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A prospective study of the association between total sleep duration and incident hypertension

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The aim of this prospective study was to evaluate total sleep duration as a potential risk factor for the development of hypertension after a mean of 2.6 years of follow-up. The study participants comprised 1715 Korean adults aged 40 to 70 years. The participants were without hypertension at baseline (2005–2008) and during follow-up (2008–2011) to determine the incident cases of hypertension. Based on a self-reported questionnaire, the individuals were stratified according to total sleep duration (<6 hours, 6–7.9 hours, 8–9.9 hours, ≥10 hours). Hypertension was defined according to the Eighth Joint National Committee (JNC 8) guidelines. After an average of 2.6 years of follow-up, 164 (9.56%) participants developed hypertension. In multivariate adjusted models, the odds ratio for new-onset hypertension was 1.71 (95% confidence interval, 1.01–2.89) in participants with a short sleep duration (<6 hours) compared with those who reported 6 to 7.9 hours of sleep. Long sleep duration (more than 8 hours) did not have any significant difference on incident hypertension. Among middle-aged and elderly Korean adults, short sleepers were independently associated with a higher risk of developing hypertension.

1 | INTRODUCTION

Hypertension is a major public health concern worldwide because of its high prevalence and status as an important risk factor for renal and cardiovascular diseases.[1,2] High blood pressure (BP) aggravates the risk of diabetes and related complications of microvascular and macrovascular diseases.[3,4] Despite the implementation of various intervention and preventative measures, the prevalence of hypertension has increased to 35% in East Asia.[5] The Korean National and Nutritional Examination Survey revealed that the prevalence of hypertension was 28.5% in Korea.[6] Hypertensive patients typically must modify their lifestyle and dietary habits to reduce the effects of the condition. Given the high prevalence of hypertension and its potential consequences, there is substantial interest in understanding its lifestyle risk factors in population-based longitudinal studies.

Sleep plays a vital role in maintaining the overall growth of the human body and mind. Epidemiological studies have suggested that chronic sleep deprivation results in various negative health outcomes, including obesity, type 2 diabetes, metabolic syndrome, and coronary heart disease.[7–11] Sleep restriction impedes many essential homeostatic mechanisms and is accompanied by detrimental effects on the hormonal and metabolic systems.[12] A sleep deficiency study observed a significant elevation in BP in healthy participants and in patients with preexisting hypertension after sleep was curtailed to 3.6 to 4.5 hours per night.[13,14]

The association between short sleep duration and hypertension has recently been reported by many researchers.[15–19] However, most of these studies have been conducted in Western populations. Hence, their results are not generalizable to other ethnicities, including Koreans. Moreover, the association between sleep duration and hypertension was based on a cross-sectional design; very few prospective studies have been performed on the cause and effect relationship between these factors.[18–20] Clinicians and policymakers should focus on these risk factors when developing preventive approaches to

reduce the burden associated with hypertension. In contrast, other studies have shown that short sleep duration was not associated with hypertension in a longitudinal setting, which produced inconsistent findings.[21,22] Therefore, we need to study this relationship using a population-based approach to identify sleep duration as a major predictor of hypertension and its associated morbidity.

In this study, we investigated the prospective association between short sleep duration and the risk of incident hypertension in a Korean cohort study. We hypothesized that short sleep duration (<6 hours) would be a predictor of progression to hypertension.

2 | METHODS

2.1 | Study participants

Our study data were collected from a prospective cohort within the Korean Genome and Epidemiology Study on Atherosclerosis Risk in Rural Areas in the Korean General Population (KoGES-ARIRANG), a continuing community-based cohort. The basic aim of this longitudinal cohort was to estimate the risk factors for chronic metabolic diseases, such as diabetes, dyslipidemia, obesity, metabolic syndrome, and cardiovascular disease.[23,24] Participants in this survey comprised 5178 men and women aged 40 to 70 years. The study individuals lived in rural areas of Wonju and Pyeongchang in South Korea. This region is less likely to experience demographic shifts; therefore, the study participants should be available for long-term follow-up.

Figure 1 shows the flow chart description of the study population. The baseline study included 5178 adults from November 2005 to January 2008. The first follow-up survey invited participants to take part in the study from April 2008 to January 2011, and 3862 (74.6%) participants responded. We excluded 2108 participants who had hypertension at baseline, 26 participants with a history or current diagnosis of cardiovascular disease, and 13 participants with missing data. Finally, 1715 participants without hypertension at baseline were recruited for the present analysis. Before the study began, written informed consent was obtained from the study participants to declare their willingness to take part in the survey. The protocol for the study was approved by the institutional review board of Wonju Christian Hospital (approval number: CR105024-026).

2.2 | Data collection and measurements

At baseline and during the follow-up survey, participants underwent a comprehensive health examination, which included medical history and a lifestyle questionnaire. Body weight and height were measured while participants wore light indoor clothing without shoes. Body mass index was calculated as the ratio of weight (in kilograms) to the square of height (in meters). Waist circumference was assessed using a tape measure (SECA-200; SECA, Hamburg, Germany), as previously described.[23] Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured on the right arm using a standard mercury sphygmomanometer (Baumanometer, Copiague, NY, USA) after the participant had relaxed for 5 minutes. Two measurements were performed at 5-minute intervals, and the average of these two blood pressure readings was used in the study. The proper cuff size was selected for each participant according to mid-arm circumference. Mean arterial pressure (MAP) represents the average arterial pressure during a single cardiac cycle and was calculated using the formula (SBP+2XDBP)/3. The reference range of MAP is 70 to 105 mm Hg. Baseline data on smoking, drinking, and regular exercise were collected with a self-reported questionnaire (yes/no); the protocol has been described elsewhere.[23]

Daily energy intake was determined using a recall questionnaire and was represented in kcals/d. Education level was divided into elementary, middle school, high school, and college. Marital status was determined using the response of either "single" or "married" on a self-reported questionnaire (yes/no). The monthly income of the study participants was categorized into low- (<1 million Korean won), medium- (1–2 million Korean won), and high- (>2 million Korean won) income groups.

After the participants had fasted for more than 12 hours or overnight, venous blood samples were drawn. Fasting blood glucose, triglycerides, total cholesterol, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol concentrations were measured using enzymatic methods by a chemistry analyzer (ADVIA 1650; Siemens, Tarrytown, NY, USA). The fasting insulin was analyzed by a double-antibody radioimmunoassay using a commercial kit (Biosource Europe SA, Nivelles, Belgium). Insulin resistance was determined by Matthews and colleagues[25] using a formula calculated as fasting plasma glucose (milligrams per deciliter)×fasting insulin (milli-international units per milliliter))/22.5 homeostasis model assessment of insulin resistance. High-sensitivity C-reactive protein was measured using the Denka Seiken (Tokyo, Japan) assay.

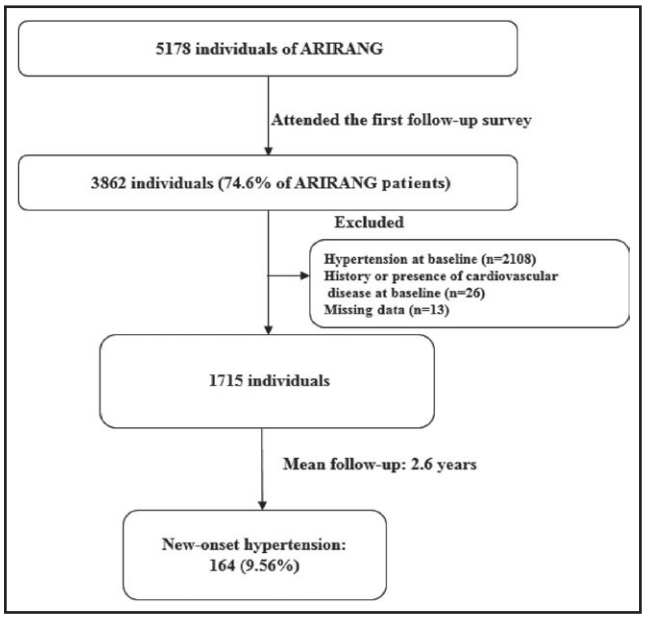

FIGURE 1 Description of the study population. ARIRANG indicates Atherosclerosis Risk in Rural Areas in the Korean General Population.

2.3 | Sleep duration

The study assessed sleep hours through a face-to-face interview. Sleep duration was assessed using a questionnaire conducted with the help of trained interviewers, who inquired, "What was your average daily sleep duration during the past year?" We divided the participants' responses into four groups according to sleep duration: (1) <6 hours; (2) 6 to 7.9 hours; (3) 8 to 9.9 hours; and (4) 10 hours or more. We found that 6 to 7.9 hours was the median of sleep duration in the study sample; therefore, we chose 6 to 7.9 hours as a reference category.

2.4 | End point definition

The study end point was the development of hypertension after 2.6 years of follow-up based on the definition by the Eighth Joint National Committee (JNC 8) guidelines.[26] Hypertension was defined as an SBP of at least 140 mm Hg or a DBP of at least 90 mm Hg or current use of antihypertensive drugs.

2.5 | Statistical analysis

Study results are expressed as the mean±(standard deviation) or frequency. To compare new-onset hypertension and sleep duration (h/d), we conducted a two-sample *t* test, analysis of variance, and chi-square test (Fisher exact test) as applicable. We evaluated the association of baseline sleep duration with the incidence of new cases of hypertension at the follow-up visits over 2.6 years. The 6 to 7.9 hour sleep duration was used as a reference category. Multivariable logistic regression was used to evaluate the independent association of baseline sleep duration with incident hypertension. We used four models (1 [crude], model 2, model 3, and model 4) with a continuous degree of adjustment. The study adjusted for several variables in order to determine the independent association of sleep duration on incident hypertension: age, sex, education, smoking, alcohol status, income, regular exercise, obesity, HDL-C, trigylcerides, glucose, and MAP. The results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). *P* values <.05 were considered statistically significant, and all statistical analyses were performed using SAS version 9.2 (SAS Institute, Cary, NC, USA).

3 | RESULTS

Baseline anthropometrical and biochemical characteristics and socioeconomic factors of the participants based on the new onset of hypertension during an average of 2.6 years are depicted in Table 1. The incidence of hypertension after 2.6 years of follow-up was 9.56% (n=164). Participants who developed hypertension had a significantly higher baseline age, were more frequently male and current drinkers, and exhibited higher weight, waist circumference, SBP, DBP, MAP, triglycerides, total cholesterol, and LDL cholesterol levels than those who did not. The participants who developed hypertension had significantly lower income than those who were normotensive.

The baseline characteristics of the clinical data are shown in Table 2 based on the category of sleep duration. The participants were divided into four groups according to the sleep duration (<6 h/d, 6–7.9 h/d, 8–9.9 h/d, and ≥10 h/d). Participants who slept for <6 h/d were older than those in all other groups. Triglycerides, total cholesterol, LDL cholesterol, and fasting blood glucose were significantly higher in the participants who slept <6 h/d compared with the participants who slept 6 to 7.9 h/d. However, HDL cholesterol and high-sensitivity C-reactive protein levels were significantly higher in short sleepers compared with the reference category. Regular exercise, being a current drinker, and total energy intake were found to be significantly lower or less frequent in participants who got <6 hours of sleep each night compared with the participants who slept 6 to 7.9 h/d. Higher-income participants who experienced fewer sleeping hours were also more likely to be hypertensive compared with the participants who slept 6 to 7.9 h/d. A sleep duration ≥10 h/d was associated with older age, high triglycerides, high cholesterol, low HDL cholesterol, high LDL cholesterol, and high fasting blood glucose levels, lower exercise, and an excessive alcohol intake compared with a sleep duration of 6 to 7.9 h/d. Participants with either a low sleep duration or a high sleep duration made up a smaller percentage of college students compared with the participants who slept 6 to 7.9 h/d.

Table 3 shows the ORs (95% CIs) for new onset of hypertension according to baseline sleep duration. The reference group slept for approximately 6 to 7.9 hours. In the unadjusted model (model 1), the participants who slept <6 h/d were significantly more likely to develop hypertension compared with participants in the reference category. The unadjusted OR in participants who slept <6 h/d was 1.991 (95% CI, 1.201–3.275). Participants with a long sleep duration did not show any significant difference in OR, 1.129 (95% CI, 0.470–2.709) for developing hypertension compared with those who slept 6 to 7.9 hours. In model 2, after adjusting for age and sex, the association between sleep duration and incident hypertension was similar to that of the crude model (model 1). Participants who slept for <6 h/d were found to be at risk for developing hypertension. After adjustment for age, sex, education, smoking, alcohol status, income, regular exercise, and obesity (model 3), a slight reduction in OR was observed in participants who slept <6 hours daily. Model 4 adjusted for the biochemical parameters and metabolic factors along with those of model 2 and model 3. The results continued to be significant for incident hypertension in participants who slept <6 h/d compared with participants who slept for 6 to 7.9 h/d. The corresponding OR for developing hypertension after adjustment for several variables in model 4 was 1.712 (95% CI, 1.014–2.890) compared with the participants with a sleep duration of 6 to 7.9 hours. In contrast, participants who had long sleeping duration did not have increased risk of hypertension (both unadjusted and adjusted models).

4 | DISCUSSION

Short sleep duration (sleeping <6 hours) was significantly associated with new onset of hypertension over 2.6 years of follow-up in a middle-aged and elderly Korean population. This prospective study

TABLE 1 Baseline characteristics of study population by incident hypertension

	Incident Hypertension		
	No	Yes	*P* Value
No. (%)	1551 (90.44)	164 (9.56)	
Age, y	53.31±8.18	55.79±8.00	<.001
Male sex	552 (35.6)	77 (47.0)	.005
Weight, kg	59.83±9.15	64.12±9.80	<.001
WC, cm	80.59±8.44	85.80±7.70	<.001
BMI, kg/m^2	23.69±2.87	25.10±2.71	<.001
SBP, mm Hg	117.23±11.02	122.47±9.99	<.001
DBP, mm Hg	73.82±7.39	75.32±6.91	.014
MAP, mm Hg	88.30±7.66	91.03±7.09	<.001
Triglycerides, mg/dL	127.39±84.06	143.88±84.43	.017
Total cholesterol, mg/dL	196.90±35.78	202.83±34.51	.043
HDL cholesterol, mg/dL	46.72±10.78	45.61±11.41	.214
LDL cholesterol, mg/dL	115.26±30.86	120.43±28.45	.040
FBG, mg/dL	92.95±18.90	95.31±14.96	.122
HOMA-IR, U	1.88±1.16	2.06±1.27	.066
hs-CRP, mg/L	1.68±4.59	2.48±7.79	.202
Regular exercise, %	455/1549 (29.4)	39/163 (23.9)	.171
Current smoker, %	253/1549 (16.3)	29/162 (17.9)	.689
Current drinker, %	581/1546 (37.6)	76/162 (46.9)	.025
Daily energy intake, kcal	2060.62±221.49	2086.59±239.51	.185
Education levels			.121
Elementary	697/1542 (45.2)	86/164 (52.4)	
Middle school	293/1542 (19.0)	34/164 (20.7)	
High school	357/1542 (23.2)	31/164 (18.9)	
College	195/1542 (12.6)	13/164 (7.9)	
Sleep duration, h/d			.100
<6	143 (9.2)	25 (15.2)	
6–7.9	851 (54.9)	82 (50.0)	
8–9.9	492 (31.7)	51 (31.1)	
≥10	65 (4.2)	6 (3.7)	
Married			.378
Yes	1378/1545 (89.2)	142/164 (86.6)	
No	167/1545 (10.8)	22/164 (13.4)	
Monthly income, ×10^4 KRW			.041
<100 (low)	519/1391 (37.3)	72/151 (47.7)	
100–200 (medium)	397/1391 (28.5)	38/151 (25.2)	
>200 (high)	475/1391 (34.1)	41/151 (27.2)	

Data are expressed as number (percentage) or mean±standard deviation. BMI, body mass index; DBP, diastolic blood pressure; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; MAP, mean arterial pressure; SBP, systolic blood pressure; WC, waist circumference.

therefore suggests that a short sleep duration is an important positive predictor for the development of hypertension. However, participants who achieved long sleeping duration (≥10 hours) did not show any significant difference in incident hypertension compared with the study participants who slept for 6 to 7.9 h/d.

In this prospective study, the overall incidence of hypertension was 9.56% after an average of 2.6 years of follow-up. We observed an independent association with an OR of 1.712 (95% CI, 1.014–2.890) for new-onset hypertension in participants who slept <6 h/d compared with those who slept 6 to 7.9 h/d. Previously published studies

TABLE 2 Baseline characteristics of patient data according to sleep duration

	<6 h/d (n=168)	6–7.9 h/d (n=933)	8–9.9 h/d (n=543)	≥10 h/d (n=71)	*P* Value
Incident cases	25 (14.9)	82 (8.8)	51 (9.4)	6 (8.5)	.100
Age, y	56.36±8.12	53.02±8.23	53.42±7.91	54.80±8.49	<.001
Male sex	49 (29.2)	342 (36.7)	211 (38.9)	27 (38.0)	.152
Weight, kg	58.88±9.13	60.22±9.12	60.58±9.75	61.01±8.42	.183
WC, cm	81.26±8.08	80.70±8.41	81.61±8.78	81.68±8.59	.223
BMI, kg/m^2	23.97±3.02	23.75±2.77	23.85±3.08	24.25±2.43	.447
SBP, mm Hg	117.84±11.24	117.67±11.18	118.07±10.68	115.70±11.19	.399
DBP, mm Hg	73.98±7.03	74.08±7.39	73.85±7.42	73.63±7.24	.924
MAP, mm Hg	88.60±7.58	88.61±7.72	88.59±7.53	87.66±7.78	.792
Triglycerides, mg/dL	129.26±129.74	125.23±75.65	131.32±77.06	158.86±100.15	.010
Total cholesterol, mg/dL	203.28±36.32	195.29±35.03	198.05±36.12	207.17±36.66	.004
HDL-C, mg/dL	48.43±10.91	46.29±10.89	46.82±10.71	44.54±10.67	.039
LDL-C, mg/dL	118.60±30.90	114.24±30.31	116.33±31.10	124.31±29.58	.024
FBG, mg/dL	94.80±23.54	92.93±16.63	92.39±14.94	98.42±40.83	.044
HOMA-IR, U	1.97±1.31	1.88±1.08	1.86±1.04	2.20±2.25	.111
hs-CRP, mg/L	1.900±3.681	1.506±3.132	2.178±7.512	1.515±2.254	.087
Regular exercise	25/167 (15.0)	307/931 (33.0)	148/543 (27.3)	14/71 (19.7)	<.001
Current smoker	23/168 (13.7)	150/931 (16.1)	98/541 (18.1)	11/71 (15.5)	.513
Current drinker	52/168 (31.0)	345/931 (37.1)	231/538 (42.9)	29/71 (40.8)	.027
Energy intake (calorie)	2001.19±202.08	2068.67±227.64	2075.23±218.85	2044.29±227.55	.001
Education levels					<.001
Elementary	105/167 (62.9)	376/930 (40.4)	260/539 (48.2)	42/70 (60.0)	
Middle school	29/167 (17.4)	180/930 (19.4)	108/539 (20.0)	10/70 (14.3)	
High school	23/167 (13.8)	234/930 (25.2)	117/539 (21.7)	14/70 (20.0)	
College	10/167 (6.0)	140/930 (15.1)	54/539 (10.0)	4/70 (5.7)	
Married					.376
Yes	143/168 (85.1)	836/929 (90.0)	478/541 (88.4)	63/71 (88.7)	
No	25/168 (14.9)	93/929 (10.0)	63/541 (11.6)	8/71 (11.3)	
Monthly income, ×10^4 KRW					<.001
<100 (low)	92/154 (59.7)	270/849 (31.8)	195/476 (41.0)	34/63 (54.0)	
100–200 (medium)	33/154 (21.4)	255/849 (30.0)	134/476 (28.2)	13/63 (20.6)	
>200 (high)	29/154 (18.8)	324/849 (38.2)	147/476 (30.9)	16/63 (25.4)	

Data are expressed as number (percentage) or mean±standard deviation. BMI, body mass index; DBP, diastolic blood pressure; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; MAP, mean arterial pressure; SBP, systolic blood pressure; WC, waist circumference.

that have defined the association between objective sleep duration and risk of hypertension reported an OR of 1.66 (95% CI, 1.35–2.04) and 1.56 (95% CI, 1.14–2.11), which were quite similar to that found in our study. However, these previous studies used a cross-sectional design.[16,27]

Epidemiological studies and a few meta-analyses have reported the relationship between short sleep duration and risk of hypertension.[17,18,20,22] These studies differed by sample size, follow-up period, ethnic background, criteria of sleep duration, and method of hypertension assessment. In a recent systematic and meta-analysis of longitudinal studies, Guo and colleagues[18] reported that short sleep duration was significantly associated with the development of hypertension; the relative risk in the study was 1.23 (95% CI, 1.06–1.42). Meng and colleagues[28] found a similar observation and reported a relative risk of 1.21 (95% CI, 1.05–1.40) for incident hypertension in participants with a short sleep duration. The biological mechanism underlying the link between short sleep duration and hypertension remains unknown. The association between sleep deprivation and hypertension may be mediated by an increase in sympathetic nervous activity.[29] Sleep deprivation has also been reported to compromise insulin sensitivity and further imbalances the secretion of hunger and satiety hormones (leptin and ghrelin), which lead to obesity and type 2 diabetes, known risk factors

TABLE 3 ORs (95% confidence intervals) for new-onset hypertension according to baseline sleep duration

Incident Hypertension				
	Model 1	Model 2	Model 3	Model 4
Sleep duration, h				
<6	1.991 (1.210–3.275)[a]	1.854 (1.115–3.082)[a]	1.761 (1.048–2.958)[a]	1.712 (1.014–2.890)[a]
6–7.9	1 (reference)	1 (reference)	1 (reference)	1 (reference)
8–9.9	1.148 (0.779–1.692)	1.103 (0.746–1.629)	1.036 (0.698–1.537)	1.053 (0.708–1.565)
≥10	1.129 (0.470–2.709)	1.070 (0.443–2.583)	0.965 (0.396–2.352)	0.940 (0.383–2.309)

Model 1: Crude odds ratio (OR).
Model 2: Adjusted OR: adjusted for age and sex.
Model 3: Adjusted OR: adjusted for age, sex, education, smoking, alcohol status, income, regular exercise, and obesity.
Model 4: Adjusted OR: adjusted for age, sex, education, smoking, alcohol status, income, regular exercise, obesity, high-density lipoprotein cholesterol, triglyceride, glucose, and mean arterial pressure.
[a]$P<.05$ compare with the reference.

for hypertension.[30] Other reports have also suggested that the involvement of endothelial dysfunction and an unhealthy lifestyle could elevate the risk of hypertension in people with sleep deprivation.[31]

It has been reported that sleep quality also plays a significant role in the pathophysiology of incident hypertension. The quality of sleep was calculated by Pittsburgh Sleep Quality Index (PSQI) and recently researchers are interested in measuring sleep quality.[32] Previously published studies have reported that poor sleep quality in individuals was associated with higher prevalence of hypertension.[33,34] Lu and colleagues[35] highlighted the additive interaction of poor sleep quality and short sleep duration on the prevalence of hypertension in adult Chinese men. A follow-up study is needed to understand the true relationship between sleep quality and risk of future development of hypertension. However, our prospective study did not evaluate sleep quality and its association with incident hypertension.

A few cross-sectional and longitudinal studies have demonstrated that short sleep duration is not associated with a greater risk of hypertension. In other words, short sleep duration may not be a risk factor for hypertension in population-based studies. Bansil and colleagues[36] and Hall and colleagues[37] reported an insignificant association between short sleep duration and hypertension from cross-sectional studies. The ORs in their studies were 1.03 (95% CI, 0.91–1.18) and 1.10 (95% CI, 0.75–1.63), respectively. Longitudinal studies conducted by Lopez-Garcia and colleagues[21] and Knutson and colleagues[22] also found an insignificant relationship between short sleep duration and incident hypertension in a Spanish and a US population, respectively. Nonetheless, none of the longitudinal studies thus far have demonstrated an association between long sleep duration and incident hypertension, although cross-sectional studies involving 90 356 participants in a meta-analysis by Wang and colleagues[20] showed that there was a significant association between long sleep duration and risk of hypertension. Previous studies have also identified independent, U-shaped relationships between sleep duration and risk of type 2 diabetes[8] and cardiovascular outcome.[38] Similarly, Fang and colleagues[39] identified a U-shaped relationship between hours of sleep and hypertension in a cross-sectional study. However, our prospective study did not report a U-shaped association between sleep duration and incident hypertension; the adjusted OR for participants with long sleeping hours and incident hypertension was 0.940 (95% CI, 0.383–2.309) compared with participants who slept for 6 to 7.9 hours. The reason for this finding is unknown; therefore, more prospective studies are needed to verify our results with other sleep assessment tools.

To our knowledge, only one prospective study has been reported in the Korean population, and it produced similar findings to our study results; short sleep duration was a significant risk factor for new-onset hypertension.[40] However, the study had different sleep duration categories, and the report focused on genetic associations. The novelty of this study was a population-based longitudinal study with large sample size and adjusted possible confounding factors including socioeconomic factors, energy intake, cholesterol profiles, and mean arterial blood pressure.

4.1 | Study Limitations

Some limitations of this study need to be considered. First, the information in our study about sleep duration was based on self-reported questionnaires that mean the information could not obtain the objective assessment of sleep duration related to sleep quality. Nonetheless, questionnaire-based measures of sleep have demonstrated good agreement against quantitative sleep assessments.[17,41] Second, our data did not provide information about depressive symptoms, which can affect sleep duration.[42] Third, we did not report information regarding insomnia or sleep-disordered breathing, which are both important confounding factors of hypertension. Fourth, our cohort follow-up period was only 2.6 years; therefore, our findings should be verified by a longer prospective study. Finally, we did not exclude participants who were currently taking medications (neuroleptic drugs, glucocorticoids) that might have disturbed their sleep patterns.[43]

5 | CONCLUSIONS

Our study suggests that short sleep duration is independently associated with the development of hypertension among middle-aged and elderly Korean adults. The study highlights the importance of sleep

duration as a risk predictor of incident hypertension and encourages clinicians and researchers to promote healthy sleep habits in society.

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DISCLOSURES

The authors of this manuscript have no conflicts of interest to disclose.

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