Original Article

Short sleep duration and increased risk of hypertension: a primary care medicine investigation

Brice Faraut[a], Évelyne Touchette[a,b], Harvey Gamble[c], Sylvie Royant-Parola[d], Michel E. Safar[e], Brigitte Varsat[c], and Damien Léger[a]

See editorial comment on page 1313

Objectives: Compelling evidence from laboratory-based and population-based studies link sleep loss to negative cardiovascular health outcomes. However, little is known about the association between sleep duration and hypertension in primary care health settings, independently of other well controlled clinical and biochemical characteristics. We investigated the association between sleep duration and the prevalence of hypertension adjusting for 21 potential confounding factors in a noncontrolled primary care sample.

Methods: The sample included 1046 French adults older than 40 years (mean age, 55.5 years), who visited any of the general practitioners of primary care centers in the Paris area. Blood pressure (BP) readings, blood samples and standardized health and sleep questionnaires were performed on each participant. Hypertension inclusion criteria were either high BP measurements (SBP $\geq$140 mmHg or DBP $\geq$90 mmHg) or the use of antihypertensive medications. Sleep duration was recorded as the self-reported average number of hours of sleep per night during the week. Logistic regressions were performed to test the association between hypertension and sleep duration adjusted for sociodemographic, clinical, biochemical, lifestyle, psychological and sleep disorder covariates.

Results: Compared to the group sleeping 7 h, individuals sleeping 5 h or less had an increased odds ratio (OR) for the prevalence of hypertension [OR = 1.80, 95% confidence interval (1.06–3.05)], after adjusting for 21 potential confounders which did not markedly attenuate this association.

Conclusion: Our data provide further epidemiologic evidence that with no specific selection in primary care medicine, usual short-sleep duration increases the risk of hypertension prevalence in adults over 40 years.

Keywords: hypertension prevalence, primary care medicine, short-sleep duration

Abbreviations: BP, blood pressure; HAD, hospital anxiety and depression; HDL-c, high-density lipoprotein cholesterol; HPA, hypothalamic-pituitary-adrenal; LDL-c, low-density lipoprotein cholesterol; REM, rapid eye movement; SWS, slow-wave sleep

INTRODUCTION

Short-sleep duration is commonly defined as reporting less than 6 h sleep per 24 h [1]. A meta-analysis in 2006 observed the prevalence of short sleepers in the United States from 1975 to 2006 and concluded that over the 31-year period there was an increased odds ratio (OR) in the prevalence of short sleepers among full-time workers compared to the rest of the sample. Accordingly, the 2009 National Sleep Foundation survey reported that the percentage of the population sleeping less than 6 h per night on working days has almost doubled over the last 10 years, increasing from 12% in 1998 to 20% in 2009 [2]. These data suggest an emerging trend of reduced sleep duration especially on weekdays leading to a growing sleep debt among the general population in western countries.

Epidemiological studies have shown that chronic exposure to sleep restriction could have gradual and cumulative negative health effects and increase cardiovascular risk (obesity, diabetes, atherosclerosis, and hypertension) [3–7]. Elevated blood pressure (BP) is a major risk factor for cardiovascular diseases, for example coronary heart diseases, heart failure, stroke mortality [8]. A report indicated that the age-adjusted and sex-adjusted prevalence of hypertension was 44% in European countries and 28% in the North American countries using the 140/90 mmHg threshold [9]. During nocturnal sleep, there is a decrease of 10–20% in BP mean ('nocturnal dipping') compared with the daytime mean. Sleep is a major component of circadian BP variations with lowest BP during slow-wave sleep (SWS) and relatively higher BP during light and rapid eye movement (REM) sleep [10]. This suggests that processes such as changes in sleep duration could affect the 24-h BP profile and potentially alter cardiovascular events. In a 5-year

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[a] Centre du sommeil et de la vigilance, APHP, Université Paris Descartes, Hôtel-Dieu de Paris, Sorbonne Paris Cité, [b]INSERM U669, Université Paris Descartes, Université Paris-Sud-XI, [c]Caisse Primaire d'assurance maladie, Centres d'examens de santé, Paris, [d]Réseau MORPHEE, Garches and [e]Unité hypertension artérielle, Prévention et Thérapeutique Cardiovasculaire, Hôtel-Dieu de Paris, Université Paris Descartes, Sorbonne Paris Cité, APHP, Paris, France

Correspondence to Professor Damien Leger, Centre du sommeil et de la vigilance, Hôtel-Dieu de Paris, Sorbonne Paris Cité, APHP, Paris, France. Tel: +33 1 42 34 86 26; fax: +33 1 42 34 82 27; e-mail: damien.leger@htd.aphp.fr

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longitudinal study assessing sleep by actigraphy and BP in 578 adults (35–45 years old), shorter sleep duration and lower sleep maintenance predicted significantly higher SBP and DBP levels cross–sectionally as well as further adverse changes in SBP and DBP levels over 5 years [11]. Short-sleep duration also seems to be a strong predictor for future cardiovascular events in patients with hypertension [12]. Most studies performed to date on the associations between sleep duration and hypertension were based on self-reported sleep duration [4,7,12,13]. It is not considered realistic to obtain more accurate and objective measures of sleep in large prospective population-based studies, even more so in primary care as investigations were performed during health promotion medical examinations.

To our knowledge, no published study has adjusted for confounders when researching the link between subjective sleep duration and hypertension in the primary care health setting independently of other well controlled clinical and biochemical characteristics. The aim of this study was to test the association between sleep duration and hypertension adjusting for 21 potential confounding factors in a non-controlled primary care sample.

METHODS

Participants

The sample included 1046 French adults, aged 40–71 years who consecutively visited any of the general practitioners of Paris' primary care centers [Caisse Primaire d'Assurance Maladies (CPAM) de Paris]. It was planned to include a total of 1000 participants and the study finally stopped recruiting after the inclusion of the 1046th patient. Participants filled out self-administered standardized health questionnaires and sleep questionnaires, which were written based on the International Classification of Sleep Disorders (ICSD-2) and Diagnostic and statistical Manual of Mental disorders (DSM-IV) definitions of sleep disorders [14,15]. Adults involved were asked by their general practitioner or came independently based on their ability to have a free of charge preventive health check-up. BP readings and blood samples were performed for each participant. Participants who visited the general practitioners at the CPAM were informed that they would have a subsequent visit including complete medical examination, blood samples, and eventually radiographs. They did know they would have to complete questionnaires. Doctors were made aware that all items of the questionnaires had to be completed and followed a common check list file for examination. Consecutively, no individual refused to participate to the study and all 1046 first individuals who visited during the study period were included.

Ethics

This epidemiological study was approved by the ethical committee of the CPAM Paris. All data were anonymously analysed according to the ethical rules and authorization of the Commission Nationale Informatique et Libertés. Patients were informed and signed approval to be included in the study. Patients and their general practitioners (those authorized) received individual reports including all personal medical data.

Hypertension

The principal-dependent variable for this study was the presence of hypertension. Three BP readings were performed using a mercury gauge sphygmomanometer during the same appointment according to the 2005 Haute Autorité de Santé guidelines [16]. The mean of the three BP measurements taken at 5-min intervals was recorded (SBP and DBP). Individuals who had SBP readings 140 mmHg or higher or DBP readings 90 mmHg or higher at the time of their visit in primary care center, or who had been prescribed or used antihypertensive medications were included in the hypertensive group.

Sleep questionnaire

The major independent variable for this study was sleep duration. The individuals' average sleep duration in hours was recorded from their answer to the question: 'How many hours of sleep do you usually get a night during weekdays?' Sleep duration was categorized ($\geq$5 h, 6 h, 7 h, 8 h and $\geq$9 h). Sleep disorders (insomnia, sleep apnea, periodic leg movements) were assessed with recognized instruments previously used in epidemiological settings and based on the ICSD-2 and DSM-IV definitions of sleep disorders [14,15]. Subjective sleepiness was assessed using the Epworth Sleepiness Scale [17].

Sociodemographic, lifestyle variables and clinical characteristics

Demographic characteristics (age, sex), working status (yes/no) and lifestyle factors such as smoking status (number of cigarettes per day), alcohol consumption (number of alcohol drink per days), physical activity level and the social vulnerability (EPICES Score >30) were examined. Briefly, the EPICES scores for 'Evaluation de la Précarité et des Inégalités de santé dans les Centres d'Examens de Santé' (Evaluation of Precarity and Inequalities in Health Examination Centers) was calculated on the basis of individual conditions of deprivation (traditional socioeconomic indicators and questions related to family structure, housing conditions, employment, social benefits, financial difficulties, leisure activities, social support, childhood/adult life events, self-perceived health, and healthcare utilization) [18]. Medical diagnosis associated with cardiovascular risk included obesity (BMI >30), diabetes or dyslipidemia diagnoses.

Biochemical blood measurements

Lipids [total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), triglycerides] were measured using a Beckman Coulter (Olympus) Auto*Mate* (Beckman Coulter, Fullerton, California, USA). Leukocyte counts and subsets were determined on a Beckman Coulter LH750 haemocytometer (Beckman Coulter). Borderline high cutoff levels were used for cholesterol level, HDL-c and LDL-c. Cholesterol level more than 6.18 mmol/l, LDL-c less than 4.13 mmol/l, HDL-c 1.56 mmol/l or less and triglycerides 1.70 mmol/l or more and increased leukocyte counts (mainly neutrophils) were considered as markers of increased cardiovascular risk [19–21].

Depression and anxiety

We used the standard cutoff score of 10 out of a total score of 21 on the 14-question Hospital Anxiety and Depression (HAD) Scale to measure the symptoms of depression (even numbers) and anxiety (odd numbers) [22]. Of the 64 participants who reported taking antidepressants, 52 scored in the nondepressed range on the HAD scale. However, we included these individuals in the depressed category.

Statistical analysis

Data were analysed using SPSS version 15.0 software (IBM, Somers, New York, USA). χ^2 tests were used to test the association between hypertension and categorical potentially confounding variables (Tables 1 and 2). Student's *t*-tests (Table 1) or ANOVAs (Table 2) were used to verify associations between hypertension and continuous potentially confounding variables. Logistic regressions were performed to test the associations between hypertension and usual sleep duration adjusted for multiple confounding relevant variables (Table 3). A probability level of P less than 0.05 was considered as statistically significant.

RESULTS

The baseline characteristics of hypertensive and normotensive individuals are summarized in Table 1. Hypertension was diagnosed in 42% of the individuals examined during a health promotion medical examination using the criteria of BP measurements or current antihypertensive treatment. Among the hypertensive individuals, 34.5% were on antihypertensive medication, 28.5% were detected as having both elevated SBP and DBP (SBP higher than 140 mmHg and DBP higher than 90 mmHg), 32.9% were detected as only having raised SBP and 4.1% only had an elevated DBP. Within the patients taking antihypertensive medications, BP measurements indicated that 34% had hypertension with raised SBP and DBP, 23% had elevated SBP alone 0.02% had elevated DBP alone. When excluding the 151 patients who were taking antihypertensive medications, mean ± SD DBP and SBP (mmHg) in the different sleep category durations were respectively 82.2 ± 10.1 and 135.4 ± 18.6 (≤5-h sleep); 78.6 ± 10.9 and 129.9 ± 18.8 (6-h sleep); 78.4 ± 11.2 and 130.1 ± 20.1 (7-h sleep); 80.2 ± 10.2 and 132.1 ± 18.3 (8-h sleep); 78.6 ± 8.5 and 129.2 ± 15.4 (≥9-h sleep). The differences in the mean values among the five sleep duration categories were significant for DBP ($P = 0.008$) and just under significance for SBP ($P = 0.06$). The shorter 5-h sleep duration category was associated both to higher DBP and SBP when compared to the 7-h reference sleep duration category ($P = 0.001$ and $P = 0.01$ respectively). Hypertension was associated with male sex, increased age, nonworking status, cardiovascular risk factors (obesity, dyslipidemia and diabetes), higher social vulnerability, larger cigarette and alcohol consumptions and lower sleepiness levels. Hypertension was also found to be associated to several blood markers linked to increased cardiovascular risk, that is increased triglycerides (≥1.70 mmol/l) and leukocyte counts (mainly neutrophils and monocytes) and reduced HDL-c (≤1.56 mmol/l). Depression and anxiety were not significantly associated with hypertension.

Figure 1 shows the proportion of hypertensive patients in each category of sleep duration per night. Shorter sleep durations were associated with a higher prevalence of hypertension as shown by the presence of hypertension in over half of the individuals reporting sleeping less than 5 h/night.

Table 2 indicates the characteristics of individuals according to nocturnal sleep duration categories. The most represented sleep duration category was 7 h/night which corresponds to 33.1% of the participants. Sleep duration of less than 7 h/night was reported by 34.9% of individuals of which 14.3% sleeping less than 6 h/night. A proportion of 32% of the individuals reported sleeping 8 h/night or more of which 7.4% of the individuals were sleeping 9 h or more per night. Shorter sleep durations were associated with a significant higher prevalence of hypertension but not with other cardiovascular comorbidities, that is obesity, dyslipidemia or diabetes ($P = 0.02$). No significant differences in biomarker levels measured among different sleep duration categories were found. Individuals with the longer hours of sleep per night were older with a higher proportion of nonworkers. Extreme sleep durations and especially those sleeping less than 5 h/night were more likely to have depressive and anxiety symptoms and higher social vulnerability. The shorter sleep durations were also associated with insomnia, higher levels of sleepiness, sleep apnea and periodic leg movements.

Logistic regressions were performed to test the associations between hypertension and usual sleep duration progressively adjusted for potential confounders (Table 3). The individuals that slept 5 h or less per night were significantly more likely to be hypertensive [OR 1.75 (1.18–2.59), 95% confidence interval (CI), $P = 0.005$; unadjusted model 1]. The sleep duration category 5 h or less per night remained significantly associated with hypertension after adjusting for confounding variables: sociodemographic status [OR 1.84 (1.21–2.78), 95% CI, $P = 0.004$; model 2]; obesity, dyslipidemia and diabetes [OR 1.75 (1.14–2.69), 95% CI, $P = 0.01$; model 3]; blood lipids and leukocytes [OR 1.77 (1.12–2.82), 95% CI, $P = 0.01$, model 4]; alcohol and cigarette consumption, physical activity and social vulnerability [OR 1.68 (1.04–2.70), 95% CI, $P = 0.03$; model 5]; depression and anxiety [OR 1.65 (1.02–2.70), 95% CI, $P = 0.04$; model 6] and sleep disorders such as insomnia, sleepiness, sleep apnea or periodic leg movements [OR 1.80 (1.06–3.05), 95% CI, $P = 0.03$; model 7]. Thus, after adjusting for potential confounding factors, the shorter sleep duration (≤5 h per night) remains significantly associated with hypertension with no difference in the OR value. Being a man, older characteristics, a high social vulnerability status, obesity and diabetes were also linked to significant higher adjusted ORs of hypertension prevalence.

DISCUSSION

Our data provide further epidemiologic evidence that usually short-sleep duration is associated to increased hypertension prevalence in adults over 40 assessed with no specific selection in primary care health centres. A gradient of increasing proportion of hypertensive patients along with the reduction in nocturnal sleep duration was

TABLE 1. Baseline characteristics of the individuals

Characteristic	Prevalence *n*% 1046	Hypertension[b] Yes *n*% 438 (42.0)	Hypertension[b] No *n*% 605 (57.8)	*P*
Nocturnal sleep duration				
Average per week				
≤5 h	147 (14.3)	75 (17.5)	72 (12.1)	
6 h	211 (20.6)	94 (22.0)	117 (19.6)	
7 h (reference)	338 (33.0)	126 (29.4)	212 (35.5)	0.02
8 h	253 (24.7)	108 (25.2)	145 (24.3)	
≥9 h	76 (7.4)	25 (5.8)	51 (8.5)	
Demographic variables				
Sex				
Women	460 (44.1)	155 (35.4)	305 (50.4)	<0.001
Men	583 (55.9)	283 (64.6)	300 (49.6)	
Age in years[a]	1043 (99.7)	57.98 ± 7.36	53.72 ± 7.70	<0.001
Working status				
Yes	552 (53.2)	201 (46.0)	351 (58.4)	<0.001
No	486 (46.8)	236 (54.0)	250 (41.6)	
Clinical characteristics				
Obesity (BMI ≥30 kg/m^2)				
Yes	124 (11.9)	86 (19.7)	38 (6.3)	<0.001
No	917 (88.1)	350 (80.3)	567 (93.7)	
Dyslipidemia				
Yes	133 (12.8)	83 (18.9)	50 (8.3)	<0.001
No	910 (87.2)	355 (81.1)	555 (91.7)	
Diabetes				
Yes	27 (2.6)	24 (5.5)	3 (0.5)	<0.001
No	1016 (97.4)	414 (94.5)	602 (99.5)	
Biochemical features				
Leukocytes counts[a]	1029 (98.4)	6.61 ± 1.66	6.38 ± 1.68	0.03
Neutrophils	1029 (98.4)	3.85 ± 1.43	3.69 ± 1.46	0.09
Eosinophils	1029 (98.4)	0.18 ± 0.12	0.17 ± 0.14	0.87
Basophils	1029 (98.4)	0.04 ± 0.02	0.04 ± 0.02	0.53
Lymphocytes	1029 (98.4)	2.01 ± 0.56	1.97 ± 0.56	0.20
Monocytes	1029 (98.4)	0.56 ± 0.17	0.52 ± 0.17	<0.001
Cholesterol (>6.18 mmol/l)				
Yes	343 (35.7)	149 (36.8)	194 (35.0)	0.56
No	617 (64.3)	256 (63.2)	361 (65.0)	
Low-density lipoprotein (>4.13 mmol/l)				
Yes	258 (27.5)	117 (30.2)	141 (25.7)	0.13
No	679 (72.5)	271 (69.8)	408 (74.3)	
High-density lipoprotein (≤1.56 mmol/l)				
Yes	501 (52.2)	232 (57.3)	269 (48.5)	0.007
No	459 (47.8)	173 (42.7)	286 (51.5)	
Triglycerides (≥1.7 mmol/l)				
Yes	187 (19.6)	104 (26.0)	83 (15.0)	<0.001
No	766 (80.4)	296 (74.0)	470 (85.0)	
Lifestyle variables				
Vulnerable condition (Epices score <30)				
Yes	305 (30.6)	144 (34.5)	161 (27.7)	0.02
No	693 (69.4)	273 (65.5)	420 (72.3)	
Cigarettes per day[a]	1043 (99.7)	8.10 ± 11.93	6.30 ± 9.67	0.009
Alcohol drinks per day[a]	1043 (99.7)	0.86 ± 1.46	0.61 ± 1.35	0.004
Physical activity level				
Sedentary	323 (31.0)	146 (33.3)	177 (29.3)	0.16
Regular to intense	720 (66.7)	292 (66.7)	428 (70.7)	
Psychological characteristics				
Depression (HAD >10)				
Yes	75 (7.2)	35 (8.0)	40 (6.6)	0.40
No	968 (92.8)	403 (92.0)	565 (93.4)	
Anxiety (HAD >10)				
Yes	231 (22.1)	93 (21.2)	138 (22.8)	0.55
No	812 (77.9)	345 (78.8)	467 (77.2)	
Sleep disorders				
Insomnia				
Yes	262 (25.1)	118 (26.9)	144 (23.8)	0.25
No	781 (74.9)	320 (73.0)	461 (76.2)	

TABLE 1 (*Continued*)

Characteristic	Prevalence *n*% 1046	Hypertension[b] Yes *n*% 438 (42.0)	Hypertension[b] No *n*% 605 (57.8)	*P*
Sleepiness (Epworth ≥10)				
Yes	267 (26.2)	96 (22.4)	171 (29.0)	0.02
No	751 (73.8)	333 (77.6)	418 (71.0)	
Sleep apnea				
Yes	149 (14.3)	62 (14.2)	87 (14.4)	0.92
No	894 (85.7)	376 (85.8)	518 (85.6)	
Periodic leg movements				
Yes	58 (5.6)	24 (5.5)	34 (5.6)	0.92
No	985 (94.4)	414 (94.5)	571 (94.4)	

[a]Means continuous variables.
[b]Hypertension was defined as a SBP of 140 mmHg or higher, a DBP of 90 mmHg or higher or the use of antihypertensive medications.

observed. Longitudinal studies indicate that cardiovascular events (e.g. coronary heart diseases and heart failure events) increase proportionately to BP increase [23]. Chronic sleep restriction and associated elevated BP could contribute to predispose to cardiovascular pathogenesis. Accordingly, two recent meta-analyses indicate that sleep deprivation is associated with an increased risk of cardiovascular events [24,25].

The prevalence of hypertension, diabetes and obesity and sex distribution reported in this sample was similar to the prevalences classically observed among the same aged general population of France [26–28]. The same ranges of resistant hypertension were found in the sample as in the general population (20–30%) [29]. Classical cardiovascular risk cofactors associated to a higher prevalence of hypertension, that is male sex, older age, obesity, being diagnosed for diabetes and dyslipidemia, increased blood triglycerides 1.70 mmol/l or more and reduced blood HDL-c 1.56 mmol/l or less, higher cigarette and alcohol consumptions were reported and accounted for. We also found that hypertension was found to be linked to higher social vulnerability and lower sleepiness. Chronic stressful and undesirable life events are common features of higher social vulnerability that may precede and precipitate the development of raised BP. A chronic activation of the stress pathway and excess release of mediators such as cortisol and catecholamines are often observed during chronic sleep restriction and may contribute to elevated BP. Higher stress levels could also be an explanation for the reduced sleepiness levels observed in hypertensive patients.

Data from a previous population-based study indicated that patients who suffered from depression were more likely to be diagnosed with hypertension [30]. In contrast, other longitudinal population-based studies do not support a role for depression or anxiety symptoms in the development of hypertension and report that these could predict lower BP [31,32]. However, numerous potential confounding factors were not always well controlled such as age and sex effects on BP, the use of different instruments to measure anxiety and depression or the effects of antidepressants on BP [33]. In our study, however, depression and anxiety scores were not associated with a significantly reduced or increased hypertension prevalence. This had potential limitations as a large proportion of the individuals included in the depressed category were taking antidepressant medications or the use of different depression scales made strict comparisons with other studies on the topic difficult. Notwithstanding the complex interrelations between depression and hypertension, sleep duration could act as a mediator of the relationship between depression and hypertension incidence in middle-aged adults as recently reported in a multivariate longitudinal study [12]. We also found that participants with short (≤5 h) and long-sleep (≥9 h) durations were more likely to have depressive and anxious symptoms, higher social vulnerability and displayed higher hypertension prevalence. Our results are consistent with several analyses studying sleep duration and depression, which report a U-shaped association with increased depressive symptoms in both extremes of sleep duration [34].

The mean age of individuals in this study was 55.5 years, a range of age for which short-sleep duration has been found to be associated to hypertension, which was not the case for older individuals [4]. The shorter sleep durations (≤5 and 6 h) were significantly associated with insomnia, higher sleepiness, sleep apnea and periodic leg movements in agreement with previous reports [4,7]. Short-sleep durations were not associated with any significant change in lipid profile as previously reported in middle-aged adults of both sex [35].

Reduced night-time sleep durations can promote risk factors for cardiovascular diseases such as obesity (stronger risk in children and young adults and reduced proportionately to age) and diabetes (higher risk in men) [3,5,36,37].

In multivariable models, the association between short-sleep duration and BP outcomes remain robust after adjusting for numerous potential confounders including sleep apnea, insomnia, sleepiness, depression or cardiovascular risk status. As expected, male sex, older age, obesity and diabetes were linked to significantly higher adjusted ORs for hypertension prevalence. However, diabetes and obesity slightly attenuated the relationships between individuals who reported getting 5 h or less of sleep and hypertension prevalence. This suggests that the association between reduced sleep duration and hypertension is only partially mediated through obesity or diabetes and that the

TABLE 2. Baseline characteristics of individuals and nocturnal sleep durations

		Nocturnal sleep duration					
		≤5 h	6 h	7 h	8 h	≥9 h	
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	
Characteristic	**1046**	**147 (14.3)**	**211 (20.6)**	**340 (33.1)**	**253 (24.6)**	**76 (7.4)**	***P***
Hypertension[a]							
Yes	428 (41.8)	75 (51.0)	94 (44.5)	126 (37.3)	108 (42.7)	25 (32.9)	0.02
No	597 (58.2)	72 (49.0)	117 (55.5)	212 (62.7)	145 (57.3)	51 (67.1)	
Demographic variables							
Sex							
Women	454 (44.2)	67 (45.6)	102 (48.1)	140 (41.2)	106 (41.9)	39 (51.3)	0.31
Men	574 (55.8)	80 (54.4)	110 (51.9)	200 (58.8)	147 (58.1)	37 (48.7)	
Age in years[c]	1028 (98.3)	55.24 ± 7.91	55.24 ± 7.91	54.43 ± 7.87	56.65 ± 7.82	58.22 ± 7.24	0.001
Working status							
Yes	546 (53.3)	77 (52.7)	126 (59.7)	202 (59.4)	117 (46.2)	24 (32.4)	0.001
No	478 (46.7)	69 (47.3)	85 (40.3)	138 (40.6)	136 (53.8)	50 (67.6)	
Clinical characteristics							
Obesity (BMI ≥30 kg/m^2)							
Yes	121 (11.8)	19 (15.7)	31 (14.6)	34 (10.1)	23 (9.1)	14 (18.4)	0.10
No	905 (88.2)	128 (87.1)	181 (85.4)	304 (89.9)	230 (90.9)	62 (81.6)	
Lipidemy							
Yes	127 (12.4)	21 (14.3)	29 (13.7)	33 (9.7)	34 (13.4)	10 (13.2)	0.50
No	901 (87.6)	126 (85.7)	183 (86.3)	307 (90.3)	219 (86.6)	66 (86.8)	
Diabetes[b]							
Yes	26 (2.5)	7 (4.8)	6 (2.8)	7 (2.1)	3 (1.2)	3 (3.9)	0.21
No	1016 (97.5)	140 (95.2)	206 (97.2)	33 (97.9)	250 (98.8)	73 (96.1)	
Biochemical features							
Leukocytes counts[c]	1011 (96.7)	6.62 ± 1.80	6.22 ± 1.41	6.43 ± 1.71	6.61 ± 1.76	6.46 ± 1.56	0.10
Neutrophils		3.82 ± 1.55	3.56 ± 1.13	3.71 ± 1.49	3.87 ± 1.55	3.88 ± 1.38	0.19
Eosinophils		0.17 ± ± 0.10	0.17 ± 0.16	0.17 ± 0.11	0.18 ± 0.14	0.17 ± 0.12	0.76
Basophils		0.04 ± 0.02	0.04 ± 0.02	0.04 ± 0.02	0.04 ± 0.02	0.03 ± 0.01	0.05
Lymphocytes		2.07 ± 0.61	1.93 ± 0.51	2.00 ± 0.58	1.99 ± 0.55	1.88 ± 0.50	0.07
Monocytes		0.54 ± 0.17	0.53 ± 0.16	0.53 ± 0.17	0.55 ± 0.19	0.51 ± 0.14	0.27
Cholesterol (>6.18 mmol/l)							
Yes	338 (35.7)	46 (33.3)	64 (32.2)	112 (36.7)	92 (38.8)	24 (35.8)	0.63
No	608 (64.3)	92 (66.7)	135 (67.8)	193 (63.3)	145 (61.2)	43 (64.2)	
Low-density lipoprotein (>4.13 mmol/l)							
Yes	255 (27.6)	32 (24.6)	55 (28.1)	89 (29.7)	62 (26.7)	17 (25.4)	0.83
No	670 (72.4)	98 (75.4)	141 (71.9)	211 (70.3)	170 (73.3)	50 (74.6)	
High-density lipoprotein (<1.56 mmol/l)							
Yes	494 (52.2)	77 (55.8)	102 (51.3)	158 (51.8)	123 (51.9)	34 (50.7)	0.93
No	452 (47.8)	61 (44.2)	97 (48.7)	147 (48.2)	114 (48.1)	33 (49.3)	
Triglycerides (>1.70 mmol/l)							
Yes	185 (19.7)	33 (24.3)	39 (19.6)	53 (17.5)	48 (20.4)	12 (17.9)	0.57
No	755 (80.3)	103 (75.7)	160 (80.4)	250 (82.5)	187 (79.6)	55 (82.1)	
Lifestyle variables							
Vulnerable condition (Epices score <30)							
Yes	295 (29.9)	55 (38.7)	66 (32.0)	83 (25.5)	65 (27.3)	26 (35.6)	0.03
No	690 (70.1)	87 (61.3)	140 (68.0)	243 (74.5)	173 (72.7)	47 (64.4)	
Cigarettes per day[c]	1028 (98.3)	8.01 ± 12.69	6.67 ± 9.79	6.63 ± 10.42	7.78 ± 11.406	68 ± 8.38	0.53
Alcohol drinks per day[c]	1028 (98.3)	0.76 ± 1.61	0.61 ± 1.18	0.66 ± 1.09	0.83 ± 1.79	0.68 ± 1.18	0.47
Physical activity level							
Sedentary	316 (30.7)	48 (32.7)	73 (34.4)	95 (27.9)	71 (28.1)	29 (38.2)	0.23
Regular to intense	712 (69.3)	99 (67.3)	139 (65.6)	245 (72.1)	182 (71.9)	47 (61.8)	
Psychological characteristics							
Depression (HAD >10)							
Yes	73 (7.1)	25 (17.0)	19 (9.0)	9 (2.6)	11 (4.3)	9 (11.8)	<0.001
No	955 (92.9)	122 (83.0)	193 (91.0)	331 (97.4)	242 (95.7)	67 (88.2)	
Anxiety (HAD >10)							
Yes	227 (22.1)	60 (40.8)	48 (22.6)	59 (17.4)	43 (17.0)	17 (22.4)	<0.001
No	801 (77.9)	87 (59.2)	164 (77.4)	281 (82.6)	210 (83.0)	59 (77.6)	
Sleep disorders							
Insomnia							
Yes	257 (25.0)	97 (66.0)	65 (30.7)	45 (13.2)	37 (14.6)	13 (17.1)	<0.001
No	771 (75.0)	50 (34.0)	147 (69.3)	295 (86.8)	216 (85.4)	63 (82.9)	
Sleepiness (Epworth ≥10)							
Yes	261 (26.0)	55 (38.5)	70 (33.7)	75 (22.5)	47 (19.0)	14 (19.7)	<0.001
No	742 (74.0)	88 (61.5)	138 (66.3)	259 (77.5)	200 (81.0)	57 (80.3	

TABLE 2 (*Continued*)

Characteristic	*n* (%)	Nocturnal sleep duration ≤5 h	6 h	7 h	8 h	≥9 h	*P*
	1046	*n* (%) 147 (14.3)	*n* (%) 211 (20.6)	*n* (%) 340 (33.1)	*n* (%) 253 (24.6)	*n* (%) 76 (7.4)	
Sleep apnea							
Yes	148 (14.4)	41 (27.9)	34 (16.0)	34 (10.0)	31 (12.3)	8 (10.5)	<0.001
No	880 (85.6)	106 (72.1)	178 (84.0)	306 (90.0)	222 (87.7)	68 (89.5)	
Periodic leg movements[d]							
Yes	58 (5.6)	22 (15.0)	12 (5.7)	13 (3.8)	9 (3.6)	2 (2.6)	<0.001
No	970 (94.4)	125 (85.0)	200 (94.3)	327 (96.2)	244 (96.4)	74 (97.4)	

[a]Hypertension was defined as a SBP of140 mmHg or higher, a DBP of 90 mmHg or higher, or the use of antihypertensive medications.
[b]Two cells (25.0%) have expected count less than 5. The minimum expected count is 1.92.
[c]Continuous variables.
[d]One cell (12.5%) has expected count less than 5. The minimum expected count is 4.29.

effects of short-sleep on hypertension may be mediated by the lack of sleep *per se*. Short-sleep duration may be a factor through which insomnia promotes the development of hypertension, as insomnia with objective short-sleep, recorded by polysomnography (≤6 h per night) appears to be the most significant predictor of hypertension [38]. Here, the association of short-sleep duration with hypertension remained significant after adjustment for insomnia suggesting that voluntary sleep restriction may predispose to hypertension development. Some studies indicated that long-duration sleepers were more likely to be diagnosed with hypertension [7,39]. Here, we did not report consistent associations between the longest sleep duration (9 h), which was the smallest subgroup and displayed the lowest proportion of hypertensive patients.

No association between short-sleep length and BP were found in the elderly, and short-sleep durations in women but not in men were linked to higher hypertension incidence in some publications [13,40–42]. These data suggest both an age and sex effect on the relationship between sleep and hypertension and a stronger association in middle-aged individuals, although it remains to be further documented.

A methodological limitation of the current study is the measure of sleep quantity that was recorded with self-reported sleep duration per night. However, although subjective sleep duration were used in the majority of the previous studies, consistent findings were reported for a higher prevalence of hypertension in short sleepers, which was confirmed with objective sleep length measures [11,38]. Assessment of sleep duration in a primary healthcare setting on a large unselected adult population requires a fast and simple method. Self-administered questionnaires, although not as accurate as time-consuming actigraphy or polysomnography (the gold standard reference), provide a useful measure of sleep duration. Although a single measure could not accurately represent sleep duration over time, individual variation year-to-year showed a low variability for sleep duration, that is a yearly SD of 0.39 h for sleep duration in actigraphically recorded middle-aged adults [43]. In summary, further objective measures of sleep duration per 24 h are required to assess the relationship between sleep length, quality and hypertension.

A second limitation is that the detection of hypertension in individuals not taking antihypertensive medications was identified with daily BP measurements. Although the mean of three consecutive BP measurements was recorded and used, an unknown environment could influence BP readings in individuals who are more vulnerable to anxiety such as in the short sleeper category. Sleep is also an important and constant source of circadian BP variation and reduced nocturnal dipping of BP is considered as a predictor for negative cardiovascular outcomes [44]. A study examined the relationship between self-reported and 24-h ambulatory BP monitoring and reported that a 1-h reduction in sleep duration was associated with nondipping status (nocturnal BP fall) without an elevated morning BP surge [45]. Interestingly, long-sleep duration was associated with a morning BP surge and less nondipping status. Polysomnography investigations coupled to 24-h ambulatory BP monitoring are now needed in order to consider circadian variations and the effect of sleep deprivation on BP and to better characterize sleep stages-dependant BP alterations in short sleepers.

Notwithstanding the previous limitations, we confirmed the association between short-sleep duration and hypertension in unselected adults in primary care medicine. Primary care enabled us to control for 21 well recognized clinical and biochemical potential confounding factors, which is almost impossible to get in the general population. Regardless of these numerous adjustments, a strength of the present study resulted from a robust association between hypertension and short-sleep duration. The ORs were not majorly altered after adjustment for 21 covariates including potential mediators of the association between curtailed sleep and hypertension, for example obesity, diabetes, sleep apnea, insomnia.

The mechanism(s) that underlie the association between short-sleep duration and elevated BP are not yet fully understood. Activation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system are common characteristics of sleep debt condition and are potentially involved in higher BP and cardiovascular pathogenesis [46,47]. Several pathways have been proposed to explain the link between chronic activation of HPA axis and hypertension, for example cortisol-mediated inhibition of cholinergic vasodilatation and enhanced mineralocorticoid activity [48,49].

In summary, cross–sectional analyses support a relationship between insufficient sleep and elevated BP with a strong variation according to sex and age. Despite this, the

TABLE 3. Odds ratio (95% confidence interval) for hypertension, as a function of different models

Variable	Model 1[a]	P	Model 2[b]	P	Model 3[c]	P	Model 4[d]	P	Model 5[e]	P	Model 6[f]	P	Model 7[g]	P
Nocturnal sleep duration														
<5 h	1.75 (1.18–2.59)	0.005	1.84 (1.21–2.78)	0.004	1.75 (1.14–2.69)	0.01	1.77 (1.12–2.82)	0.01	1.68 (1.04–2.70)	0.03	1.65 (1.02–2.70)	0.04	180 (1.06–3.05)	0.03
6 h	1.35 (0.95–1.91)	0.09	1.40 (0.97–2.03)	0.06	1.30 (0.89–1.91)	0.17	1.38 (0.92–2.08)	0.12	1.38 (0.90–2.11)	0.13	1.38 (0.91–2.11)	0.13	1.54 (0.99–2.39)	0.05
7 h (reference)	1.00		1.00		1.00		1.00		1.00		1.00		1.00	
8 h	1.25 (0.89–1.74)	0.18	1.07 (0.75–1.53)	0.68	1.12 (0.78–1.62)	0.51	1.19 (0.81–1.76)	0.38	1.27 (0.85–1.91)	0.24	1.28 (0.85–1.92)	0.23	1.31 (0.87–1.98)	0.19
≥9 h	0.82 (0.48–1.39)	0.47	0.67 (0.38–1.18)	0.17	0.58 (0.32–1.05)	0.07	0.69 (0.37–1.31)	0.26	0.65 (0.34–1.26)	0.21	0.66 (0.34–1.28)	0.22	0.76 (0.39–1.48)	0.42
Demographic variables														
Men/women			1.88 (1.44–2.46)	<0.001	1.77 (1.34–2.3)	<0.001	1.79 (1.30–2.47)	<0.001	1.82 (1.28–2.59)	<0.001	1.86 (1.30–2.65)	<0.001	2.17 (1.49–3.16)	<0.001
Age in years[h]			1.08 (1.06–1.11)	<0.001	1.08 (1.05–1.10)	<0.001	1.09 (1.06–1.12)	<0.001	1.10 (1.07–1.13)	<0.001	1.10 (1.07–1.13)	<0.001 1	10 (1.06–1.12)	<0.001
Working status/no			0.94 (0.69–1.27)	0.69	0.91 (0.66–1.25)	0.56	0.93 (0.67–1.31)	0.69	1.10 (1.07–1.12)	0.52	0.89 (0.62–1.26)	0.52	0.91 (0.63–1.31)	0.61
Clinical characteristics														
Obesity (BMI ≥30)/no					4.03 (2.58–6.30)	<0.001	3.58 (2.20–5.83)	<0.001	3.23 (1.95–5.33)	<0.001	3.22 (1.95–5.33)	<0.001	3.46 (2.06–5.82)	<0.001
Dyslipidemia/no					1.46 (0.96–2.24)	0.07	1.37 (0.85–2.19)	0.19	1.40 (0.85–2.28)	0.17	1.40 (0.85–2.28)	0.19	1.39 (0.84–2.31)	0.19
Diabetes/no					5.53 (1.57–19.50)	0.007	4.64 (1.29–16.73)	0.02	6.64 (1.42–31.20)	0.01	6.64 (1.42–31.21)	0.01	5.46 (1.14–26.05)	0.03
Biochemical features														
Leucocytes counts[h]							1.04 (0.95–1.13)	0.39	1.02 (0.93–1.12)	0.63	1.02 (0.93–1.12)	0.65	1.03 (0.93–1.13)	0.55
Cholesterol (>6.18 mmol/l)/no							0.86 (0.54–1.39)	0.55	0.88 (0.54–1.44)	0.61	0.88 (0.54–1.43)	0.60	2.34 (1.28–4.27)	0.005
Low-density lipoprotein (>4.13 mmol/l)/no							1.47 (0.91–2.40)	0.11	1.56 (0.95–2.59)	0.08	1.57 (0.95–2.60)	0.08	0.44 (0.23–0.84)	0.01
High-density lipoprotein (<1.56 mmol/l)/no							0.90 (0.63–1.39)	0.55	0.90 (0.63–1.29)	0.57	0.90 (0.63–1.29)	0.57	1.38 (0.88–2.18)	0.15
Triglycerides (>1.70 mmol/l)/no							1.45 (0.97–2.16)	0.06	1.41 (0.94–2.11)	0.10	1.41 (0.94–2.13)	0.09	0.96 (0.60–1.55)	0.88
Lifestyle variables														
Social vulnerability/no									1.63 (1.15–2.31)	0.005	1.64 (1.52–2.34)	0.006	1.67 (1.17–2.40)	0.005
Number of cigarettes per day[h]									1.00 (0.99–1.02)	0.75	1.00 (0.98–1.02)	0.75	1.00 (0.99–1.02)	0.64
Number of alcohol drinks per day[h]									1.01 (0.91–1.12)	0.84	1.00 (0.90–1.12)	0.86	1.00 (0.89–1.12)	0.97
Sedentary/regular to intense physical activity									1.27 (0.91–1.80)	0.15	1.28 (0.91–1.81)	0.09	1.35 (0.95–1.91)	0.09
Psychological characteristics														
Depression/no											0.89 (0.48–1.65)	0.72	0.83 (0.43–1.62)	0.59
Anxiety/no											1.13 (0.76–1.67)	0.53	1.18 (0.78–1.80)	0.42
Sleep disorders														
Insomnia/no													1.18 (0.78–1.80)	0.75
Sleepiness/no													0.73 (0.49–1.11)	0.14
Sleep apnea/no													0.75 (0.45–1.25)	0.27
Perdiodic leg movements/no													1.28 (0.61–2.69)	0.50

[a]Model 1, unadjusted.
[b]Model 2, adjusted for demographic variables.
[c]Model 3, adjusted for demographic variables and clinical characteristics.
[d]Model 4, adjusted for demographic variables, clinical characteristics and biochemical features.
[e]Model 5, adjusted for demographic variables, clinical characteristics, biochemical features and lifestyle variables.
[f]Model 6, adjusted for characteristics, biochemical features, lifestyle variables and psychological characteristics.
[g]Model 7, adjusted for demographic variables, clinical characteristics, biochemical features, lifestyle demographic variables, clinical variables, psychological characteristics and sleep disorders.
[h]Continuous variables.
⊥Hypertension was defined as a SBP of 140 mmHg or higher, a DBP of 90 mmHg or higher, or the use of antihypertensive medications.

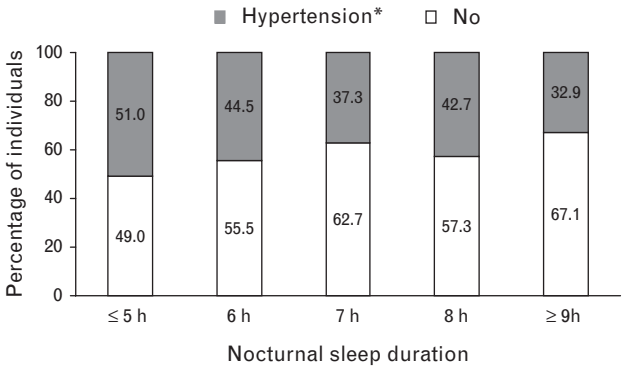

FIGURE 1 Nocturnal sleep duration and the prevalence of hypertension. Proportions of adults with hypertension are represented in grey according to reported nocturnal sleep duration categories. *Hypertension was defined as a SBP of 140 mmHg or higher, a DBP of 90 mmHg or higher, or the use of antihypertensive medications.

pathophysiological pathway(s) involved remain to be elucidated. The coexistence of other cardiovascular risk factors is known to add to the risk associated of BP elevation. Individuals in midlife reporting an average sleep duration of 5 h or less at night should be considered as an additional higher risk group for adverse cardiovascular events.

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Conflicts of interest

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REFERENCES

1. Knutson KL, Van Cauter E, Rathouz PJ, DeLeire T, Lauderdale DS. Trends in the prevalence of short sleepers in the USA: 1975–2006. *Sleep* 2010; 33:37–45.
2. National Sleep Foundation. Sleep in America Poll, 2009. Available from: <http://www.sleepfoundation.org/sites/default/files/2009%20Sleep%20in%20America%20SOF%20EMBARGOED.pdf>.
3. Capuccio FP, D'Elia L, Strazullo P, Miller LA. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. *Diabet Care* 2010; 33:414–420.
4. Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, *et al*. Short sleep duration as a risk factor for hypertension: analyses of the first National Health and Nutrition Examination Survey. *Hypertension* 2006; 47:833–839.
5. Singh M, Drake CL, Roehrs T, Hudgel DW, Roth T. The association between obesity and short sleep duration: a population-based study. *J Clin Sleep Med* 2005; 1:357–363.
6. King CR, Knutson KL, Rathouz PJ, Sidney S, Liu K, Lauderdale DS. Short sleep duration and incident coronary artery calcification. *JAMA* 2008; 300:2859–2866.
7. Gottlieb DJ, Redline S, Nieto FJ, Baldwin CM, Newman AB, Resnick HE, *et al*. Association of usual sleep duration with hypertension: the Sleep Heart Health Study. *Sleep* 2006; 29:1009–1014.
8. Safar ME, Blacher J, Jankowski P. Arterial stiffness, pulse pressure, and cardiovascular disease: is it possible to break the vicious circle? *Atherosclerosis* 2011. [Epub ahead of print].
9. Wolf-Maier K, Cooper RS, Banegas JR, Giampaoli S, Hense HW, Joffres M, *et al*. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. *JAMA* 2003; 289:2363–2369.
10. Smolensky MH, Hermida RC, Castriotta RJ, Portaluppi F. Role of sleep-wake cycle on blood pressure circadian rhythms and hypertension. *Sleep Med* 2007; 8:668–680.
11. Knutson KL, Van Cauter E, Rathouz PJ, Yan LL, Hulley SB, Liu K, *et al*. Association between sleep and blood pressure in midlife: the CARDIA sleep study. *Arch Intern Med* 2009; 169:1055–1061.
12. Gangwisch JE, Malaspina D, Posner K, Babiss LA, Heymsfield SB, Turner JB, *et al*. Insomnia and sleep duration as mediators of the relationship between depression and hypertension incidence. *Am J Hypertens* 2010; 23:62–69.
13. Cappuccio FP, Stranges S, Kandala NB, Miller MA, Taggart FM, Kumari M, *et al*. Gender-specific associations of short sleep duration with prevalent and incident hypertension: the Whitehall II Study. *Hypertension* 2007; 50:693–700.
14. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4th ed. *Text revision (DSM-IV-TR)*. Arlington, VA, USA: American Psychiatric Association; 2000.
15. American Academy of Sleep Medicine. *International classification of sleep disorders*, 2nd ed. *Diagnostic and coding manual*. Wetchester, Illinois, USA: American Academy of Sleep Medicine; 2005. p. 297.
16. Haute Autorité de Santé (HAS). Prise en charge des patients adultes atteints d'hypertension artérielle essentielle, actualisation 2005. [French guidelines on hypertension prevention in clinical practice 2005.] http://www.hassante.fr/portail/upload/docs/application/pdf/HTA_2005_recos.pdf. [Accessed 7 May 2012].
17. Leger D, Annesi-Maesano I, Carat F, Rugina M, Pribil C, Chanal I, *et al*. Allergic rhinitis and its consequences on quality of sleep; an unexplored area. *Arch Intern Med* 2006; 166:1744–1748.
18. Bihan H, Silvana L, Sass C, Nguyen G, Huot C, Moulin JJ, *et al*. Association between individual deprivation, glycemic control and diabetic complication-The EPICES score. *Diabet Care* 2005; 28:2680–2685.
19. European guidelines on cardiovascular disease prevention in clinical practice: executive summary. ESC Committee for Practice Guidelines. *Atherosclerosis* 2007; 194:1–45.
20. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the national cholesterol education program expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. *JAMA* 2001; 285:2486–2497.
21. Ruggiero C, Metter EJ, Cherubini A, Maggio M, Sen R, Najjar SS, *et al*. White blood cell count and mortality in the baltimore longitudinal study of aging. *J Am Coll Cardiol* 2007; 49:1841–1850.
22. Snaith RP, Zigmond AS. The hospital anxiety and depression scale. *Br Med J (Clin Res Ed)*. 1986; 292:344.
23. Haider AW, Larson MG, Franklin SS, Levy D. Systolic blood pressure, diastolic blood pressure, and pulse pressure as predictors of risk for congestive heart failure in the Framingham Heart Study. *Ann Intern Med* 2003; 138:10–16.
24. Gallicchio L, Kalesan B. Sleep duration and mortality: a systematic review and meta-analysis. *J Sleep Res* 2009; 18:148–158.
25. Cappuccio FP, Cooper D, D'Elia L, Strazzullo P, Miller MA. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *Eur Heart J* 2011; 32:1484–1492.
26. Wagner A, Sadoun A, Dallongeville J, Ferrières J, Amouyel P, Ruidavets JB, *et al*. High blood pressure prevalence and control in a middle-aged French population and their associated factors: the MONA LISA study. *J Hypertens* 2011; 29:43–50.
27. Kusnik-Joinville O, Weill A, Salanave B, Ricordeau P, Allemand H. Prevalence and treatment of diabetes in France: trends between 2000 and 2005. *Diabetes Metab* 2008; 34:266–272.
28. Charles MA, Eschwège E, Basdevant A. Monitoring the obesity epidemic in France: the Obepi surveys 1997–2006. *Obesity* 2008; 16:2182–2186.
29. Calhoun DA, Jones D, Textor S, Goff DC, Murphy TP, Toto RD, *et al*. Resistant hypertension: diagnosis, evaluation, and treatment. *Circulation* 2008; 117:e510–e526.

30. Patten SB, Williams JV, Lavorato DH, Campbell NR, Eliasziw M, Campbell TS. Major depression as a risk factor for high blood pressure: epidemiologic evidence from a national longitudinal study. *Psychosom Med* 2009; 71:273–279.
31. Delaney JA, Oddson BE, Kramer H, Shea S, Psaty BM, McClelland RL. Baseline depressive symptoms are not associated with clinically important levels of incident hypertension during two years of follow-up: the multiethnic study of atherosclerosis. *Hypertension* 2010; 55:408–414.
32. Hildrum B, Romild U, Holmen J. Anxiety and depression lowers blood pressure: 22-year follow-up of the population based HUNT study, Norway. *BMC Public Health* 2011; 11:601.
33. Licht CM, de Geus EJ, Seldenrijk A, van Hout HP, Zitman FG, van Dyck R, Penninx BW. Depression is associated with decreased blood pressure, but antidepressant use increases the risk for hypertension. *Hypertension* 2009; 53:631–638.
34. Krueger PM, Friedman EM. Sleep duration in the United States: a crosssectional population-based study. *Am J Epidemiol* 2009; 169:1052–1063.
35. Miller MA, Kandala NB, Kivimaki M, Kumari M, Brunner EJ, Lowe GD, *et al*. Gender differences in the cross-sectional relationships between sleep duration and markers of inflammation: Whitehall II study. *Sleep* 2009; 32:857–864.
36. Hasler G, Buysse DJ, Klaghofer R, Gamma A, Ajdacic V, Eich D, *et al*. The association between short sleep duration and obesity in young adults: a 13-year prospective study. *Sleep* 2004; 27:661–666.
37. Ayas NT, White DP, Al-Delaimy WK, Manson JE, Stampfer MJ, Speizer FE, *et al*. A prospective study of self-reported sleep duration and incident diabetes in women. *Diabet Care* 2003; 26:380–384.
38. Vgontzas AN, Liao D, Bixler EO, Chrousos GP, Vela-Bueno A. Insomnia with objective short sleep duration is associated with a high risk for hypertension. *Sleep* 2009; 32:491–497.
39. Buxton OM, Marcelli E. Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States. *Soc Sci Med* 2010; 71:1027–1036.
40. van den Berg JF, Tulen JH, Neven AK, Hofman A, Miedema HM, Witteman JC, *et al*. Sleep duration and hypertension are not associated in the elderly. *Hypertension* 2007; 50:585–589.
41. Lima-Costa MF, Peixoto SV, Rocha FL. Usual sleep duration is not associated with hypertension in Brazilian elderly: the Bambui Health Aging Study (BHAS). *Sleep Med* 2008; 9:806–807.
42. Stranges S, Dorn JM, Cappuccio FP, Donahue RP, Rafalson LB, Hovey KM, *et al*. A population-based study of reduced sleep duration and hypertension: the strongest association may be in premenopausal women. *J Hypertens* 2010; 28:896–902.
43. Knutson KL, Rathouz PJ, Yan LL, Liu K, Lauderdale DS. Intra-individual daily and yearly variability in actigraphically recorded sleep measures: the CARDIA study. *Sleep* 2007; 30:793–796.
44. Hansen TW, Li Y, Boggia J, Thijs L, Richart T, Staessen JA. Predictive role of the nighttime blood pressure. *Hypertension* 2011; 57:3–10.
45. Friedman O, Shukla Y, Logan AG. Relationship between self-reported sleep duration and changes in circadian blood pressure. *Am J Hypertens* 2009; 22:1205–1211.
46. Faraut B, Boudjeltia KZ, Vanhamme L, Kerkhofs M. Immune, inflammatory and cardiovascular consequences of sleep restriction and recovery. *Sleep Med Rev* 2011; [Epub ahead of print].
47. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 1999; 354:1435–1439.
48. Mangos GJ, Walker BR, Kelly JJ, Lawson JA, Webb DJ, Whitworth JA. Cortisol inhibits cholinergic vasodilation in the human forearm. *Am J Hypertens* 2000; 13:1155–1160.
49. Vasan RS, Evans JC, Larson MG, Wilson PW, Meigs JB, Rifai N, *et al*. Serum aldosterone and the incidence of hypertension in nonhypertensive persons. *N Engl J Med* 2004; 351:33–41.