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## Gender-specific associations of short sleep duration with prevalent and incident hypertension: the Whitehall II Study

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### Abstract

Sleep deprivation ( $\leq 5$ h per night) was associated with a higher risk of hypertension in middle-aged American adults but not among older individuals. However, the outcome was based on self-reported diagnosis of incident hypertension and no gender-specific analyses were included. We examined cross-sectional and prospective associations of sleep duration with prevalent and incident hypertension in a cohort of 10,308 British civil servants aged 35–55 at baseline (Phase 1, 1985–88). Data were gathered from phase 5 (1997–1999) and phase 7 (2003–2004). Sleep duration and other covariates were assessed at phase 5. At both examinations, hypertension was defined as blood pressure  $\geq 140/90$  mmHg or regular use of antihypertensive medications. In cross-sectional analyses at phase 5 ( $n=5,766$ ), short duration of sleep ( $\leq 5$ h per night) was associated with higher risk of hypertension compared to the group sleeping 7h, among women (OR 2.01; 95% CI 1.13 to 3.58), independent of confounders, with an inverse linear trend across decreasing hours of sleep ( $p=0.003$ ). No association was detected in men. In prospective analyses (mean follow-up 5 years), the cumulative incidence of hypertension was 20.0% ( $n=740$ ) among 3,691 normotensive individuals at phase 5. In women, short duration of sleep was associated with higher risk of hypertension in a reduced model (age, employment) [6h per night: 1.56 (1.07 to 2.27),  $\leq 5$ h per night: 1.94 (1.08 to 3.50) vs 7h]. The associations were attenuated after accounting for cardiovascular risk factors and psychiatric co-morbidities [1.42 (0.94 to 2.16); 1.31 (0.65 to 2.63), respectively]. Sleep deprivation may produce detrimental cardiovascular effects among women.

### Keywords

sleep duration; blood pressure; hypertension; gender differences; confounders; co-morbidities

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Sleep-disordered breathing (SDB) has been linked to elevated blood pressure and risk of hypertension in several epidemiological observational studies (1–5). Growing evidence indicates that sleep deprivation as well is associated with a number of health outcomes including hypertension (6–14). For example, in a recent longitudinal analysis of the first

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National Health and Nutrition Examination Survey (NHANES-I), short sleep duration ( $\leq 5$ h per night) was associated with a 60% higher risk of incident hypertension in middle-aged (32-59 years) American adults without apparent sleep disorders, during a mean follow-up of 8-10 years (13). No association was found in individuals 60 years of age or older. However, the outcome was based on self-reported diagnosis of incident hypertension and no gender-specific analyses were included. Furthermore, a cross-sectional analysis from the Sleep Heart Health Study on a sample of nearly 6,000 US adults showed a significant higher prevalence of hypertension among individuals with usual sleep duration above or below the median of 7 to less than 8 hours per night (14). The association was stronger, i.e. a 66% higher risk of hypertension, among short sleepers ( $< 6$  hours per night). While this study attempted to account for a number of potential confounders including psychiatric and cardiovascular co-morbidities, the cross-sectional design did not allow inference on the temporal relationship between sleep duration and hypertension.

Several studies in humans indicate potential pathophysiological mechanisms supporting the biological plausibility of the association between sleep deprivation and hypertension. For example, acute curtailments of sleep may induce an over-activity of the sympathetic nervous system leading to higher blood pressure in both normotensive and hypertensive individuals (15-17). Other contributing mechanisms may include over-activity of the renin-angiotensin-aldosterone system, pro-inflammatory responses, endothelial dysfunction, and renal impairment (18, 19). On the other hand, intervention studies to improve duration and quality of sleep have been effective in reducing both daytime and night-time blood pressures (19, 20). However, there is concern that sleep habits may represent a marker of health status and quality of life rather than a casual factor for hypertension and other health outcomes (21).

In the present analysis, we sought to examine both the cross-sectional and prospective associations of sleep duration with prevalent and incident hypertension in the Whitehall II study, a prospective cohort of 10,308 white-collar British civil servants aged 35-55 at baseline (Phase 1, 1985-88). Because reduced durations of sleep might be associated with more detrimental effects on cardiovascular outcomes among women (6-9), unlike previous investigations we conducted gender-specific analyses with the inclusion of a number of potential confounding variables.

## Methods

### Study population

The Whitehall II cohort was recruited in 1985-1988 (phase 1) from 20 London-based Civil Service departments. The rationale, design, and methods of the study have been described in detail elsewhere (22). Briefly, the initial response rate was 73% and the final cohort consisted of 10,308 participants; 3,413 women and 6,895 men. Follow up screening examinations took place in 1991-1993 (phase 3), 1997-1999 (phase 5), and 2003-2004 (phase 7) whereas postal questionnaires were sent to participants in 1989 (phase 2), 1995 (phase 4) and 2001 (phase 6). The participation rates of the original cohort ( $n=10,308$ ) were 83%, 76% and 68% at phases 3, 5 and 7, respectively. In this report, we used data from phase 5 and phase 7. The total sample at phase 5 consisted of 7,204 participants. The present analyses were restricted to white individuals ( $n = 6,592$ ), given the low numbers of other ethnic groups ( $n = 612$ ). For the cross-sectional analyses, only participants with a complete set of data at phase 5 were included ( $n = 5,766$ : 4,199 men; 1,567 women). Their characteristics were comparable to the overall sample (see Appendix). For the longitudinal analyses, the incidence of hypertension at phase 7 was assessed among participants who were normotensive at phase 5 ( $n = 3,691$ : 2,686 men; 1,005 women).

## Sleep duration

At phases 5 and 7 sleep duration was elicited by the question ‘How many hours of sleep do you have on an average week night?’ Response categories were 5 hours or less, 6, 7, 8, and 9 hours or more.

## Covariates

For the present analyses, age and other covariates were derived from the questionnaires at phase 5. Employment grade was determined from the participant's last known Civil Service grade title (19% had retired by phase 5) and divided into three categories in order of decreasing salary: administrative, professional/executive, and clerical/support. Participants were allocated to one of four smoking categories: never, ex-smoker, pipe and/or cigar only, or current cigarette smoker (manufactured or hand-rolled cigarettes). Alcohol consumption in the previous week was recorded (units per week). Leisure-time physical activity was categorised by energy utilisation in two categories: ‘vigorous’ (subjects who reported at least 1.5h of vigorous activity per week) and no vigorous activity. General health status was assessed using the physical and mental health component summaries of the Short Form-36 (SF-36) health survey questionnaire (23): low scores indicate low functioning. Psychiatric morbidity including depression was assessed with a modified General Health Questionnaire (GHQ) score. Participants taking sleep medication (hypnotics) or cardiovascular drugs were identified through a questionnaire item on current medication. At both phase 5 and 7 screening examinations anthropometric measures were recorded, including height, weight, and waist circumference; body mass index (BMI) was calculated as weight in kilograms (kg)/height in metres (m)<sup>2</sup>. Blood pressure was measured three times using a standard mercury manometer by trained and certified technicians in both examinations. The onset of the first phase (systolic) and fifth phase (diastolic) Korotkoff sounds were recorded. The mean of the second and third measures were used in the analyses. At both examinations, hypertension was defined as blood pressure  $\geq 140/90$  or regular use of antihypertensive medications.

## Ethical approval

Ethical approval for the Whitehall II study was obtained from the University College London Medical School committee on the ethics of human research.

## Statistical analysis

For continuous and categorical variables, respectively, Kruskal Wallis and  $\chi^2$  tests were used to determine the statistical significance of any difference in the distribution of baseline variables at phase 5 across categories of sleep duration. The statistical significance of the interaction between each baseline characteristic and sleep duration, adjusted for other important baseline variables, was tested in multivariate logistic regression models that included this interaction and the corresponding main effect terms. The interaction between sleep duration and gender was significant ( $p < 0.05$ ), therefore all analyses were stratified by gender. No significant interactions were found between sleep duration and age or other relevant covariates. Univariate and multivariate regression analyses were conducted to test the association between categories of sleep duration and prevalent hypertension at phase 5 (cross-sectional analyses), as well as the association between sleep duration at phase 5 and incident hypertension at phase 7 (prospective analyses). Covariates included: baseline age, employment, alcohol consumption, smoking, physical activity, BMI, CVD drugs (other than antihypertensive medications), the SF-36 mental and physical health component scores, depression, and use of hypnotics. In these analyses, 7h of sleep was selected as the reference category. All techniques were implemented using STATA 9.0.

## Results

### Descriptives

Characteristics for both male (Table 1a) and female (Table 1b) participants at phase 5 (baseline) are reported by categories of sleep duration. Among men, participants sleeping 9h or more were, on average, older than other participants; in addition, they were less likely to be physically active, and more likely to be under medication for CVD drugs. On the other hand, male participants sleeping 5h or less had a lower employment status, had higher mean levels of BMI and waist circumference, were more likely to be depressed and under medication for hypnotics, and reported lower scores for mental and physical health than other participants. No significant differences in drinking and smoking habits were reported across categories of sleep duration. For blood pressures, no consistent pattern of association was noted; however, the mean levels of systolic blood pressure (and pulse pressure) as well as the prevalence of hypertension were significantly higher among participants sleeping 8h than in other participants.

Among women, participants at the two extreme categories of sleep duration (ie, 5h or less and 9h or more) were characterized, in general, by a poorer health status and lifestyle profile. In particular, they had a lower employment status, were less likely to be physically active, more likely to be under medication for CVD drugs or hypnotics, more likely to be depressed, and reported lower scores for mental and physical health than other categories. No significant differences in drinking and smoking habits were reported across categories of sleep duration. For blood pressures, there was a consistent pattern of association among female participants sleeping 5h or less, who reported higher mean levels of systolic blood pressure (and pulse pressure) as well as a significantly higher prevalence of hypertension than other participants (in both treated and untreated individuals).

### Cross-sectional analysis

Table 2 displays the odds ratios (OR) and 95% confidence intervals (CI) of prevalent hypertension across categories of sleep duration at phase 5, using 7h of sleep as the reference category. Among men, no consistent pattern of association was noted. Among women, in fully adjusted analyses short duration of sleep ( $\leq 5$ h per night) was associated with a significant higher risk of hypertension compared to the group sleeping 7h (OR 2.01; 95% CI 1.13 to 3.58), independent of several potential confounders, with a significant inverse linear trend across decreasing hours of sleep ( $p=0.003$ ).

### Prospective analysis

Table 3 displays the odds ratios (and 95% CIs) of incident hypertension at phase 7 among participants who were normotensive at phase 5. During a mean follow-up of 5 years, the cumulative incidence of hypertension was 20.0% (740/3,691: 539/2,686 in men; 201/1,005 in women). Among men, no consistent pattern of association was seen across categories of sleep duration. Among women, short duration of sleep was associated with significant higher risks of hypertension compared to the group sleeping 7h in unadjusted analyses as well as in a reduced model (age, employment) [6h per night: 1.56 (1.07 to 2.27),  $\leq 5$ h per night: 1.94 (1.08 to 3.50)]. However, these associations were attenuated after accounting for cardiovascular risk factors and psychiatric co-morbidities [1.42 (0.94 to 2.16); 1.30 (0.65 to 2.62), respectively]. The proportion of the variance of the risk of developing hypertension explained by the age and employment-adjusted model was 2.95%. In the fully-adjusted model it was 6.14%. The major contributor in the full multivariate model was body mass index (explaining 2.23% of the added variance) whereas the remaining covariates all accounted for  $<1\%$  of the remaining difference. Results were virtually unchanged after further adjustment for the baseline values of systolic blood pressure (data not shown).

## Other analyses

We also carried out linear regression analyses to test the association between sleep duration and blood pressures (systolic, diastolic, and pulse pressures) at phase 5 (cross-sectional analyses), as well as the association between sleep duration at phase 5 and changes in blood pressures between the two phases (prospective analyses), among participants not taking antihypertensive medications. In cross sectional analyses, there were consistent, significant inverse associations ( $p < 0.05$ ) between duration of sleep and either systolic blood pressure ( $\beta = -1.24$  mmHg per h sleep; 95% CIs,  $-2.23$  to  $-0.24$ ) or pulse pressure ( $\beta = -0.91$  mmHg per h sleep;  $-1.63$  to  $-0.20$ ), only among women, in fully adjusted models. In prospective analyses, no significant associations were found for any of the blood pressure measures among either male or female participants (data not shown).

## Discussion

Findings from the Whitehall II cohort showed gender-specific associations between sleep duration and hypertension. Specifically, short duration of sleep was associated with higher risks of prevalent and incident hypertension only among women. We could not detect consistent associations among men or for long duration of sleep. For the latter finding, it should be noted, however, that there were very few individuals ( $\sim 1.3\%$  of the overall sample) in our study who reported sleeping 9h or more per night thus yielding a limited statistical power to examine the association between sleep duration and hypertension in this subgroup. Furthermore, our findings point to the importance of a comprehensive scrutiny of potential confounders and mediators when examining the associations between durations of sleep and health outcomes. In fact, the observed associations among women were attenuated after accounting for a number of cardiovascular risk factors, measures of general health, and psychiatric co-morbidities.

A substantial amount of evidence exists about the link between sleep-disordered breathing (SDB) and hypertension (1-5). The epidemiological data has been corroborated by findings of mechanistic studies emphasising the critical role of sympathetic overactivity in the aetiology of SDB-related hypertension (24-25), although other mechanisms are likely to be involved (19). More recently, sleep deprivation has been indicated as a risk factor for several chronic health outcomes in individuals without overt sleep disorders (6-12). However, we are aware of only two population-based studies so far, both coming from the US, that have attempted to examine the association between self-reported durations of sleep and risk of hypertension (13-14). Specifically, in a longitudinal analysis of the NHANES-I ( $n=4,810$ ), short sleep duration ( $\leq 5$ h per night) was associated with a 60% higher risk of incident hypertension, in fully adjusted models, among middle-aged (32-59 years) American adults without apparent sleep disorders (13). No association was found in individuals 60 years of age or older. However, in this study the diagnosis of incident hypertension was based on self-report with a potential of misclassification (under-diagnosis), as suggested by a lower cumulative incidence ( $647/4,810 = \sim 13.5\%$ ) as compared to that observed in our study ( $740/3,691 = 20.0\%$ ), despite comparable age-ranges between the two studies and a longer follow-up period in the NHANES-I (8-10 years vs. 5 years, respectively). In addition, no gender-specific analyses were included in the NHANES-I. Moreover, in a cross-sectional analysis of the large sample of the Sleep Heart Health Study ( $\sim 6,000$  US adults) a significant higher prevalence of hypertension was reported among either short ( $< 6$ h per night) or long sleepers ( $\geq 9$ h per night) as compared to the median duration of sleep of 7 to less than 8h per night (14). However, the association was stronger among short sleepers than in long sleepers (ie, 66% vs. 30% higher risk of hypertension, respectively). While this study accounted for a number of potential covariates including psychiatric and cardiovascular co-morbidities, the cross-sectional design does not allow to exclude the possibility of residual confounding by unknown variables as well as to exclude the potential of reverse causality.



In addition, the Sleep Heart Health Study sample was, on average, older than both NHANES-I and Whitehall II, thus with a higher likelihood of geriatric co-morbidities potentially affecting sleep patterns (26).

## Strengths

Unlike these earlier investigations, our study examined both cross-sectional and prospective gender-specific associations between sleep duration and hypertension with the inclusion of a number of potential confounding variables. Our findings suggest a potential role of sleep deprivation in the aetiology of hypertension and other adverse health outcomes.

First, the observation that reduced duration of sleep may be associated with a higher risk of hypertension only among women is a novel finding. Indeed, while previous investigations have emphasized the potential impact of both short and long durations of sleep on chronic disease risk among women (6-9), the mechanisms underlying the gender-specific association between sleep deprivation and hypertension, observed in our study, are unknown. Given the mean age of our female participants falling around the menopausal period (~ 55 years), we can speculate that the periods marking shifts in the reproductive stages, such as menopause, are particularly vulnerable times for women, because they are associated with major hormonal turmoil and psychosocial stresses that may in turn lead to adverse health outcomes (27). For example, in our sample of female participants, the prevalence of depression cases was higher among women reporting short duration of sleep ( $\leq 5$ h per night) than in other subgroups. In addition, as shown in our descriptive analyses, the distribution of correlates of short sleep duration that have the potential to affect hypertension risk was different between genders and may have partially contributed to the observed associations. Finally, we cannot rule out the possibility of differential self-reporting of sleep habits between men and women, as suggested in a previous analysis from the Sleep Heart Health Study examining the relationship of gender to subjective measures of sleepiness (28).

Second, while findings from cross-sectional analyses consistently showed a strong, significant association between short sleep duration and risk of hypertension among women, in prospective analyses the risk estimates were attenuated after accounting for cardiovascular risk factors, measures of general health, and psychiatric co-morbidities. Thus, these findings emphasize the importance of a comprehensive examination of correlates that are likely to confound or may be on the causal pathway between sleep deprivation and adverse health outcomes. Nevertheless, recent prospective analyses from the MONICA Augsburg survey indicate a modest but significant association between short sleep duration and incident myocardial infarction in middle-aged women, but not men, from the general population (29).

Third, our descriptive analyses clearly demonstrate that both short and long duration of sleep may indeed identify population subgroups with a distinct cluster of socio-demographic characteristics, lifestyle behaviours, and disease conditions, that is likely to be affected by the cultural setting in which the research is being conducted (30-33). In this regard, the study of health consequences related to curtailments of sleep seems to be epidemiologically relevant in the general population, given the downward trends in the average duration of sleep and the increasingly higher prevalence of “short sleepers” in many Western countries (34-35). Conversely, it may be difficult for epidemiological studies to examine the health consequences of long durations of sleep in middle-aged, healthy populations, considering the relatively low prevalence of “long sleepers” in the absence of overt psychiatric co-morbidities (36).

## Limitations

There are limitations in this study. First, the population under investigation is an occupational cohort of white-collar workers and limited to whites, which may reduce the generalizability of our findings to other populations. However, this would not affect the internal validity of our results with respect to the prospective analyses. Second, information about sleep duration was self-reported by the participants. Nevertheless, self-report assessments of sleep have been shown to be valid measures compared to quantitative sleep assessments with actigraphy (37-38). Moreover, because the outcome was also assessed prospectively, any misclassification of sleep duration would be non-differential with respect to incident hypertension, thus resulting in underestimation of the true effects. A further limitation of this study is the relatively short time of follow-up (5 years), which may have precluded us from detecting larger and significant effects of sleep deprivation on subsequent hypertension incidence. The strengths of this study include the simultaneous inclusion of a number of covariates known to be related to both sleep patterns and hypertension. A further strength is that the diagnosis of incident hypertension was also based on directly measured blood pressures at both examinations, thus minimizing the potential of misclassification that occurs when using self report alone.

## Conclusions

In summary, findings from the Whitehall II cohort suggest gender-specific associations between sleep duration and hypertension risk. Specifically, cross-sectional analyses showed a significant, consistent association between short sleep duration ( $\leq 5$ h per night) and risk of hypertension only among women, which was attenuated in prospective analyses after multivariate adjustment.

## Perspectives

Sustained sleep curtailment, ensuing excessive daytime sleepiness and the higher cardiovascular risk are causes for concern. Emerging evidence also suggests a potential role for sleep deprivation as predictor or risk factor for conditions like obesity, diabetes and metabolic syndrome not only in adults (39) but also in children (40). Further prospective studies with improved assessment of long-term exposure (repeated self-reported sleep duration or repeated actigraphy), and better control for confounders are needed before causality can be determined.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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**Table 1a**  
**Baseline characteristics (Phase 5: 1997-1999) across categories of sleep duration in 4,199 men. The Whitehall II study (n=5,766)**

Characteristics	Sleep duration (hours)					p-values*
	≤5	6	7	8	9+	
No of subjects	265	1,383	1,886	620	45	
Age (yr)	54.6(5.6)	54.6(5.7)	55.5(6.1)	57.5(6.1)	57.7(5.4)	<0.001
DBP (mmHg)	79.2(10.9)	78.2(10.4)	78.6(10.5)	79.0(10.9)	78.1(10.6)	0.57
SBP (mmHg)	124.0(16.7)	122.3(15.4)	123.9(15.8)	125.1(17.3)	122.5(16.6)	0.009
Pulse pressure (mmHg)	44.7(11.5)	44.1(10.7)	45.3(11.0)	46.1(12.4)	44.4(11.1)	0.003
Lowest employment n(%)	26(9.8)	66(4.8)	62(3.3)	18(2.9)	3(6.7)	<0.001
BMI (kg/m <sup>2</sup> )	27.0(4.1)	26.4(3.5)	25.9(3.4)	25.4(3.3)	25.7(3.3)	<0.001
Waist (cm)	94.9(11.6)	92.8(10.2)	91.6(9.7)	90.2(9.9)	91.5(8.5)	<0.001
Weekly/Alcohol (units)	18.0(21.2)	16.9(15.9)	16.5(15.9)	16.3(17.1)	17.7(20)	0.58
CVD drugs n(%)	49(18.6)	185(13.4)	259(13.8)	111(18.0)	9(20.0)	0.013
Physical activity n(%)	108(40.8)	687(49.7)	930(49.3)	320(51.6)	17(37.8)	0.022
SF-36 Mental (score)	47.1(12.2)	50.5(9.2)	52.5(8.2)	53.7(7.5)	52.0(9.9)	<0.001
SF-36 Physical (score)	50.2(8.8)	52.1(7.1)	52.4(6.4)	51.9(6.8)	50.9(8.5)	0.019
Use of hypnotics n(%)	7(2.7)	8(0.6)	4(0.2)	2(0.3)	(0)0.0	<0.001
Depression cases n(%)	62(23.8)	178(13.1)	173(9.3)	55(9.0)	4(8.9)	<0.001
Current smoking n(%)	30(11.5)	123(8.9)	154(8.2)	49(7.9)	4(8.9)	0.45
Hypertensive n(%)	84(31.7)	358(25.9)	533(28.3)	205(33.1)	14(31.1)	0.014
No medication	45(17.0)	218(15.8)	323(17.1)	115(18.6)	9(20.0)	
Medication	38(14.3)†	140(10.1)	208(11.0)‡	90(14.5)	5(11.1)	

Data are expressed as the mean (standard deviation) or as n (%).

\* p-value for comparison across sleep duration groups using the chi-squared analysis for categorical variables and Kruskal Wallis test for continuous variables.

† one missing value

‡ two missing values

Table 1b. Baseline characteristics (Phase 5: 1997-1999) across categories of sleep duration in 1,567 women. The Whitehall II study (n=5,766)

Characteristics	Sleep duration (hours)					p-values*
	≤5	6	7	8	9+	
No of subjects	157	511	597	272	30	
Age (yr)	56.4(5.9)	56.1(6.1)	55.7(6.1)	56.8(6.1)	54.7(5.7)	0.07
DBP (mmHg)	75.7(10.4)	74.9(10.1)	74.4(9.8)	74.2(10.0)	74.0(10.0)	0.50
SBP (mmHg)	124.2(18.3)	121.2(16.8)	120.3(17.1)	121.4(16.8)	118(17.2)	0.07
Pulse pressure (mmHg)	48.5(13.8)	46.3(12.3)	45.9(12.3)	47.2(11.6)	44.0(10.9)	0.07
Lowest employment n(%)	52(33.1)	143(28.1)	162(27.2)	67(25.0)	11(36.7)	0.003
BMI (kg/m <sup>2</sup> )	27.1(5.8)	26.2(5.1)	25.9(4.4)	26.3(5.0)	25.8(4.6)	0.31
Waist (cm)	82.5(14.3)	80.2(11.8)	80.1(11.2)	80.4(11.9)	81.5(12.0)	0.66
Weekly/Alcohol (units)	7.3(10.8)	8.0(8.5)	8.2(9.5)	8.4(10.0)	9.8(9.4)	0.10
CVD drugs n(%)	36(23.1)	58(11.4)	94(15.8)	46(16.9)	4(13.3)	0.007
Physical activity n(%)	38(24.2)	169(33.1)	215(36)	108(39.7)	6(20.0)	0.006
SF-36 Mental (score)	42.9(13.1)	49.0(10.3)	50.8(9.2)	52.6(8.8)	46.9(15.0)	<0.001
SF-36 Physical (score)	46.0(12.5)	50.2(8.4)	49.8(9.0)	49.9(8.8)	45.2(10.1)	<0.001
Use of hypnotics n(%)	2(1.3)	5(1.0)	4(0.7)	2(0.7)	1(3.3)	0.60
Depression cases n(%)	50(32.5)	72(14.4)	66(11.2)	20(7.4)	5(16.7)	<0.001
Current smoking n(%)	20(12.9)	79(15.5)	76(12.8)	31(11.4)	1(3.3)	0.22
Hypertensive n(%)	58(36.9)	124(24.3)	152(25.5)	66(24.3)	7(23.3)	0.024
No medication	27(17.2)	72(14.1)	69(11.6)	29(10.7)	5(16.7)	
Medication	31(19.7)	52(10.2)	83(13.9)	37(13.6)	2(6.7)	

Data are expressed as the mean (standard deviation) or as n (%).

\* p-value for comparison across sleep duration groups using the chi-squared analysis for categorical variables and Kruskal Wallis test for continuous variables.

**Table 2**  
**Odds Ratio (95% CI) of prevalent hypertension across categories of sleep duration at phase 5 (1997-1999). The Whitehall II study (n=5,766)**

Sample Models	Sleep duration (hours)							p-value*							
	<=5	6	7	8	9+	Linear	Non-linear								
<b>Male (n=4,199)</b>															
No	n=265	n=1,383	n=1,886	n=620	n=45										
Cases	n=84	n=358	n=533	n=205	n=14										
Model 1 †	1.18	0.89	1.55	0.76	1.04	1	1.25 †	1.03	1.52	1.15	0.61	2.17	0.06	0.015	
Model 2 §	1.23	0.92	1.63	0.93	0.79	1.09	1	1.11	0.91	1.36	0.99	0.52	1.90	0.70	0.09
Model 3 #	0.88	0.63	1.23	0.86	0.72	1.03	1	1.12	0.89	1.41	0.92	0.44	1.92	0.05	0.54
<b>Female (n=1,567)</b>															
No	n=157	n=511	n=597	n=272	n=30										
Cases	n=58	n=124	n=152	n=66	n=7										
Model 1 †	1.72 †	1.18	2.49	0.94	0.71	1.23	1	0.94	0.67	1.31	0.89	0.37	2.12	0.13	0.06
Model 2 §	1.64 †	1.11	2.42	0.90	0.68	1.19	1	0.83	0.58	1.17	0.98	0.40	2.40	0.12	0.15
Model 3 #	1.72 †	1.07	2.75	0.92	0.67	1.28	1	0.74	0.50	1.11	0.70	0.21	2.37	0.037 †	0.31

\* p value for test of linear and nonlinear trends.

† p<0.05.

‡ Model 1: unadjusted.

§ Model 2: adjusted for age and employment.

# Model 3: Model 2 + alcohol consumption, smoking, physical activity, BMI, SF36 Mental, SF36 Physical, depression cases, hypnotics use, CVD drugs

**Table 3**  
**Odds Ratio (95% CI) of incident hypertension at phase 7 (2002-2003) across categories of sleep duration at phase 5 (1997-1999). The Whitehall II study (n=3,691)**

Sample Models	Sleep duration (hours)						p-value *									
	<=5	6	7	8	9+	Linear	Non-linear									
No	n=160	n=900	Male (n=2,686)		n=372	n=30										
Cases	n=29	n=179	n=243	n=85	n=3											
Model 1 †	0.89	0.58	1.37	1.00	0.81	1.24	1	1.20	0.90	1.58	0.45	0.13	1.49	0.60	0.53	
Model 2 §	0.96	0.62	1.48	1.07	0.86	1.34	1	1.07	0.80	1.42	0.36	0.11	1.19	0.51	0.42	
Model 3 #	0.89	0.55	1.43	1.02	0.80	1.31	1	1.11	0.80	1.53	0.13 †	0.02	0.98	0.55	0.18	
								Female (n=1,005)								
No	n=75	n=330	n=394	n=186	n=20											
Cases	n=20	n=77	n=64	n=37	n=3											
Model 1 †	1.88 †	1.05	3.34	1.56 †	1.08	2.27	1	1.28	0.82	2.01	0.91	0.26	3.20	0.029 †	0.38	
Model 2 §	1.94 †	1.08	3.50	1.56 †	1.07	2.27	1	1.17	0.74	1.86	0.92	0.26	3.27	0.015 †	0.42	
Model 3 #	1.31	0.65	2.63	1.42	0.93	2.16	1	0.99	0.59	1.69	1.07	0.29	3.94	0.12	0.61	

\* p value for test of linear and nonlinear trends.

† p<0.05.

‡ Model 1: unadjusted.

§ Model 2: adjusted for age and employment.

# Model 3: Model 2 + alcohol consumption, smoking, physical activity, BMI, SF36 Mental, SF36 Physical, depression cases, hypnotics use, CVD drugs