12,29). It is hypothesized that shorter sleep duration makes women more vulnerable to poor cardiovascular outcomes. Gender influence on the association of sleep duration needs more clarification of underlying mechanisms and further population-based studies based on more objective measures of sleep duration (30).

According to a National Sleep Foundation report, the optimal sleep duration for staying healthy is 7–9 hours a day among adults (18–64 years) (31). The principal findings of our study show that sleep duration of ≤ 5 hours a day is associated with a greater prevalence of hypertension and the negative association was observed with a 9-hour sleep duration. National Health Interview Survey (NHIS) data show that short (< 6 hours for men and < 8 hours for women) and long (≥ 10 hours for men) sleep durations are associated with a higher likelihood of hypertension (14).

Najafian et al. studied hypertension and self-reported sleep duration in central Iran. Even after adjusting for age, sex, BMI and abdominal obesity, sleeping less than 5 hours a day was associated with hypertension in people over 19 years old. However, the negative association between sleep duration of more than 9 hours and hypertension was statistically insignificant (32). These findings agree with ours; however, after further adjustment with potential confounders among those with sleep duration of ≤ 5 hours, we did not observe a positive association with hypertension.

A study in China showed a positive association among those with sleep duration of more than 10 hours a day and a negative association among those with sleep duration of 6 hours a day (13). They also found a U-shaped (non-linear) trend between sleep duration and hypertension among men; in our study, only women showed a non-linear trend. A linear negative dose–response relation was detected among the total population. One systematic review of 36 studies (26 cross-sectional and 10 cohorts) reported that short sleep duration was related to higher blood pressure and hypertension (33).

Consistent with our findings, Grandner et al. indicated higher odds for long sleepers (≥ 10 hours a day) in the non-adjusted model of their study covariates (18). However, in their study, both 9- and ≥ 10 -hour groups were found to have higher odds for hypertension in the adjusted model. This difference in findings may be due to different measures of sleep duration between these 2 studies (sleep duration in the day vs sleep duration at night in our study) and different covariates in adjusted models.

In this study, we evaluated the association among adults aged 35–65 years, while the mechanism of the sleep duration on hypertension may differ among children, adolescents, young adults or the elderly (33). This will be a serious concern if we know that each 10 mm Hg increase in adolescent SBP/DBP increases the risk of early adulthood hypertension by 4% across 10 years of follow-up (11,34).

Limitations

This study's strength is that it provides an epidemiological profile by assessing the dose–response association in a large sample of men and women in Islamic Republic of Iran, a country in the Middle East and North Africa (MENA) region. However, several limitations must be considered.

First, because it was a cross-sectional study, we were unable to determine causality. Second, it used self-reported sleep duration, smoking habits and alcohol use, and therefore recall bias is possible. Actigraphy and polysomnography could provide objective and detailed data on sleep characteristics.

Quality of sleep, daytime dysfunction, reported difference between weekday and weekend sleep duration and interference of sleep duration with daily activities could also influence the association between high blood pressure and sleep duration. Covariates like insomnia, obstructive sleep apnoea, employment status and shiftwork could affect hypertension and sleep duration, and require consideration in future studies.

The models showed low pseudo-R-squared values, which may suggest that a considerable portion of the variation remains unaccounted for. However, it is crucial to note that pseudo-R-squared was different from R-squared in linear regression models. This is because pseudo-R-squared may be low even when the models fit the data exceptionally, as discussed by Rose and Singh Mann (35).

Conclusion

This study found that longer sleep duration negatively correlates with hypertension. Further longitudinal studies are suggested to find a causal association between hypertension and sleep duration.

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Étude transversale en population des associations dose-réponse selon le sexe entre la durée du sommeil nocturne et l'hypertension en République islamique d'Iran

Résumé

Contexte : Plusieurs études ont suggéré que les troubles du sommeil ont des effets indésirables sur l'hypertension artérielle. Cependant, ces résultats restent controversés et seules quelques études se sont penchées sur l'association entre la durée du sommeil et l'hypertension dans tous les sous-groupes d'âge et de sexe.

Objectif : Évaluer la relation dose-réponse entre la durée du sommeil et la tension artérielle dans la population iranienne en utilisant l'étude de cohorte sur les maladies non transmissibles réalisée à Ravansar.

Méthodes : La présente étude transversale a été menée auprès de 9865 personnes âgées de 35 à 65 ans ayant participé à l'étude de cohorte sur les maladies non transmissibles réalisée entre 2014 et 2017 à Ravansar. La durée du sommeil nocturne a été classifiée comme suit : inférieure ou égale à cinq heures, six heures, sept heures, huit heures, neuf heures et supérieure ou égale à dix heures. La relation entre la durée du sommeil auto-déclarée et l'hypertension a été examinée à l'aide de la régression logistique multivariée réalisée avec le logiciel STATA version 14. L'analyse par spline cubique restreinte a mis en évidence l'association dose-réponse entre la durée du sommeil et l'hypertension.

Résultats : La prévalence de l'hypertension ajustée sur l'âge était de 16,50 % chez l'homme de 24,20 % chez la femme et de 20,50 % dans la population totale. Comparativement à la durée du sommeil de référence de sept heures dans la population totale, l'odds ratio multivarié [OR (IC à 95 %)] pour l'hypertension était de 0,70 (0,55-0,88) pour le groupe dont la durée du sommeil était de neuf heures et de 0,90 (0,74-1,09) pour le groupe dont la durée du sommeil était inférieure ou égale à cinq heures. Chez les femmes pré-ménopausées, nous avons observé une association inverse entre la durée du sommeil de neuf heures et l'hypertension [0,62 (0,42-0,90)]. L'analyse par spline cubique ajustée sur l'âge a révélé une association linéaire inverse entre la durée du sommeil et la prévalence de l'hypertension chez l'homme et dans la population totale, et une association non-linéaire chez la femme.

Conclusion : Une durée de sommeil plus longue (supérieure à neuf heures) était négativement associée à l'hypertension. Des études supplémentaires sont nécessaires pour identifier les facteurs de risque associés à la durée du sommeil et à l'hypertension dans la population générale de la République islamique d'Iran.

دراسة سكانية مقطعية لارتباطات الاستجابة لجرعات محددة حسب نوع الجنس بين مدة النوم ليلاً وارتفاع ضغط الدم في جمهورية إيران الإسلامية

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الخلاصة

الخلفية: أشارت عدة دراسات إلى أن اضطرابات النوم لها آثار سلبية على ارتفاع ضغط الدم. ومع ذلك، لا تزال النتائج مثيرة للجدل، ولم تستقص سوى دراسات قليلة العلاقة بين مدة النوم وارتفاع ضغط الدم بين جميع الفئات الفرعية العمرية والجنسية.

الأهداف: هدفت هذه الدراسة إلى تقييم علاقة الاستجابة للجرعات بين مدة النوم وضغط الدم لدى السكان الإيرانيين باستخدام الدراسة الأثرية عن الأمراض غير السارية في مدينة رفسنر.

طرق البحث: أجريت هذه الدراسة المقطعية على 9865 مشاركاً تتراوح أعمارهم بين 35 و65 عاماً في دراسة أثرية عن الأمراض غير السارية في مدينة رفسنر في الفترة بين عامي 2014 و2017. وصُنِّفت مدة النوم الليلي إلى مدة نوم ≥ 5 ساعات، و6 ساعات، و7 ساعات، و8 ساعات، و9 ساعات، و ≤ 10 ساعات. وفحصت العلاقة بين مدة النوم المبلغ عنها ذاتياً وارتفاع ضغط الدم باستخدام الانحدار اللوجستي المتعدد المتغيرات في الإصدار 14 من برنامج STATA. وأظهر تحليل الشريحة التكميلية المقيدة ارتباط الاستجابة للجرعات بين مدة النوم وارتفاع ضغط الدم.

النتائج: بلغ معدل انتشار ارتفاع ضغط الدم المعدل حسب العمر 16,50% لدى الرجال، و24,20% لدى النساء، و20,50% لدى عامة السكان. وبالمقارنة مع مدة النوم المرجعية (7 ساعات) لدى عامة السكان، كانت نسبة الأرجحية المتعددة المتغيرات [نسبة الأرجحية (فاصل الثقة 95%)] لارتفاع ضغط الدم [0,70 (0,55-0,88)] للمجموعة التي تنام 9 ساعات و[0,90 (0,74-1,09)] للمجموعة تنام 5 ساعات أو أقل. وفي النساء في مرحلة ما قبل انقطاع الطمث، لاحظنا ارتباطاً عكسياً بين مدة النوم البالغة 9 ساعات وارتفاع ضغط الدم [0,62 (0,42-0,90)].

وأشارت الشريحة التكميلية المعدلة حسب العمر إلى وجود ارتباط عكسي خطي بين مدة النوم ومعدل انتشار ارتفاع ضغط الدم بين الرجال وعامة السكان، وارتباط غير خطي بين النساء.

الاستنتاجات: كانت مدة النوم الأطول (بداية من 9 ساعات) مرتبطة ارتباطاً سلبياً بارتفاع ضغط الدم. ويلزم إجراء مزيد من الدراسات لتحديد عوامل الخطر المرتبطة بمدة النوم وارتفاع ضغط الدم بين عامة السكان في جمهورية إيران الإسلامية.

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