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SCIENTIFIC INVESTIGATIONS

Association of sleep trajectory in adulthood with risk of hypertension and its related risk factors: the China Health and Nutrition Survey

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Study Objectives: Few studies have examined the association between sleep duration trajectories and hypertension. This study aims to examine association of sleep duration trajectories with risk of hypertension and its related factors.

Methods: This study used longitudinal data for 7,397 adults who provided valid responses in questionnaire with regard to information of sleep and hypertension from the China Health and Nutrition Survey (2004–2011). Subgroup analyses included 5,532 participants in whom hypertension-related factors were measured using blood samples. Latent class trajectory analysis was used to identify different sleep duration trajectories. Multivariate Cox regression models and general linear regression models were used to assess association of trajectories with hypertension and its related factors.

Results: Compared to stable sleep duration around 8 hours, the trajectory showing a persistent decrease in sleep duration with aging was significantly associated with increased risk of hypertension (hazard ratio 1.12, 95% confidence interval 1.01–1.24), whereas no significant association was observed between the trajectory showing an increase in sleep duration to 9 hours with aging and risk of hypertension (hazard ratio 1.05, 95% confidence interval 0.93–1.19). Further, uric acid levels, fasting glucose levels, total cholesterol levels, and apolipoprotein B levels were significantly higher in the trajectory showing a persistent decrease in sleep duration with aging than the other two trajectories (all $P < .05$).

Conclusions: Decreasing sleep duration during aging is significantly associated with increased risk of hypertension and higher levels of its biomarkers throughout adulthood.

Keywords: biomarker, China Health and Nutrition Survey, hypertension, sleep trajectory

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INTRODUCTION

Hypertension is highly prevalent worldwide in an estimated 1.39 billion persons representing 31% of all adults, and is also a major risk factor for cardiovascular diseases.[1,2] Lifestyle modifications plus drug therapy have been proposed as the best treatment for patients with hypertension.[3] Identifying more modifiable lifestyle risk factors may therefore play an important role in prevention and treatment of hypertension.

In term of lifestyle, sleep is an important physiologic process, which plays an important role during the course of life.[4] Sleep is likely associated with hypertension, probably through regulating stress hormones and the nervous system.[5] However, results from population-based studies regarding association between sleep duration and risk of hypertension remains controversial. U-shape or threshold associations have been documented in these studies.[6–9] For example, meta-analysis based on prospective studies have indicated that long sleep duration was not associated with increased risk of hypertension,[10] whereas the recent cross-sectional studies based on the sample size of more than 700,000 adults showed the opposite.[11] Notably, previous studies frequently adopted single baseline measures of sleep duration, while ignoring variation of sleep duration over time. Previous studies have reported that sleep is a dynamic process and sleep patterns change over the course of an individual's life.[12,13] However, such dynamic changes have not typically been reflected in the sleep epidemiology literature. Therefore, establishing trajectory of sleep duration during the course of adulthood using repeated measurements may add more knowledge for understanding the sleep-hypertension association and provide evidence-based lifestyle recommendations for prevention of hypertension.

With fast economic development and the parallel rise in aging of the population, sleep duration in China is changing.[14] For example, increased consumption of stimulant drinks and widespread use of electronic devices frequently result in short sleep duration.[15] A recent multiethnic study regarding sleep duration indicated that Chinese adults now have shorter sleep duration than other ethnic groups,[16] and associations between poor sleep duration and increased blood pressure, as well as increased risk of hypertension, have been documented in Chinese adolescents and adults.[17–19] These findings presented a unique model for sleep-pattern change, and provided sufficient variation in the shape of sleep duration trajectories to investigate a potential differential association with hypertension. Therefore, using latent class trajectory modeling to characterize sleep duration trajectories over 7 years with longitudinal weight data from China, this study aims to examine the effect of sleep duration trajectories on risk of hypertension and its related factors.

METHODS

The China Health and Nutrition Survey

The China Health and Nutrition Survey (CHNS) is a nationwide survey aiming to investigate health and nutritional status in Chinese populations, which was designed to reflect national age, sex, and education profiles.[20,21] The study sample was drawn from 228 communities of 9 diverse provinces in China including 8 surveys during 1991 to 2011.[22] Forty-seven percent of the Chinese population was constituted in the provinces of the CHNS sample by 2011. Each survey maintains a desired range of economic and demographic circumstances, and new participants are recruited to compensate loses to follow-up. The survey protocols, instruments, and the process for obtaining the informed consent for CHNS participants were approved by the Institutional Review Committees of the University of North Carolina at Chapel Hill, North Carolina, USA, and the China National Institute of Nutrition and Food Safety at the Chinese Center for Disease Control and Prevention, Beijing, China. All participants provided written informed consent prior to completing the surveys.

Study population

Because data regarding on sleep duration were not collected before 2004 surveys, this study included the surveys from 2004 to 2011. After exclusion of participants who have obesity, diabetes, hypertension, and cardiovascular disease at baseline, the current analysis limited eligibility to adult men and nonpregnant women aged 18 years at study entry with at least 2 repeat sleep duration measures to derive trajectories of sleep duration without hypertension at their first visit. The number of visits providing sleep duration measures ranged from 2 to 4 measurement occasions (2 visits, n = 2,395; three visits, n = 2,016; 4 visits, n = 2,986; median = 3 visits; total n = 7,397 participants across 22,782 observations).

Questionnaire

Detailed in-person interviews were administered by trained personnel using a structured questionnaire to collect information on demographic characteristics, dietary habits, lifestyles, physical condition, and anthropometric characteristics. The questionnaire on sleep duration included the question: "How many hours each day do you usually sleep, including during both daytime and nighttime?" Dietary assessment is based on a combination of 3 consecutive 24-hour recall episodes. Food groups were generated based on similar nutrient profiles similar to the food items used in the 2002 Chinese National Nutrition Survey.[21] Current smoking was defined as a positive answer to the question "do you still smoke cigarettes or a pipe?" Information on alcohol consumption was collected with the answers to the question "During the past year, what was your consumption frequency and quantity of beer, liquor, and wine?" Physical activity level was defined as the combination of occupational activity and home activity, as previously reported.[23] The total metabolic equivalents (METs) of physical activity were calculated as MET-hour per week. Urbanicity was defined using a multidimensional 12-component urbanization index capturing community-level physical, social, cultural, and economic environments.[24] Hypertension was defined as self-reports of a history of hypertension diagnosis, or a blood pressure ≥ 140/90 mm Hg or currently taking antihypertension drugs.

Anthropometric measurements and biochemical analyses

At each survey, height was measured without shoes to the nearest 0.2 cm using a portable SECA stadiometer (SECA; Hamburg, Germany). Weight was measured without shoes and in light clothing to the nearest 0.1 kg using a calibrated beam scale. Body mass index (BMI) was calculated as weight (kg) divided by the square of the height in meters (m^2). A 12-mL blood sample was collected by venipuncture after overnight fasting in the 2009 survey. Whole blood was immediately centrifuged and serum was tested for glucose using the glucose oxidase method (Randox, Crumlin, United Kingdom) and a Hitachi 7600 analyzer (Hitachi; Tokyo, Japan). Serum insulin was tested using radioimmunology assay kit (North Institute of Biological Technology; Beijing, China) using a XH-6020 gamma counter (North Institute of Biological Technology). Whole-blood hemoglobin A1C (HbA1c) high- performance liquid chromatography analysis (modelHLC-723G7; Tosoh Corporation, Tokyo, Japan) generated continuous outcomes for fasting glucose, HbA1c, and insulin. Triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) were measured using glycerol phosphate oxidase and enzymatic method (Kyowa, Japan) and a Hitachi 7600 analyzer (Hitachi; Tokyo, Japan). High-sensitivity C-reactive protein was measured using the immunoturbidmetric method (Denka Seiken, Japan), and uric acid was measured using an enzymatic colorimetric method (Randox, Crumlin, United Kingdom) with a Hitachi 7600 analyzer (Hitachi; Tokyo, Japan).

Statistical analysis

All statistical analyses were performed using R 3.4.3 (http://www.r-project.org/). A 2-sided value of $P < .05$ was considered statistically significant. Sleep duration was normalized by Tukey-transformed data to improve the normality of the distribution.

The Latent class trajectory modeling (LCTA) was used to identify sleep duration trajectories through R package *LCMM* with a censored normal model.[25] LCTA has been frequently used to identify trajectory classes in epidemiologic data. Unlike traditional growth curve analysis, assuming that individuals vary around a single mean growth curve, LCTA has the advantage of classifying individuals into distinct groups with similar underlying trajectories. We modeled changes in sleep duration, allowing for a variety of different order polynomials. We used statistically rigorous criteria to determine best fit via: (1) model selection using lowest Bayesian information criterion, a well-accepted model comparison metric often used for latent class models; and (2) inclusion of at least 2% of the sample population within each trajectory class. After trajectories of sleep duration were determined, a nominal categorical variable was created to describe the trajectory membership of each individual, which was then used in central analyses. Further, to avoid the effect of "sick-quitter" on sleep duration trajectories,

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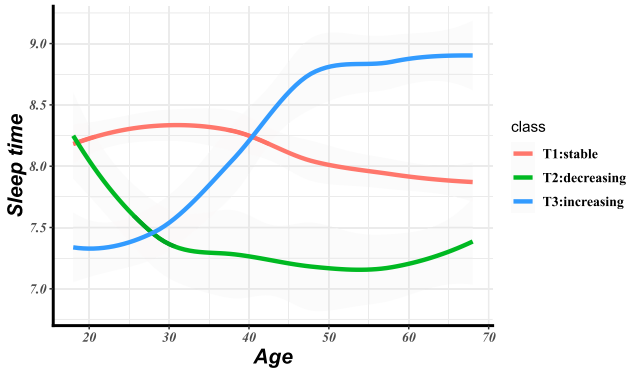
Figure 1—Trajectories of sleep duration in participants (n = 7,397) from the China Health and Nutrition Survey by latent class trajectory model.



this study also performed joint longitudinal latent class model with time-to-event outcome to identify trajectories and then automatically link the latent classes with the outcome by survival curve.[26]

Cox multivariate regression models, with age as the time scale, were used to estimate the association between trajectories of sleep duration throughout life course and risk of hypertension, and hazard ratio (HR) and 95% confidence interval (95% CI) were calculated. Time at entry was the age at the beginning of follow-up, exit time was the age when participants received the diagnosis of hypertension, were lost to follow-up, or were censored at the end of the follow-up period (in 2011), whichever came first. Models were adjusted for age, sex, degree of education, province, rural or city status, alcohol status, smoking status, physical activity, calorie intake, BMI, and urbanization index at baseline. When data for covariates were missing for fewer than 5% of participants, we replaced the missing values with the median values.

Generalized linear models were performed to test differences in continuous hypertension-related outcomes measured in 2009 (fasting glucose, HbA_{1c}, homeostatic model assessment of insulin resistance, TG, HDL-C, uric acid, and high-sensitivity C-reactive protein) across trajectories of sleep duration throughout life, and calculate mean of continuous hypertension-related outcomes with adjustment for age, sex, degree of education, province, rural or city status, alcohol status, smoking status, physical activity, calorie intake, BMI, and urbanization index at baseline.

RESULTS

Trajectories of sleep duration throughout life

Trajectories of sleep duration are shown in **Figure 1**. The first trajectory, labeled "T1:stable," corresponds to participants who have stable sleep duration around 8 hours throughout adulthood. The second trajectory, "T2:decreasing," corresponds to participants who have reduced sleep duration from 8 to 7 hours throughout adulthood. The third trajectory, "T3:increasing," corresponds to participants who have increased sleep duration from 7 to 9 hours throughout adulthood. The trajectories from T1 to T3 were estimated to include 61.1%, 24.8%, and 14.1% of participants, respectively.

Baseline characteristics by different trajectories of sleep duration

Table 1 presents baseline characteristics of studying variables by different trajectories of sleep duration. Participants in the T3 trajectory were older than those in the other two trajectories. BMI, smoking rate, high school education rate, physical activity level, income, calorie intake, and urban index significantly varied across different trajectories of sleep duration (all P for the difference < .05).

Association between sleep duration trajectories and hypertension

Differences of blood pressure across different sleep-duration trajectories are presented in **Table 2**. Mean of accumulated systolic blood pressure, follow-up systolic blood pressure and diastolic blood pressure in the trajectory labeled T2 was significantly higher than those in T1. Associations between sleep duration trajectories and risk of hypertension are presented in **Table 3**. Compared to the stable sleep duration, the trajectory labeled T2 was significantly associated with increased risk of hypertension (HR 1.12, 95% CI 1.01, 1.24) with adjustment for covariates, and no association was observed between trajectory T3 and risk of hypertension (HR 1.05, 95% CI 0.93, 1.19). No sex effect on these associations was observed with $P_{\text{for interaction with sex}} > .05$. Sleep duration trajectories with time-to-hypertension and survival curve of these trajectories are presented in **Figure S1** in the supplemental material. The trajectory with high risk of hypertension showed a varying trend similar to the original trajectory, which was consistent with **Figure 1**, **Table 2**, and **Table 3**.

Trajectories of sleep duration and risk factors of hypertension

Differences for biomarkers across sleep duration trajectories classes are shown in **Table 4**. Uric acid, fasting glucose, total cholesterol (TC), and apolipoprotein B (Apo-B) in the decreasing trajectory were significantly higher than the other two trajectories (all $P_{\text{for trend}} < .05$). HDL, TG, Apo-A, and HbA1c in the decreasing trajectory showed nonsignificant higher trends than the other two trajectories.

DISCUSSION

Using longitudinal data with repeated measurement of sleep duration, we observed that a trajectory showing a persistent decrease in sleep duration with aging was significantly associated with an increased risk of hypertension, and this trajectory had higher uric acid levels, fasting glucose levels, TC levels, and Apo-B levels. Further, a trajectory showing an increase in sleep duration with aging was not associated with reduced risk of hypertension. To best of our knowledge, this study is the first

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Table 1—Baseline characteristics of study variables by different trajectories of sleep duration.

Baseline Variables	Sleep Duration Trajectory			*P*
	T1 (n = 4,521)	T2 (n = 1,835)	T3 (n = 1,041)	
Age (years)	41.4 (12.9)	44.6 (10.9)	52.8 (13.7)	< .001
Male, n (%)	2,104 (46.5)	875 (47.7)	496 (47.6)	.636
Smoking, n (%)	1,290 (28.5)	587 (32.0)	335 (32.2)	.005
Alcohol, n (%)	1,450 (32.1)	647 (35.3)	349 (33.5)	.047
PAL (MET-h/wk)	256.5 (142.4)	256.7 (137.2)	219.4 (132.9)	< .001
High school education, n (%)	1,307 (28.9)	506 (27.6)	180 (17.3)	< .001
BMI (kg/m^2)	22.2 (2.4)	22.5 (2.5)	22.1 (2.5)	< .001
Income (yuan)	13,169 (15,464)	14,313 (15,488)	10,491 (11,673)	< .001
Calorie intake (kilojoules/d)	9,318.5 (2,713.0)	9,072.1 (2,727.1)	9,294.8 (2,751.3)	.002
Urbanization index	64.9 (19.0)	66.9 (18.6)	62.2 (19.0)	< .001
Baseline sleep time (hours)	8.2 (0.86)	7.3 (0.89)	8.3 (1.11)	< .001

Data presented as mean (standard deviation) or n (%) where indicated. Physical activity level (PAL) included 2 aspects: occupational activity and home activity. Hypertension was defined as self-reports of a history of hypertension diagnosis, and/or systolic pressure ≥ 140 mm Hg, and/or diastolic pressure ≥ 90 mm Hg, and/or receiving treatment for hypertension. Generalized linear models and chi-square test were used to probe for differences in continuous variables and dichotomous variables adjusted for age and sex. BMI = body mass index, MET-h = metabolic equivalent hours, T1 = stable, T2 = decreasing, T3 = increasing.

Table 2—Differences of accumulated or follow-up blood pressure across sleep duration trajectories.

Blood Pressure	Sleep Duration Trajectory			*P*
	T1	T2	T3	
Accumulated SBP	115.47 (10.45)	117.88 (10.87)	119.35 (11.90)	< .001
Accumulated DBP	77.66 (7.69)	78.33 (7.63)	78.19 (7.91)	.308
Follow-up SBP	117.86 (15.02)	120.72 (16.14)	122.90 (17.86)	.022
Follow-up DBP	78.29 (10.34)	79.42 (10.84)	78.88 (11.37)	.010

Data presented as mean (standard deviation) in mm Hg. Generalized linear model was used to probe for differences across different trajectories with adjustment for age, sex, smoking rate, alcohol rate, education, metabolic equivalent hours, body mass index, and baseline blood pressure. DBP = diastolic blood pressure, SBP = systolic blood pressure, T1 = stable, T2 = decreasing, T3 = increasing.

Table 3—Association between trajectories of sleep duration and risk of hypertension.

Sleep Duration Trajectory	Incidence of Hypertension			
	Case/n	Model 1	Model 2	Model 3
T1	977/4,521	1.00	1.00	1.00
T2	492/1,835	1.12 (1.00–1.24)	1.12 (1.01–1.25)	1.12 (1.01–1.24)
T3	378/1,041	1.07 (0.94–1.21)	1.05 (0.93–1.19)	1.05 (0.93–1.19)
P		.123	.115	.123
$P_{\text{for interaction with sex}}$		.416	.509	.496

Data presented as hazard ratio (95% CI). Model 1 was adjusted by age and sex. Model 2 was further adjusted by smoking, alcohol consumption, education, metabolic equivalent hours and body mass index. Model 3 was further adjusted by urbanization index. T1 = stable, T2 = decreasing, T3 = increasing.

to examine sleep-hypertension association by establishing a trajectory of sleep duration.

Although association between sleep duration and hypertension has been abundantly studied, inconsistent results regarding U-shape or threshold associations regarding this issue have been documented in previous researches. Establishing a sleep duration trajectory that can capture dynamic changes in sleep duration throughout adulthood may provide more strong evidence for a sleep-hypertension association. In this study, 3 sleep duration trajectories were identified. Compared to the stable trajectory with sleep duration around 8 hours, the trajectory showing a decrease to 7 hours with aging was significantly associated with increased risk of hypertension, indicating that shortened sleep duration could contribute to development

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Table 4—Difference for hypertension-related factors across sleep duration trajectories.

Risk Factors	Sleep Duration Trajectory			*P*
	T1	T2	T3	
Uric acid (μmol/L)	290.79 ± 99.57	311.84 ± 106.47	303.23 ± 103.26	< .001
HDL (mmol/L)	1.45 ± 0.42	1.46 ± 0.50	1.48 ± 0.71	.871
LDL (mmol/L)	2.90 ± 0.91	2.99 ± 0.99	3.02 ± 0.99	.124
Insulin (μIU/mL)	12.95 ± 19.35	13.50 ± 22.72	13.89 ± 27.53	.401
Glu (mmol/L)	5.14 ± 1.10	5.27 ± 1.19	5.25 ± 1.40	.045
ALT (U/L)	23.98 ± 22.90	24.72 ± 17.86	23.10 ± 18.52	.319
TC (mmol/L)	4.75 ± 0.94	4.88 ± 1.01	4.88 ± 0.93	.016
TG (mmol/L)	1.44 ± 1.21	1.54 ± 1.37	1.53 ± 1.42	.169
Hba1c (%)	5.47 ± 0.69	5.60 ± 1.02	5.55 ± 0.70	.078
Apo-A (mg/dL)	115.83 ± 35.72	117.91 ± 42.45	116.06 ± 27.92	.145
Apo-B (mg/dL)	87.16 ± 25.01	91.58 ± 26.00	90.13 ± 25.81	< .001

Data presented as mean ± standard deviation. Generalized linear model was used to probe for differences across different trajectories with adjustment for age, sex, smoking rate, alcohol consumption, education, metabolic equivalent hours, and body mass index. ALT = alanine aminotransferase, Apo = apolipoprotein, Glu = glucose, HDL-C = high-density lipoprotein cholesterol, HbA1c = hemoglobin A1c, LDL = low-density lipoprotein cholesterol, T1 = stable, T2 = decreasing, T3 = increasing, TC = total cholesterol, TG = triacylglycerol.

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of hypertension. This observation was supported by previous prospective studies regarding this issue, which indicate that short sleep duration was associated with incident hypertension.[27] In contrast to these previous prospective studies, this study demonstrated that changes in sleep pattern during aging may be more important, because this association is independent of baseline sleep duration, which was consistent with a previous study that examined the association of changes in sleep duration with hypertension[28] and emphasized the importance of maintaining moderate sleep duration to prevent hypertension. The activation of the sympathetic nervous system was likely the possible mechanism for this association. It has been reported that as sleep duration shortens, the waking state is maintained and the sympathetic nervous system stimulated, increasing nighttime blood pressure levels, heart rate, and elevated salt intake,[29] which increase the risk of hypertension.

Moreover, in comparison with stable sleep duration, the trajectory showing increased sleep duration from 7 hours to 9 hours was not significantly associated with increased risk of hypertension, suggesting that gradually increasing sleep duration during aging probably was not associated with the incidence of hypertension. This observation was partially supported by a recent meta-analysis, which suggested that risk of hypertension did not differ significantly between > 9 hours and 7 hours of sleep.[30] Although this study did not observe an association between increased sleep duration and risk of hypertension, high sleep duration has been reported to be associated with obesity, incident diabetes mellitus, cardiovascular disease, and increased mortality in previous research.[31,32] However, these studies all adopted single baseline measures of sleep duration while ignoring variation of sleep duration over time. Future studies including repeated measurements of sleep duration during adulthood are still needed to validate the observation in this study and may provide more evidence for the association between increased sleep duration and hypertension.

Further, levels of uric acid, fasting glucose, TC, and Apo-B were significantly higher in the decreasing sleep duration trajectory than the other two trajectories, suggesting that short sleep duration probably leads to an increase in these biomarkers, which have all been documented as risk factors for hypertension.[33–35] Previous studies have reported that short sleep duration was associated with high uric acid levels, probably through increased inflammation and oxidative stress.[36,37] For fasting glucose, it has been reported that short sleep duration could decrease insulin sensitivity without adequate compensation in beta-cell function, and could down-regulate the satiety hormone leptin, upregulates the appetite-stimulating hormone ghrelin, and increases hunger and food intake, resulting in weight gain and increased diabetes risk.[38] Several epidemiologic studies also demonstrated that short sleep duration was significantly associated with incident diabetes, findings consistent with this study.[39] For TC and Apo-B, a decrease in the blood concentration of leptin or an increase in the blood concentration of ghrelin due to sleep restriction may be involved in the biologic mechanisms responsible for the associations between short sleep duration and dyslipidemia based on previous studies.[40,41]

This study established sleep duration trajectories, which may properly capture sleep duration variation throughout the course of adulthood and increases our knowledge of the sleep-hypertension relationship. This study is the first on sleep duration trajectories conducted in an Asian population with a relatively large cohort size. However, it also had certain limitations. First, this study only included Asian participants, which is likely to limit the generalizability of our findings to other ethnic populations. Second, as in any observation study, this study is limited by the possibility of residual confounding and measurement error, such as

the determinant sleep duration, the presence of which would affect the accuracy of estimates in this study. Third, this study did not distinguish sleep duration between day and night because this information was not included in the CHNS. Information on insomnia also was also not included in this study. This missing information may lead to some potential bias, such as that the shortest sleep duration in the decreasing sleep group is still longer than 7 hours. Future study modeling of this information may help provide more information regarding this issue. Fourth, the "stable" group still showed general decline in sleep duration over time, which probably minimized results in comparison with the "decreasing" group.

In conclusion, this study demonstrated that gradually decreased sleep duration throughout adulthood was significantly associated with high levels of risk factors and increased incidence of hypertension, emphasizing the importance of adequate sleep duration in prevention of hypertension. Further, gradually increased sleep duration during aging was not associated with risk of hypertension.

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DISCLOSURE STATEMENT

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