ORIGINAL ARTICLE

Associations of Sleep Duration and Disturbances With Hypertension in Metropolitan Cities of Delhi, Chennai, and Karachi in South Asia: Cross-Sectional Analysis of the CARRS Study

Roopa Shivashankar MD, MSc[1,2,3]; Dimple Kondal PhD[1,2,3]; Mohammed K. Ali MD, MSc, MBA[1,4]; Ruby Gupta PhD[1,2]; Rajendra Pradeepa PhD[5]; Viswanathan Mohan MD, FRCP, PhD, DSc[5]; Muhammad Masood Kadir MBBS MPH MOPS[6]; K.M. Venkat Narayan MD, MSc[1,4]; Nikhil Tandon MD, PhD[1,7]; Dorairaj Prabhakaran DM, MSc, FRCP, FNASc[1,2,3,8]; Anne Peasey MSc, PhD[8,9]

[1]Centre for Control of Chronic Conditions (4C), New Delhi, India; [2]Public Health Foundation of India, New Delhi, India; [3]Centre for Chronic Disease Control, New Delhi, India; [4]Emory Global Diabetes Research Center, Rollins School of Public Health, Emory University, Atlanta, GA; [5]Madras Diabetes Research Foundation and & Dr. Mohan's Diabetes Specialties Centre, Chennai, Tamil Nadu, India; [6]Department of Community Health Sciences, Aga Khan University, Karachi, Pakistan; [7]Department of Endocrinology and Metabolism, All India Institute of Medical Sciences, New Delhi, India; [8]London School of Hygiene and Tropical Medicine, London, United Kingdom; [9]Department of Epidemiology and Public Health, University College London, London, United Kingdom

Objectives: Sleep duration and disturbances may be risk factors for hypertension. Despite the high burden of hypertension in South Asia, little is known about this relationship in this region.

Methods: We analyzed population-level cross-sectional data from the Centre for Cardiometabolic Risk Reduction in South Asia (CARRS) study that recruited representative samples of adults ≥ 20 years from three cities—Delhi, Chennai (India), and Karachi (Pakistan) during 2010–2011. We defined hypertension as self-reported treatment or measured blood pressure (BP) ≥140/90 mm Hg. Data on usual duration of sleep, insomnia, and snoring were collected using "The Sleep Habits Questionnaire" and excessive daytime sleepiness (EDS) using Epworth Sleepiness Score. Logistic and linear regression were done with hypertension and BP as outcome variables, respectively. Age, gender, education, wealth index, family history, and body mass index (BMI) were included as covariates. We used multiple imputation to account for missing variables.

Results: Prevalence of hypertension was 30.1%. The mean (*SD*) sleep duration was 7.3 (1.2) hours. Insomnia, snoring, and EDS were present in 13.6%, 28.7%, and 4.6%, respectively. Moderate and habitual snoring were associated with increased odds of hypertension (odds ratio [OR] = 1.18, 95% confidence interval [CI] [1.04 to 1.33] and 1.47 [1.29 to 1.67], respectively), after adjusting for covariates. Rare, occasional, and frequent insomnia were associated with increased hypertension (OR 1.41 [1.12 to 1.77], 1.39 [1.16 to 1.67], and 1.34 [1.09 to 1.65], respectively). Sleep duration and EDS were not associated with hypertension.

Conclusion: Self-reported snoring and insomnia were associated with hypertension in South Asia. This relationship needs further exploration through robust longitudinal studies in this region.

Keywords: hypertension, blood pressure, sleep duration, insomnia, snoring, daytime sleepiness, South Asia.

Statement of Significance

This study addresses the important lacunae in population-level sleep and health relationship in rapidly developing South Asian cities. The study found high prevalence insomnia and self-reported snoring among adults in urban South Asia. The odds of hypertension were 18% and 47% higher for moderate and habitual snorers and 34–41% higher among participants with insomnia. Duration of sleep and excessive daytime sleepiness were not associated with hypertension.

INTRODUCTION

Rapid unplanned urbanization and globalization in South Asia have resulted in adoption of lifestyles that includes high consumption of calorie-dense foods, low physical activity, and psychological stresses leading to overweight and an increased susceptibility to hypertension, diabetes, and cardiac diseases.[1] Lifestyle changes, stress, and obesity are also risk factors for sleep loss and disturbances.[2,3] Urban lifestyles are associated with longer commutes to work, more hours spent watching television, and habitual internet use which can potentially result in shorter sleep duration and disturbances. Additionally, sleep duration[4,5] and disturbances are possible risk factors for hypertension.[6–8] Insomnia, a disorder in which there is difficulty in falling asleep or staying asleep or both, despite adequate opportunity to sleep,[9] is considered a risk factor for hypertension,[10] but the evidence for this is not consistent.[5,6,11,12] Snoring, a symptom of obstructive sleep apnea (OSA),[13,14] is identified as an independent risk factor for hypertension.[7,15–17] But there is little evidence on independent associations between excessive daytime sleepiness (EDS), another cardinal symptom of OSA and hypertension.[15,18]

In South Asia, population data on usual duration and disturbances of sleep (insomnia, snoring, and EDS) are limited.[19,20] Nearly, one-third of the adult populations in countries such as India and Pakistan are estimated to have hypertension.[21–23] Despite this high burden, the relationship of hypertension with sleep duration and disturbances have remained largely unexplored. To address this gap, we examined potential associations between sleep parameters and hypertension by analyzing data from a cross-sectional representative survey of 16 287 adults aged ≥20 years in three mega-cities (Chennai, Delhi, and Karachi) of South Asia.

METHODS

We used data from the baseline survey of the Centre for Cardiometabolic Risk Reduction in South Asia (CARRS) study collected during 2010–2011.[24] The methods, participant recruitment, and data collection in CARRS cohort study are published

in detail elsewhere.[24] Briefly, participants were recruited by multistage cluster random sampling technique stratified by gender and city of residence. Primary sampling units (PSUs) were wards or larger municipal subdivisions for Delhi and Chennai, respectively, while clusters were the PSUs for Karachi. At the time of data collection, the most recent census in each country (India—2001 census; Pakistan—1998 census) was used to randomly select the wards, clusters, or census enumeration blocks (CEB; smallest municipal subdivision in Indian cities) and households. In order to account for changes that may have happened in the interim, we manually listed and mapped all households in each CEB before randomly selecting them.[24,25] The "Kish Method," used in the World Health Organization (WHO)'s STEPS surveys,[26] was used to select two participants (one male and one female), aged 20 years or older from each household. Pregnant women, bed-ridden individuals, and persons unable to understand the questionnaire due to severe mental illness were excluded. Response rates were 94·7% for questionnaire completion and 84·3% for bio-specimens.[25]

Data were collected through personal interviews at the participants' homes using structured questionnaires. Blood pressure was measured twice at participants' homes by trained study staff using an electronic sphygmomanometer (Omron Dailan Co., Ltd, Dalian, Liaoning, China) in a seated position with a 5-minute gap between the measurements. A third measurement was obtained if the difference between the first two systolic (SBP) or diastolic (DBP) measurements was more than 10 mm Hg or 5 mm Hg, respectively. The time of blood pressure measurement varied between participants and was usually between 7 am and 4 pm. The mean of the first two BP measurements, or the second and third measurements if a third measurement was taken, was used for analyses. Participant was classified as having hypertension if SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg or self-reported hypertension medication.[27]

Data on sleep habits were collected using "The Sleep Habits Questionnaire" to assess usual sleep habits and sleep disorders including insomnia and snoring; this questionnaire has been used previously in Sleep Heart Health Study.[28] The Epworth sleepiness scores (ESS)[29,30] was used to measure daytime sleepiness. The English questionnaires were translated to local languages (Tamil, Hindi, and Urdu) and back-translated to English. The questionnaires were piloted, and interviewer debriefing method was used assess any difficulty or variation in understanding of the items in the questions, and wordings of the questionnaire were modified accordingly.[24] To assess internal consistency (reliability) for insomnia and ESS, we measured Cronbach's α for overall population and subgroups (age, gender, and city). Participants were asked about sleep duration (number of hours) during usual weekdays and weekends. The average duration of sleep per day was obtained by [(average of duration of sleep during weekdays × 6) + (average duration of sleep during weekends × 1)]/7. Weekdays was multiplied by 6 because most establishments in South Asia have 6 working days a week. The average duration of sleep was categorized as <5, 5–5.9, 6–6.9, 7–7.9, 8–8.9, and ≥ 9 hours.[4] Participants were asked if they experienced any of the following experiences of insomnia—"Have trouble falling asleep"; "Wake up during the night and have difficulty getting back to sleep"; "Wake up too early in the morning and unable to get back to sleep"; and "Take sleeping pills or other medication to help you sleep" over past month. Insomnia was categorized into "no," "rare," "occasional," and "frequent" if the frequency of any insomnia was <2, 2–4, 5–15, and 16–30 nights/ month, respectively. If an individual had more than one symptom, insomnia was categorized based on the most frequent symptom.[4] The Cronbach's α for insomnia was 0.81, overall and varied between 0.78 and 0.85 for various subgroups.

Participants were asked if they ever snored and if yes, how often they snored. They were coded as nonsnorer if they either never snored or did not snore anymore, moderate snorer if they said rarely or sometimes (up to two nights per week), and habitual snorer if they answered frequently, almost always, or always to the question.[31] Regarding excessive daytime sleepiness (EDS), the ESS was used and categorized participant responses to chances (no chance = 0; slight chance = 1; moderate chance = 2; high chance = 3) of falling asleep in the following eight situations—sitting and reading; watching television; sitting inactive in a public place (such as a theatre or a meeting); riding as a passenger in a car for an hour without a break; lying down to rest in the afternoon when circumstances permit; sitting and talking to someone; sitting quietly after a lunch; in a car, while stopped for a few minutes in traffic. The scores for all items were added together. A participant was coded as unlikely sleepy, average day time sleepiness, excessive sleepiness depending on the situation, and excessive sleepiness if the total score was 0–7, 8–9, 10–15, and 16–24, respectively, based on ESS.[29] There were only 50 participants with ESS 16–24; therefore, this category was merged with ESS 10–15 and renamed EDS. The Cronbach's α for daytime sleepiness was 0.80 overall and varied between 0.78 and 0.85 for various subgroups.

Regarding participant characteristics, self-reported age at baseline in completed years was used and categorized into three age groups: 20–44, 45–59, and 60+ years. Participant-reported gender was used to code individuals as male or female. Self-reported highest education level attained was categorized into four categories—up to primary schooling; high or secondary schooling; up to university education; and university and higher. A wealth index based on different household amenities (separate cooking room and toilet facilities) and assets (television, refrigerator, washing machine, microwave, mixer-grinder, mobile phone, DVD player, computer, car, motor cycle, and bicycle) was used. Total scores were categorized into tertiles (lowest tertile representing poorest and highest tertile representing wealthiest).[25] Self-reported current use of smoked tobacco (both cigarettes and bidis), history of heart disease, and stroke was obtained from the questionnaire. We used a modified food frequency questionnaire to estimate average servings of fruits and vegetable intake per day and coded these into <2, 2–4, and ≥5 servings a day.

Anthropometric parameters (height, weight, body composition, waist circumference, hip circumference, and skin-fold thickness) were measured using standard techniques either at participant's home or at temporary blood collection clinics organized closer to participant's home. Measured height (meters) and weight (kilograms) was used to calculate body mass index (BMI) as weight/height.[2] A 15-ml fasting venous blood sample was collected for biochemical measurements. Fasting plasma glucose (FPG) was estimated using hexokinase/kinetic methods and total cholesterol (TC) estimated by enzymatic colorimetric cholesterol oxidase peroxidase.[25,32]

ETHICAL ISSUES

The CARRS Surveillance study was approved by the independent ethics committees of Public Health Foundation of India, All India Institute of Medical Sciences, New Delhi; Madras Diabetes Research Foundation, Chennai, India; Aga Khan University, Karachi, Pakistan;and Emory University, Atlanta, Georgia. The CARRS Study obtained informed consent from all participants.

ANALYSIS

This analysis included 16 287 participants. The number of missing values in CARRS data set has been published previously.[25] In short, missing values in questionnaire data, blood pressure, BMI, blood biochemistry, and overall were 0%, 4.9%, 23%, 15.8%, and 25%, respectively. To account for the missing variables, we used multiple imputation using chained equation (MICE) for imputing all missing values across all variables to create 10 completed data sets. All covariates and the outcome were included in the imputation model. Imputation methods are described in greater detail elsewhere.[25] Imputed values of missing continuous variables were modeled using linear regression and predictive mean matching, and imputed values of ordinal variables were modeled using ordinal logistic regression. Model convergence was checked, and diagnostics were performed on the imputed data set. Sample weighting was taken into account in the data analysis using stata command svyset, with a "ward" variable as primary sampling unit.[25,32,33] Standard weights and strata (6 strata of gender and age groups 20–44, 45–59, and 60+ years) were used obtain weighted estimates. All analysis commands were prefixed with "svy" to obtain weighted percentages, means, coefficients or odds ratios, and 95% confidence intervals (CIs). The distribution of sleep variables by city and gender were assessed as percentages with 95% CIs.

We assessed collinearity between sleep variables by cross tabulation (results not shown) and found no significant collinearity between the sleep variables. For all regression analyses, 7–7.9 hours was used as the reference category for duration of sleep. We used logistic regression to model hypertension. Each sleep variable was modeled separately with hypertension to obtain unadjusted odds ratios (model 1). Individual sleep variables were then modeled adjusting for age as continuous variable and gender (model 2). In model 3, we further adjusted for sociodemographic variables—education, wealth index, and city. In the model 4, we assessed for potential confounding by other variables (BMI, smoking, alcohol use, self-reported heart disease, stroke, family history of hypertension, fruits and vegetables intake, FPG, and TC) using forward stepwise regression starting from the variables most strongly associated with hypertension in the bivariate analysis. Only BMI was found to confound the associations between sleep variable and hypertension, and therefore retained in model 4. Finally, we adjusted for other sleep variables in model 5. We further assessed the interaction between sleep duration and insomnia categories and between snoring and EDS on their effects on hypertension. We computed predicted probabilities and 95% CIs (using robust standard errors) of hypertension for each of the sleep categories using model 5. We assessed interaction of age (20–44, 45–59, and ≥60 years) and gender with sleep variables by introducing interaction term. We considered effect modification if the p value of Wald test of interaction term was <.05. Further, we computed predicted probabilities and 95% CIs (using robust standard errors) of hypertension for each of the sleep categories stratified by age and gender. BMI was one of the imputed variables; therefore, stratification by BMI (which may vary between 10 imputed data sets) could have introduced bias. Hence, we were only able to assess effect modification of BMI by stratifying on BMI category (<18.5, 18.6–24.9, ≥25kg/m^2) for the nonmissing complete cases. In addition, we assessed the independent effect of separate symptoms of insomnia: initial (have trouble falling asleep); middle (wake up during the night and have difficulty getting back to sleep); late (wake up too early in the morning and be unable to get back to sleep) and medication for insomnia on hypertension using logistic regression adjusting for each other, other sleep factors, age, gender, education, wealth index, city, and BMI. None of the logistic regression models were adjusted for hypertension medication.

Further, we did two separate linear regressions with SBP and DBP as continuous outcome variables adjusting for medication for hypertension (model 1) and adding age, gender, education, wealth index, city, BMI, and other sleep variables (model 2) using multiple imputed data set. Forty-three observations (0.26%) who reported not knowing their snoring patterns were dropped from the analysis in all the models.

Finally, we performed a sensitivity analysis between the multiply imputed and complete case data sets. The weighted percentages (means, if continuous variable) of sociodemographic variables, exposures, and hypertension and adjusted estimates of logistic and linear regression models in both data sets were compared. We used Stata 14.2 for all statistical analyses.

RESULTS

The number of participants recruited from Chennai, Delhi, and Karachi were 6906, 5364, and 4017, respectively. Mean (standard deviation [SD]) age of participants total and city wise (Chennai, Delhi, and Karachi) were 42.3 (12.6), 41.7 (12.2), 44.1 (12.2) and 41.7 (13.9) years, respectively.

Women constituted 52.4%, 53.9 %, 49.9 %, and 52.9% overall, Chennai, Delhi, and Karachi, respectively. The mean (SD) years of schooling in all, Chennai, Delhi, and Karachi, were 8.0 (5.0), 7.5 (4.2), 8.7 (5.4) and 7.1 (5.3), respectively. The complete description of CARRS population has been published previously.[25]

Table 1 shows the distribution of duration of sleep and sleep disturbances in the study population by gender and city. Overall, 5.5% and 1.9% of participants slept <5 and 5–6 hours, respectively. This distribution varied by city, with Chennai having lowest prevalence of short sleep and Karachi, the highest. About 12.4% of participants slept greater than 9 hours on average. This again varied by city, with Delhi having lowest prevalence of long sleepers and Karachi, the highest. The mean (SD) duration of sleep was 7.3 (1.2) hours in total population which was longest in Chennai women (7.7 [1.1] hours) and shortest in Delhi men (6.9 [1.1] hours). With respect to insomnia, about 12% of participants reported insomnia: rare, occasional, or frequent. Women reported higher prevalence of insomnia than men in all three cities; the difference between genders was particularly striking in Karachi. Moderate and habitual snoring was reported by 14.5% and 14.2% of participants, overall. Snoring was more common among men than women overall. However, in Karachi, habitual snoring was more common among women compared to men. EDS was the least common sleep problem in these South Asian

Table1—Distribution of sleep variables by gender and city (N = 16 287)[a].

Sleep Variables	All			Chennai		Delhi		Karachi	
	Total	Men	Women	Men	Women	Men	Women	Men	Women
Numbers[b]	**16 287**	**7760**	**8527**	**3188**	**3788**	**2680**	**2684**	**1892**	**2125**
Duration of sleep, hours									
<5	**5.5 (5.0 to 6.0)**	5.2 (4.5 to 5.9)	5.8 (5.0 to 6.5)	3.0 (2.2 to 3.9)	2.2 (1.5 to 2.8)	5.1 (3.9 to 6.2)	6.2 (5.0 to 7.4)	8.8 (7.3 to 10.3)	11.4 (9.2 to 13.7)
5–5.9	**1.9 (1.6 to 2.2)**	1.8 (1.3 to 2.2)	2.0 (1.6 to 2.5)	0.8 (0.3 to 1.4)	0.6 (0.2 to 1.0)	2.5 (1.6 to 3.3)	2.6 (1.9 to 3.3)	2.3 (1.8 to 2.8)	3.8 (3.0 to 4.6)
6–6.9	**21.1 (19.9 to 22.3)**	23.0 (21.2 to 24.7)	19.4 (17.7 to 21.1)	14.4 (12.2 to 16.6)	11.4 (9.7 to 13.2)	30.4 (27.8 to 33.0)	24.6 (22.4 to 26.8)	26.7 (24.5 to 28.8)	26.5 (24.6 to 28.5)
7–7.9	**28.4 (27.3 to 29.4)**	27.9 (26.6 to 29.2)	28.8 (27.2 to 30.3)	24.7 (22.6 to 26.8)	27.5 (25.5 to 29.5)	35.9 (33.8 to 38.0)	35.1 (32.4 to 37.7)	21.8 (20.1 to 23.5)	23.1 (20.7 to 25.4)
8–8.9	**30.8 (29.0,32.6)**	31.3 (29.3 to 33.3)	30.3 (27.5 to 33.2)	41.4 (38.5 to 44.4)	41.4 (38.8 to 44.1)	20.9 (18.7 to 23.0)	26.3 (23.2 to 29.4)	29.4 (27.7 to 31.1)	16.3 (14.1 to 18.5)
≥9	**12.4 (11.4 to 13.4)**	10.9 (9.6 to 12.2)	13.7 (12.3 to 15.1)	15.6 (13.4 to 17.8)	16.9 (14.4 to 19.4)	5.3 (4.0 to 6.7)	5.2 (4.1 to 6.4)	11.1 (9.5 to 12.6)	18.9 (17.0 to 20.7)
Mean (SD)	**7.3 (1.2)**	7.2 (1.2)	7.3 (1.2)	7.6 (1.1)	7.7 (1.1)	6.9 (1.1)	7.0 (1.1)	7.1 (1.2)	7.1 (1.2)
Insomnia, nights/month									
No	**86.5 (85.2 to 87.8)**	91.2 (90.4 to 92.1)	82.2 (80.2 to 84.1)	91.5 (90.3 to 92.8)	89.0 (87.4 to 90.5)	94.3 (93.2 to 95.5)	83.7 (81.4 to 86.0)	86.4 (84.5 to 88.2)	68.4 (65.6 to 71.2)
Rare	**4.9 (4.4 to 5.4)**	3.0 (2.6 to 3.5)	6.6 (5.8 to 7.3)	3.3 (2.5 to 4.1)	4.9 (3.8 to 5.9)	2.0 (1.4 to 2.7)	8.5 (7.2 to 9.7)	4.1 (3.1 to 5.1)	7.1 (5.7 to 8.5)
Occasional	**4.7 (4.0 to 5.4)**	3.1 (2.5 to 3.6)	6.1 (5.0 to 7.3)	2.0 (1.3 to 2.7)	2.2 (1.6 to 2.8)	2.1 (1.5 to 2.8)	4.1 (3.1 to 5.2)	6.1 (4.9 to 7.2)	15.5 (13.5 to 17.6)
Frequent	**4.0 (3.5 to 4.4)**	2.6 (2.2,3.1)	5.1 (4.5 to 5.8)	3.2 (2.5 to 3.8)	4.0 (3.1 to 4.9)	1.5 (1.0 to 2.1)	3.7 (2.9 to 4.4)	3.4 (2.6 to 4.2)	9.0 (7.4 to 10.5)
Snoring frequency									
Nonsnorer	**71.0 (69.8 to 72.3)**	65.2 (63.3 to 67.1)	76.4 (74.8 to 77.9)	67.0 (64.3 to 69.7)	83.1 (81.5 to 84.7)	55.5 (52.0 to 59.1)	67.2 (64.3 to 70.1)	75.8 (72.6 to 79.1)	76.3 (72.7 to 79.8)
Moderate snorer	**14.5 (13.7 to 15.3)**	17.9 (16.6 to 19.1)	11.5 (10.6 to 12.4)	20.0 (18.2 to 21.9)	9.6 (8.3 to 10.9)	18.3 (16.1 to 20.4)	15.1 (13.0 to 17.1)	13.7 (11.9 to 15.4)	10.4 (8.9 to 12.0)
Habitual snorer	**14.2 (13.3 to 15.1)**	16.7 (15.3 to 18.1)	11.9 (10.8 to 13.0	12.4 (10.3 to 14.5)	6.9 (5.8 to 8.0)	26.1 (23.6 to 28.7)	17.6 (15.7 to 19.5)	10.4 (8.4 to 12.5)	13.3 (10.8 to 15.8)
Don't know	**0.2 (0.1 to 0.3)**	0.3 (0.1 to 0.4)	0.2 (0.1 to 0.4)	0.6 (0.2 to 0.9)	0.3 (0.1 to 0.6)	0.0 (0.0 to 0.1)	0.2 (0.0 to 0.4)	0.0 (0.0 to 0.1)	0.1 (-0.1 to 0.2)
Daytime sleepiness									
Unlikely	**95.4 (94.9 to 96.0)**	96.1 (95.4 to 96.8)	94.9 (94.0 to 95.7)	98.2 (97.4 to 99.1)	98.6 (97.9 to 99.3)	95.7 (94.7 to 96.7)	87.8 (85.9 to 89.8)	93.0 (91.6 to 94.4)	97.2 (96.3 to 98.1)
Average	**2.8 (2.4 to 3.2)**	2.2 (1.7 to 2.6)	3.4 (2.7 to 4.0)	0.9 (0.5 to 1.4)	1.1 (0.4 to 1.8)	2.2 (1.5 to 2.9)	8.0 (6.2 to 9.8)	4.1 (3.2 to 5.0)	1.4 (1.0 to 1.9)
Excessive[c]	**1.8 (1.5 to 2.1)**	1.8 (1.4 to 2.2)	1.8 (1.3 to 2.3)	0.8 (0.2 to 1.4)	0.3 (0.1 to 0.4)	2.1 (1.3 to 2.9)	4.2 (3.0 to 5.4)	2.9 (2.2 to 3.7)	1.4 (0.7 to 2.1)

The bold values indicate for whole population vis a vis others are of sub groups.
[a]Estimates are in percentage and 95% confidence interval unless specified.
[b]Raw numbers.
[c]Excessive daytime sleepiness was defined as Epworth Sleepiness Score ≥ 10 because group ≥ 15 had only 50 observations and therefore was merged with 10–15 group.

cities with 2.8% and 1.8% reporting average and EDS, respectively. EDS was lowest in Chennai and highest in Karachi.

Table 2 presents the results of logistic regression analyses examining the relationships of sleep variables with hypertension. In unadjusted logistic regression (model 1), the odds of hypertension were higher with shorter duration of sleep and lower with longer duration of sleep when compared to 7–8 hours of sleep. However, this relationship did not hold after adjustment for sociodemographic

Table 2—Logistic regression models of association between sleep variables and hypertension (N = 16 244[a]).

Sleep variables	Odds ratios (95% CI)					Predicted probability of hypertension from model- 5[b], % (95%CI)
	Model 1 (Crude)	Model 2 (age and gender adjusted)	Model 3 (model 2+ education, wealth index and city)	Model 4 (model 3+ BMI)	Model 5 (model 4+ sleep variables)	
Duration of sleep, hours						
<5	1.35 (1.15 to 1.60)	1.03 (0.87 to 1.22)	1.05 (0.88 to 1.25)	1.07 (0.89 to 1.28)	0.99 (0.82 to 1.19)	29.9 (26.8 to 33.0)
5–5.9	1.13 (0.86 to 1.49)	1.12 (0.84 to 1.50)	1.07 (0.80 to 1.43)	1.08 (0.81 to 1.45)	1.00 (0.74 to 1.35)	30.1 (24.7 to 35.4)
6–6.9	1.10 (0.98 to 1.23)	0.98 (0.87 to 1.11)	0.97 (0.86 to 1.10)	0.98 (0.86 to 1.10)	0.97 (0.86 to 1.10)	29.5 (27.9 to 31.2)
7–7.9	reference					30.0 (28.4 to 31.7)
8–8.9	0.90 (0.79 to 1.01)	0.92 (0.81 to 1.04)	0.99 (0.87 to 1.13)	0.99 (0.87 to 1.14)	1.01 (0.89 to 1.16)	30.3 (28.4 to 32.1)
≥9	0.79 (0.67 to 0.94)	0.88 (0.75 to 1.04)	0.99 (0.84 to 1.16)	1.02 (0.86 to 1.20)	1.02 (0.86 to 1.21)	30.4 (27.7 to 33.2)
Insomnia, nights/month						
No	reference					29.2 (28.1 to 30.4)
Rare	1.58 (1.29 to 1.95)	1.46 (1.18 to 1.80)	1.44 (1.17 to 1.78)	1.41 (1.13 to 1.77)	1.41 (1.12 to 1.77)	35.5 (31.3 to 39.6)
Occasional	1.61 (1.36 to 1.91)	1.41 (1.20 to 1.66)	1.44 (1.21 to 1.70)	1.43 (1.20 to 1.70)	1.39 (1.16 to 1.67)	35.2 (31.8 to 38.6)
Frequent	1.61 (1.30 to 1.99)	1.3 (1.06 to 1.60)	1.38 (1.13 to 1.68)	1.36 (1.10 to 1.67)	1.34 (1.09 to 1.65)	34.5 (30.5 to 38.5)
Snoring frequency						
Nonsnorer	reference					28.4 (27.2 to 29.6)
Moderate snorer	1.81 (1.62 to 2.02)	1.48 (1.31 to 1.66)	1.43 (1.27 to 1.60)	1.18 (1.05 to 1.34)	1.18 (1.04 to 1.33)	31.3 (29.2 to 33.5)
Habitual snorer	2.72 (2.42 to 3.05)	2.03 (1.80 to 2.29)	1.87 (1.65 to 2.12)	1.49 (1.31 to 1.69)	1.47 (1.29 to 1.67)	35.4 (33.2 to 37.7)
Daytime sleepiness						
Unlikely	reference					30.1 (29.0 to 31.2)
Average	1.21 (0.96 to 1.53)	1.09 (0.85 to 1.39)	0.97 (0.76 to 1.23)	0.92 (0.72 to 1.17)	0.91 (0.71 to 1.16)	28.4 (24.3 to 32.5)
Excessive	1.39 (1.07 to 1.82)	1.15 (0.86 to 1.52)	1.06 (0.79 to 1.41)	1.02 (0.75 to 1.38)	0.92 (0.68 to 1.25)	28.7 (23.5 to 33.8)

Abbreviations: BMI, body mass index; CI, confidence interval.
[a]Raw numbers, N = 16 244 as 43 observations who reported "don't know" for snoring were dropped from the analysis.
[b]Covariates used- age, gender, city, education, wealth index, body mass index, and other sleep variables.

variables, and the fully adjusted model 5 showed no relationship between sleep duration and hypertension. Participants with rare, occasional and frequent insomnia had significantly higher odds of hypertension compared to those with no insomnia. The effect size was slightly attenuated after adjusting for age and gender but remained significant. The magnitudes of these associations were unchanged in the remaining adjusted models. The adjusted odds ratio of hypertension for early, middle, late, and medication for insomnia were 1.09 (0.85 to 1.39), 1.44 (1.06 to 1.96), 0.87 (0.64 to 1.20), and 1.85 (1.33 to 2.59), respectively, (not in the table).

Both moderate snorers and habitual snorers had significantly higher odds of hypertension compared to nonsnorers in model 1. However, the strength of this association was attenuated after adjusting for age and gender in model 2 and BMI in model 4. Adjusting for other sleep variables had no further substantial impact on the magnitude of associations (model 5). When assessing for patterns of relationships across snoring categories, there were significant associations between snoring categories and hypertension (Wald test p value < .001 [data not shown]). In unadjusted models, the odds of hypertension were significantly

higher if the participant had EDS; however, this association no longer existed after adjusting for age and gender in model 2 and remained nonsignificant in all further models (Table 2).

Table 3 presents that association of sleep variables and hypertension, stratified by age groups. The associations between snoring and hypertension were more pronounced in young participants (20–44 years), and there was no association in the oldest group (≥60 years). There was a significant interaction between sleep duration and age groups. The odds ratio of hypertension was significantly higher among young adults who slept 6–6.9 hours but no significant association in other age-groups. Additionally, associations between insomnia and hypertension

Table 3—Logistic regression models of association between sleep variables and hypertension stratified by age group (*N* = 16 244[a]).

Sleep Characteristics	Adjusted odds ratios (95% CI) [b]				Predicted probabilities of hypertension % (95% CI) [c]		
	20–44 years	45–59 years	≥ 60 years	p [d]	20–44 years	45–59 years	≥ 60 years
Duration of sleep, hours							
<5	0.99 (0.75 to 1.32)	1.10 (0.83,1.46)	0.65 (0.40 to 1.08)	.0135	19.9 (15.7 to 24.1)	48.1 (42.1 to 54.1)	54.0 (43.4 to 64.7)
5–5.9	1.46 (1.03 to 2.09)	0.66 (0.43 to 1.03)	0.31 (0.12 to 0.80)		26.3 (19.6 to 33.0)	36.7 (27.3 to 46.0)	36.8 (16.5 to 57.2)
6–6.9	0.96 (0.81 to 1.14)	1.03 (0.85 to 1.24)	0.92 (0.61 to 1.38)		19.4 (17.5 to 21.3)	46.5 (43.1 to 50.0)	61.8 (54.5 to 69.1)
7–7.9	reference				20.0 (17.9 to 22.2)	45.8 (43.1 to 48.6)	63.6 (57.4 to 69.8)
8–8.9	0.98 (0.81 to 1.18)	1.01 (0.84 to 1.21)	1.02 (0.69 to 1.50)		19.7 (17.6 to 21.8)	46.0 (42.7 to 49.4)	64.0 (57.6 to 70.5)
≥9	0.85 (0.67 to 1.08)	1.11 (0.83 to 1.48)	1.38 (0.83 to 2.31)		17.7 (14.7 to 20.8)	48.3 (42.2 to 54.4)	70.3 (62.0 to 78.6)
Insomnia, nights/month							
No	reference			.0071	19.3 (17.9 to 20.7)	44.2 (42.3 to 46.1)	61.3 (56.7 to 65.8)
Rare	1.34 (0.94 to 1.92)	1.59 (1.16 to 2.18)	1.67 (0.92 to 3.06)		23.9 (18.4 to 29.5)	55.0 (47.8 to 62.3)	72.0 (61.5 to 82.5)
Occasional	1.20 (0.92 to 1.56)	1.58 (1.19 to 2.09)	2.37 (1.20 to 4.71)		22.1 (17.9 to 26.2)	54.9 (48.6 to 61.2)	78.2 (67.5 to 89.0)
Frequent	1.11 (0.80 to 1.54)	2.05 (1.53 to 2.74)	0.74 (0.38 to 1.41)		20.8 (15.7 to 25.9)	60.8 (54.2 to 67.5)	54.3 (40.8 to 67.8)
Snoring frequency							
Nonsnorer	reference			.0021	17.6 (16.2 to 19.0)	44.6 (42.4 to 46.8)	63.6 (59.5 to 67.6)
Moderate snorer	1.44 (1.21 to 1.71)	1.08 (0.90 to 1.30)	0.76 (0.49 to 1.18)		23.2 (20.2 to 26.2)	46.5 (42.9 to 50.1)	57.4 (47.7 to 67.2)
Habitual snorer	1.87 (1.56 to 2.24)	1.30 (1.09 to 1.54)	1.16 (0.77 to 1.74)		28.0 (24.9 to 31.0)	50.7 (46.8 to 54.6)	66.7 (58.7 to 74.6)
Daytime sleepiness							
Unlikely	reference			.865	19.7 (18.3 to 21.0)	46.3 (44.5 to 48.1)	63.1 (59.4 to 66.8)
Average	0.84 (0.54 to 1.31)	1.05 (0.77 to 1.43)	0.85 (0.43 to 1.68)		17.3 (11.2 to 23.3)	47.4 (40.4 to 54.4)	59.5 (44.5 to 74.4)
Excessive	1.04 (0.64 to 1.70)	0.86 (0.56 to 1.30)	1.02 (0.39 to 2.66)		20.3 (13.3 to 27.4)	42.7 (33.0 to 52.5)	63.6 (42.0 to 85.1)

Abbreviation: CI, confidence interval.
[a]Raw numbers, *N* = 16 244 as 43 observations who reported "don't know" for snoring were dropped from the analysis.
[b]Adjusted for age, gender, city, wealth index, education, body mass index and other sleep variables.
[c]Covariates used: age, gender, city, education, wealth index, body mass index and other sleep variables.
[d]Wald test for effect modification of age group.

were stronger in middle and older age groups implying a significant interaction between age group and insomnia. And, the snoring and hypertension association were stronger in younger age groups (Table 3). The association of sleep variables and hypertension were similar in both genders (Table 4). We found no effect modification of BMI on sleep variables and hypertension relationship in the complete case analysis (Supplementary Table 3). We also found no significant interaction by insomnia for sleep duration and hypertension nor by snoring for EDS and hypertension (data not shown).

In analyses using continuous SBP and DBP as outcomes, moderate and habitual snoring were significantly associated with higher SBP and DBP, even after adjusting for hypertension treatment. The associations were highly attenuated but remained significant after adjusting for other variables. We found no relationships between duration of sleep, insomnia, and EDS with either SBP or DBP (Table 5).

In sensitivity analyses, we found that the complete case and MI data sets did not differ in any of the demographic, socioeconomic, behavioral, exposure, or outcome parameters (see Supplementary Table 1). The estimates from logistic and linear regressions were comparable between two data sets (see Supplementary Table 2).

DISCUSSION

In this representative adult population from three mega cities of South Asia, nearly a third of the population had either moderate or habitual snoring, but only about 5% had daytime sleepiness. The percentage of adults sleeping longer than optimum hours were much higher than shorter duration of sleep. The principal findings in this large study of sleep and hypertension in South Asia was that self-reported snoring was associated with hypertension among urban adults. This association was independent of prominent risk factors for hypertension. Further, we noted significant associations across snoring categories with hypertension and linear associations with blood pressure. The associations were stronger in younger individuals and women.

The current study estimates are similar to the findings from a large cross-sectional study of 10 413 adults (50–85 years) from Guangzhou in China, where daily snorers had higher

Table 4—Logistic regression models[a] of association between sleep variables and hypertension stratified by gender (N = 16 244[b])[c].

Sleep Characteristics	Adjusted odds ratios (95% CI)			Predicted probabilities of hypertension % (95% CI) [d]	
	Men	Women	p [e]	Men	Women
Duration of sleep, hours					
<5	0.94 (0.71 to 1.24)	1.06 (0.83 to 1.34)	.9277	32.6 (27.6 to 37.6)	27.6 (23.8 to 31.4)
5–5.9	0.97 (0.61 to 1.55)	1.03 (0.70 to 1.51)		33.2 (23.8 to 42.6)	27.2 (21.1 to 33.2)
6–6.9	0.98 (0.84 to 1.15)	0.97 (0.80 to 1.16)		33.4 (31.1 to 35.7)	26.1 (23.8 to 28.4)
7–7.9	reference			33.7 (31.0 to 36.5)	26.7 (24.6 to 28.7)
8–8.9	0.99 (0.83 to 1.16)	1.04 (0.84 to 1.29)		33.5 (30.9 to 36.0)	27.4 (24.8 to 30.0)
≥9	0.95 (0.74 to 1.22)	1.09 (0.86 to 1.37)		32.7 (28.5 to 36.9)	28.0 (24.4 to 31.7)
Insomnia, nights/month					
No	reference		.4649	33.1 (31.3 to 35.0)	25.7 (24.4 to 27.0)
Rare	1.19 (0.84 to 1.67)	1.52 (1.13 to 2.04)		36.3 (30.1 to 42.6)	32.9 (27.8 to 38.1)
Occasional	1.23 (0.89 to 1.70)	1.50 (1.20 to 1.87)		37.1 (31.0 to 43.2)	32.7 (28.8 to 36.7)
Frequent	1.17 (0.82 to 1.67)	1.44 (1.11 to 1.86)		36.0 (29.0 to 43.1)	32.0 (27.2 to 36.7)
Snoring frequency					
Nonsnorer	reference		.2090	31.9 (30.0 to 33.8)	25.4 (23.9 to 26.8)
Moderate snorer	1.07 (0.91 to 1.25)	1.33 (1.10 to 1.61)		33.1 (29.9 to 36.3)	30.3 (27.3 to 33.2)
Habitual snorer	1.43 (1.19 to 1.71)	1.50 (1.27 to 1.78)		38.8 (35.4 to 42.1)	32.4 (29.4 to 35.4)
Daytime sleepiness					
Unlikely	reference		.1500	33.5 (31.8 to 35.3)	27.0 (25.7 to 28.3)
Average	0.97 (0.67 to 1.39)	0.86 (0.61 to 1.21)		32.9 (25.9 to 40.0)	24.6 (19.5 to 29.7)
Excessive	0.68 (0.42 to 1.10)	1.22 (0.83 to 1.81)		26.8 (18.9 to 34.8)	30.5 (23.7 to 37.2)

Abbreviation: CI, confidence interval.
[a]Adjusted for age, gender, city, wealth index, education, body mass index and other sleep variables.
[b]Raw numbers, N = 16 244 as 43 observations who reported "don't know" for snoring were dropped from the analysis.
[c]Estimates are in odds ratio (95% confidence interval).
[d]Covariates used: age, gender, city, education, wealth index, body mass index and other sleep variables.
[e]Wald test for effect modification of gender.

Table 5—Linear regression models of association between sleep variables with systolic and diastolic blood pressures[a] (*N* = 16 244[b]).

Sleep Characteristics	Systolic blood pressure, mm Hg (95%CI)		Diastolic blood pressure, mm Hg (95%CI)	
	Model 1[c]	Model 2[d]	Model 1[c]	Model 2[d]
Duration of sleep, hours				
<5	0.84 (−0.76 to 2.44)	−0.30 (−1.77 to 0.16)	−0.46 (−1.45 to 0.54)	−0.52 (−1.45 to 0.42)
5–5.9	−0.94 (−3.30 to 1.43)	−0.68 (−2.72 to 1.36)	0.07 (−1.39 to 1.52)	0.14 (−1.24 to 1.53)
6–6.9	0.94 (−0.02 to 1.90)	−0.01 (−0.89 to 0.87)	0.22 (−0.39 to 0.83)	−0.09 (−0.66 to 0.49)
7–7.9	reference			
8–8.9	−0.40 (−1.39 to 0.59)	0.51 (−0.32 to 1.34)	−0.55 (−1.18 to 0.09)	0.00 (−0.57 to 0.57)
≥9	−2.15 (−3.43 to −0.86)	0.69 (−0.33 to 1.71)	−1.65 (−2.42 to −0.88)	−0.13 (−0.81 to 0.54)
Insomnia, nights/month				
No	reference			
Rare	−0.77 (−2.50 to 0.95)	−0.50 (−2.15 to 1.16)	−0.03 (−1.05 to 0.99)	0.10 (−0.91 to 1.12)
Occasional	−0.71 (−2.63 to 1.21)	0.38 (−1.41 to 2.16)	−0.45 (−1.58 to 0.67)	0.35 (−0.76 to 1.46)
Frequent	0.48 (−1.36 to 2.31)	0.56 (−1.12 to 2.25)	0.05 (−1.14 to 1.24)	0.47 (−0.68 to 1.62)
Snoring frequency				
Nonsnorer	reference			
Moderate snorer	5.21 (4.21 to 6.21)	1.04 (0.08 to 2.00)	3.78 (3.18, 4.38)	1.39 (0.78, 1.99)
Habitual snorer	7.90 (6.74 to 9.06)	2.34 (1.31 to 3.36)	5.14 (4.49 to 5.79)	1.97 (1.31 to 2.62)
Daytime sleepiness				
Unlikely	reference			
Average	1.18 (−0.72 to 3.08)	−0.49 (−2.27 to 1.30)	1.00 (−0.23 to 2.24)	−0.14 (−1.38 to 1.10)
Excessive	2.16 (−0.26 to 4.58)	−0.34 (−2.46 to 1.78)	0.98 (−0.48 to 2.44)	−0.31 (−1.70 to 1.08)

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure.
[a]Estimates are blood pressure in mm Hg.
[b]Raw numbers, 16,244 as 43 observations who reported "don't know" for snoring were dropped from the analysis.
[c]Model 1: Adjusted for hypertension treatment.
[d]Model 2: adjusted for hypertension treatment, age, gender, city, wealth index, education, body mass index and other sleep variables.

odds of hypertension 1.37 (1.20–1.56) compared to nonsnorers.[34] Similarly, a 10-year follow-up study of 2451 Swedish men showed persistent snoring was independently associated with incident hypertension after adjusting for other risk factors among younger men (30–49 years at baseline) but not among older men (50–69 years at baseline).[16] However, in a population-based survey of 6779 Swedish women, snoring was associated with hypertension only in presence of daytime sleepiness,[15] while a smaller study among Hispanics Americans found no association between snoring and hypertension.[35] This indicates that there may be heterogeneity in snoring and hypertension relationships across populations. Nevertheless, a meta-analysis of longitudinal studies has confirmed that snoring is a risk factor for hypertension.[10]

While the direction of associations cannot be established in this cross-sectional study, the coexistence of snoring and hypertension has clinical implications. Although snoring is a symptom of OSA,[13] not all snorers may have OSA. Indeed, the relationships between snoring and cardiometabolic disorders could be independent of OSA.[16] The relationships between OSA and hypertension may also be bidirectional.[14] Untreated coexistence of OSA may lead to uncontrolled hypertension, despite medical treatment for hypertension.[36] Conversely, treatment of hypertension to a lower BP target may improve sleep apnea by improving upper airway tone.[14] Therefore, screening for snoring and OSA and appropriate treatment among people with hypertension specifically among young adults and vice versa may help control both conditions.

The percentage of people reporting EDS in South Asian adults was small (4%). A smaller study from Chennai[20] which used snoring, tiredness during daytime, observed apnea, and high blood pressure (STOP) questionnaire[37] found 59% of adults had daytime sleepiness. However, the ESS instrument used in our study is more robust measure, as it uses a comprehensive score and also was found to have higher specificity and positive predictive values compared to STOP.[38] We found no associations between EDS and hypertension in South Asian cities. This is unsurprising as previous studies showed EDS was associated with hypertension only in persons with OSA.[8]

The present analysis found that having insomnia, irrespective of the frequency, was associated with about 40% higher odds of hypertension. These results contrast with a cross-sectional

analysis of symptoms of insomnia and hypertension in 12 643 adults from the US' National Health And Nutrition Examinations Surveys (NHANES) which found no such association.[11] However, our findings are consistent with a meta-analysis of prospective studies that showed insomnia was positively associated with hypertension.[10] Another study from Finland, following employees with insomnia assessed during baseline, found that odds of the use of antihypertensive medication was higher by 40% and 47% among persons with occasional and frequent insomnia, respectively, at the baseline.[39] The definition of frequent insomnia as defined in our study is in line with definition of insomnia disorder, that is, insomnia symptoms for at least 3 days a week.[9] We found the odds of hypertension in this group was similar to odds for rare or occasional insomnia. But, among the individual symptoms of insomnia, only medication used for insomnia remained significantly associated with hypertension indicating higher hypertension in severe insomnia. Disruptions in hypothamic–pituitary–adrenal axis and circadian rhythmicity,[40] sympathetic activation, oxidative stress, systemic inflammation, and hypoxemia[40,41] are possible mechanisms on how insomnia might be related to elevated blood pressure. The cross-sectional analysis and lack of dose–response relationship makes it difficult to say definitively and to investigate the root causes of the relationships between insomnia and hypertension in our analysis.

We found no association between either short or long duration of sleep with hypertension after adjusting for other hypertension risk factors among South Asian adults. The Sleep Heart Health Study from the United States, suggested a "U"-shaped relationship between duration of sleep and hypertension.[4] However, there is huge variation in duration of sleep and hypertension relationships by gender,[42] age groups,[43,44] and geographical regions.[2] Further exploration of the social context of sleep loss and the role of stress and other psychological factors in its occurrence may help explain these study and regional differences. Indeed, more open-ended and qualitative perspectives may be helpful in seeking the underlying causes of poor sleep patterns.

This study has several limitations that should be considered. First, this was a cross-sectional study, and therefore directions of association between sleep variables and snoring cannot be ascertained, and there is a potential for reverse causality.[10] Second, self-reported sleep variables, specifically snoring, are subject to recall and information bias and probably best asked of the participant's partner. Validation of sleep questions against roommate/partner's report in the United States found moderate to high correlation[45]; however, no such validation studies exists in South Asia, where cultural norms may influence participant responses differently to the United States. Nevertheless, any measurement error in exposure variables is likely to be nondifferential (unlikely to be affected by hypertension status). If anything, this would possibly had pulled the associations towards the null, and therefore the true relationships may have been underestimated rather than overestimated. Additionally, due to lack of objective measurements, it was not possible to differentiate between primary or simple snoring and snoring as marker of obstructive sleep apnea.[46] Third, the models were not adjusted for time of blood pressure measurement. Since time of blood pressure measurement varied between the participants (between 7 AM and 4 PM), this may have nondifferentially affected the sleep–blood pressure relationship. In future, ambulatory blood pressure measurements may be useful to further explain this relationship. Finally, the analysis was not adjusted for many other potential confounders such as physical activity, depression, and intake of caffeine or medications that are associated with poor sleep such as aspirin, nonsteroidal anti-inflammatory drugs, and hormones. There may also be residual confounding by these variables which may have distorted the study results. However, the analysis was adjusted for the variables commonly included in publications examining associations between sleep and hypertension, and the associations reported above remained after adjustment for these variables.

The study also has several strengths. This was first study in South Asia reporting on the association between sleep factors and hypertension using large representative populations of three mega cities with such a high response rate. Second, the study used standardized protocol across all sites with stringent quality assurance and quality control. Third, blood pressure, BMI, and biochemistry were all objectively measured. Fourth, the study reported on association between sleep variables with both hypertension and continuous measures of BP. Finally, we considered a large variety of potential confounders in order to minimize residual confounding.

CONCLUSION

Moderate and habitual snoring was highly prevalent and was positively associated with hypertension, specifically in young adults and women in South Asian metropolitan cities. Any level of insomnia was also associated with higher odds of hypertension. Duration of sleep and daytime sleepiness were not associated with hypertension. Association of sleep variables with hypertension in this population needs further exploration through robust longitudinal studies in this region.

REFERENCES

1. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: current epidemiology and future directions. Circulation. 2016; 133(16): 1605–1620.
2. Gangwisch JE. A review of evidence for the link between sleep duration and hypertension. Am J Hypertens. 2014; 27(10): 1235–1242.
3. Dunai A, Keszei AP, Kopp MS, Shapiro CM, Mucsi I, Novak M. Cardiovascular disease and health-care utilization in snorers: a population survey. Sleep. 2008; 31(3): 411–416.
4. Gottlieb DJ, Redline S, Nieto FJ, et al. Association of usual sleep duration with hypertension: the Sleep Heart Health Study. Sleep. 2006; 29(8): 1009–1014.
5. Magee CA, Kritharides L, Attia J, McElduff P, Banks E. Short and long sleep duration are associated with prevalent cardiovascular disease in Australian adults. J Sleep Res. 2012; 21(4): 441–447.
6. Fernandez-Mendoza J, Vgontzas AN, Liao D, et al. Insomnia with objective short sleep duration and incident hypertension: the Penn State Cohort. Hypertension. 2012; 60(4): 929–935.
7. Norton PG, Dunn EV. Snoring as a risk factor for disease: an epidemiological survey. Br Med J (Clin Res Ed). 1985; 291(6496): 630–632.
8. Wang Q, Zhang C, Jia P, et al. The association between the phenotype of excessive daytime sleepiness and blood pressure in patients with obstructive sleep apnea-hypopnea syndrome. Int J Med Sci. 2014; 11(7): 713–720.
9. Roth T. Insomnia: definition, prevalence, etiology, and consequences. J Clin Sleep Med. 2007; 3(5 Suppl): S7–10.
10. Meng L, Zheng Y, Hui R. The relationship of sleep duration and insomnia to risk of hypertension incidence: a meta-analysis of prospective cohort studies. Hypertens Res. 2013; 36(11): 985–995.
11. Vozoris NT. Insomnia symptom frequency and hypertension risk: a population-based study. J Clin Psychiatry. 2014; 75(6): 616–623.

12. Phillips B, Bůzková P, Enright P; Cardiovascular Health Study Research Group. Insomnia did not predict incident hypertension in older adults in the cardiovascular health study. Sleep. 2009; 32(1): 65–72.
13. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. Sleep. 1999; 22(5): 667–689.
14. Jhamb M, Unruh M. Bidirectional relationship of hypertension with obstructive sleep apnea. Curr Opin Pulm Med. 2014; 20(6): 558–564.
15. Lindberg E, Berne C, Franklin KA, Svensson M, Janson C. Snoring and daytime sleepiness as risk factors for hypertension and diabetes in women—a population-based study. Respir Med. 2007; 101(6): 1283–1290.
16. Lindberg E, Janson C, Gislason T, Svärdsudd K, Hetta J, Boman G. Snoring and hypertension: a 10 year follow-up. Eur Respir J. 1998; 11(4): 884–889.
17. Kim J, Yi H, Shin KR, Kim JH, Jung KH, Shin C. Snoring as an independent risk factor for hypertension in the nonobese population: the Korean Health and Genome Study. Am J Hypertens. 2007; 20(8): 819–824.
18. Kapur VK, Resnick HE, Gottlieb DJ; Sleep Heart Health Study Group. Sleep disordered breathing and hypertension: does self-reported sleepiness modify the association? Sleep. 2008; 31(8): 1127–1132.
19. Stranges S, Tigbe W, Gómez-Olivé FX, Thorogood M, Kandala NB. Sleep problems: an emerging global epidemic? Findings from the INDEPTH WHO-SAGE study among more than 40,000 older adults from 8 countries across Africa and Asia. Sleep. 2012; 35(8): 1173–1181.
20. Roopa M, Deepa M, Indulekha K, Mohan V. Prevalence of sleep abnormalities and their association with metabolic syndrome among Asian Indians: Chennai Urban Rural Epidemiology Study (CURES-67). J Diabetes Sci Technol. 2010; 4(6): 1524–1531.
21. Dodani S, Mistry R, Khwaja A, Farooqi M, Qureshi R, Kazmi K. Prevalence and awareness of risk factors and behaviours of coronary heart disease in an urban population of Karachi, the largest city of Pakistan: a community survey. J Public Health (Oxf). 2004; 26(3): 245–249.
22. Devi P, Rao M, Sigamani A, et al. Prevalence, risk factors and awareness of hypertension in India: a systematic review. J Hum Hypertens. 2013; 27(5): 281–287.
23. World Health Organization. Global status report on noncommunicable diseases. Geneva, Switzerland: World Health Organization; 2014.
24. Nair M, Ali MK, Ajay VS, et al. CARRS Surveillance study: design and methods to assess burdens from multiple perspectives. BMC Public Health. 2012; 12: 701.
25. Ali MK, Bhaskarapillai B, Shivashankar R, et al.; CARRS investigators. Socioeconomic status and cardiovascular risk in urban South Asia: The CARRS Study. Eur J Prev Cardiol. 2016; 23(4): 408–419.
26. World Health Organization. STEPS Manual17th Feb 2015. Available from: http://www.who.int/chp/steps/manual/en/index3.html Accessed February 14, 2017.
27. Chobanian AV, Bakris GL, Black HR, et al.; National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA. 2003; 289(19): 2560–2572.
28. Quan SF, Howard BV, Iber C, et al. The Sleep Heart Health Study: design, rationale, and methods. Sleep. 1997; 20(12): 1077–1085.
29. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991; 14(6): 540–545.
30. Bhatia M, Prasad K, Pande R. Hindi version of epworth sleepiness scale: a validity study. Indian J Sleep Med. 2006; 1(4): 171–174.
31. Young T, Shahar E, Nieto FJ, et al.; Sleep Heart Health Study Research Group. Predictors of sleep-disordered breathing in community-dwelling adults: the Sleep Heart Health Study. Arch Intern Med. 2002; 162(8): 893–900.
32. Anand S, Shivashankar R, Ali MK, et al.; CARRS Investigators. Prevalence of chronic kidney disease in two major Indian cities and projections for associated cardiovascular disease. Kidney Int. 2015; 88(1): 178–185.
33. Berg CJ, Ajay VS, Ali MK, et al. A cross-sectional study of the prevalence and correlates of tobacco use in Chennai, Delhi, and Karachi: data from the CARRS study. BMC Public Health. 2015; 15: 483.
34. Thomas GN, Jiang CQ, Lao XQ, et al. Snoring and vascular risk factors and disease in a low-risk Chinese population: the Guangzhou Biobank Cohort Study. Sleep. 2006; 29(7): 896–900.
35. Schmidt-Nowara WW, Coultas DB, Wiggins C, Skipper BE, Samet JM. Snoring in a Hispanic-American population. Risk factors and association with hypertension and other morbidity. Arch Intern Med. 1990; 150(3): 597–601.
36. Walia HK, Li H, Rueschman M, et al. Association of severe obstructive sleep apnea and elevated blood pressure despite antihypertensive medication use. J Clin Sleep Med. 2014; 10(8): 835–843.
37. Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology. 2008; 108(5): 812–821.
38. El-Sayed IH. Comparison of four sleep questionnaires for screening obstructive sleep apnea. Egypt J Chest Dis Tuberc. 2012; 61(4): 433–441.
39. Haaramo P, Rahkonen O, Hublin C, Laatikainen T, Lahelma E, Lallukka T. Insomnia symptoms and subsequent cardiovascular medication: a register-linked follow-up study among middle-aged employees. J Sleep Res. 2014; 23(3): 281–289.
40. Committee on Sleep Medicine and Research. Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem. Washington, DC: The National Academy Press; 2006.
41. Grandner MA, Perlis ML. Short sleep duration and insomnia associated with hypertension incidence. Hypertens Res. 2013; 36(11): 932–933.
42. Cappuccio FP, Stranges S, Kandala NB, et al. Gender-specific associations of short sleep duration with prevalent and incident hypertension: the Whitehall II Study. Hypertension. 2007; 50(4): 693–700.
43. Gangwisch JE, Feskanich D, Malaspina D, Shen S, Forman JP. Sleep duration and risk for hypertension in women: results from the nurses' health study. Am J Hypertens. 2013; 26(7): 903–911.
44. Kim J, Jo I. Age-dependent association between sleep duration and hypertension in the adult Korean population. Am J Hypertens. 2010; 23(12): 1286–1291.
45. Kump K, Whalen C, Tishler PV, et al. Assessment of the validity and utility of a sleep-symptom questionnaire. Am J Respir Crit Care Med. 1994; 150(3): 735–741.
46. Amorós-Sebastiá LI. Radiofrequency treatment in simple snoring: tolerance, safety and results. Acta Otorrinolaringologica (English Edition). 2011; 62(4): 300–305.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *SLEEP* online.

FUNDING

The CARRS Study was funded by the National Heart, Lung, and Blood Institute of the National Institutes of Health, Department of Health and Human Services (contract no. HHSN268200900026C) and the United Health Group (Minneapolis, MN, USA). The first author (RS) was supported by a Wellcome Trust Capacity Strengthening Strategic Award Extension phase to the Public Health Foundation of India and a consortium of UK universities (WT084754/Z/08/A).

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication February, 2017
Submitted in final revised form June, 2017
Accepted for publication July, 2017
Address correspondence to: Roopa Shivashankar, Public Health Foundation of India (PHFI), Head, CVD Epidemiology Unit, Centre for Chronic Disease Control (CCDC), Plot no 47, Sector 44, Gurgaon, Haryana – 122002, India. Telephone: 91–124 – 4781400 (ext: 4419); Fax: 91-124-4722901; Email: roopa@ccdcindia.org; roopa.shivashankar@phfi.org

INSTITUTION WHERE WORK WAS PERFORMED

Public Health Foundation of India (PHFI), Plot no 47, Sector 44, Gurgaon, Haryana – 122002.

CONFLICT OF INTEREST

None disclosed.