Published in final edited form as:
Am J Epidemiol. 2008 February 1; 167(3): 321–329. doi:10.1093/aje/kwm302.

Cross-sectional versus prospective associations of sleep duration with changes in relative weight and body fat distribution: the Whitehall II Study

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Abstract

A cross-sectional relation between short sleep and obesity has not been confirmed prospectively. We examined the relationship between sleep duration and changes in body mass index (BMI) and waist circumference using the Whitehall II study, a prospective cohort of 10,308 white-collar British civil servants aged 35–55 in 1985–88. Data were gathered in 1997–9 and 2003–4. Sleep duration and other covariates were assessed. Changes in BMI and waist circumference were assessed between the two phases. The incidence of obesity (BMI ≥30 kg/m^2) was assessed among non-obese participants at baseline. In cross-sectional analyses (n=5,021), there were significant, inverse associations ($p<0.001$) between duration of sleep and both BMI and waist circumference. Compared to 7h sleep short duration of sleep (≤5h) was associated with higher BMI (β=+0.82 units; 95% CI 0.38 to 1.26) and waist circumference (β=+1.88 cm; 0.64 to 3.12), and with an increased risk of obesity (OR_{adj} 1.65; 1.22 to 2.24). In prospective analyses, short duration of sleep was not associated with significant changes in BMI (β=−0.06; −0.26 to 0.14) or waist circumference (β=+0.44; −0.23 to 1.12), nor with the incidence of obesity (OR_{adj} 1.05; 0.60 to 1.82). There is no temporal relationship between short duration of sleep and future changes in measures of body weight and central adiposity.

Keywords

sleep duration; relative weight; body fat distribution; obesity; epidemiology

INTRODUCTION

Numerous epidemiologic studies indicate possible associations between sleep disturbances or voluntary shortened sleep duration and several health outcomes, including total mortality, cardiovascular disease, type 2 diabetes mellitus, respiratory disorders, and poor self-rated

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Competing interests: F.P.C. holds the Cephalon Chair, an endowed post at Warwick Medical School, the result of a donation from the company. The appointment to the Chair was made entirely independently of the company and the post-holder is free to devise his own program of research. Cephalon do not have any stake in IP associated with the post-holder and the Chair has complete academic independence from the company.

health (1–13). In addition, several studies have suggested a relationship between short sleep duration and increased body weight or obesity in both children and adults (14–26). Specifically, an inverse association has been reported between the number of hours of sleep and body weight or body mass index. However, these observations come predominantly from cross-sectional studies (14–22). Results from a few longitudinal analyses have been inconsistent with only one study showing significant, consistent associations (23–26). Specifically, a recently published report from the Nurses' Health Study has shown that a habitual sleep time of less than 7 hours predicted a modest increase in weight and incident obesity independent of baseline weight (26). This analysis, however, was based on self-reported measures of body weight, was limited to a large cohort of highly selected middle-aged women, and did not take measures of body fat distribution into account. Altogether, the current epidemiologic evidence does not allow for conclusive inferences in favor of a causal link between short sleep duration and obesity due to a number of unresolved issues (27–28). First, sleep disorders are often co-morbid, especially in the elderly, with medical and psychiatric conditions that can compromise the quality and duration of sleep (29), thus increasing the likelihood of reverse causality and bidirectional relationships, especially in cross-sectional studies. Second, the biological mechanisms underlying the observed associations are unclear, although the evidence from experimental studies suggests possible explanations, such as alterations in hormonal and neurovegetative responses, and effects on the immune function. Third, given the lack of specificity of the observed associations, it is possible that other factors or co-morbid conditions may confound the associations between duration and quality of sleep and health outcomes.

The Whitehall II study gives a unique opportunity to address several of the open questions discussed above. Specifically, in this study we sought to examine the cross-sectional and longitudinal relationships between sleep duration and changes in continuous measures of relative weight (i.e., BMI) and body fat distribution (i.e., waist circumference) as well as with the incidence of obesity and overweight, taking into account a number of potential confounding factors.

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 (30). Briefly, the study was set up to determine the pathways underlying the social differences in health that were apparent in the original Whitehall Study (31), and was specifically designed to focus on pathways not originally examined such as psychosocial and dietary factors. 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, after excluding those who did not provide complete data (n=630). 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 =5,021). For the longitudinal analyses on changes in continuous measures of BMI and waist circumference between phase 5 and 7, only participants with a complete set of data at both phases were included (n=4,378). Finally, the incidence of obesity (BMI ≥ 30 kg/m^2) at phase 7 was assessed among non-obese participants at phase 5 (n=3,786). For the purpose of

sensitivity analysis, the combined incidence of overweight and obesity (BMI ≥ 25 kg/m^2) at phase 7 was also assessed among normal-weight participants at phase 5 (*n*=1,887).

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 questionnaire at phase 5. Employment grade, as a measure of current or recent social position, was determined from the participants' 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). For leisure-time physical activity, participants were asked to indicate the number of occasions and hours spent doing a series of specific activities over the past 4 weeks. These activities were classified into light, moderate or vigorous activities on the basis of their energy expenditure (METS). In this study, leisure-time physical activity was categorized by energy use in two categories: 'vigorous' (subjects who reported at least 1.5 h 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 (32); low scores indicate low functioning. Psychiatric morbidity including depressive symptoms 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. Specifically, participants listed all medications that had been prescribed by a doctor that they had taken in the last 14 days. These medications were coded to classify types of medications. Medication for cardiovascular disease (CVD) included any antihypertensive drugs, lipid lowering drugs, nitrates or anti-platelet drugs. At both phase 5 and 7 screening examinations, several anthropometric measures were recorded, including height, weight, and waist circumference; body mass index (BMI) was calculated as weight in kilograms (kg)/ height in meters (m^2).

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 χ^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. Multivariate linear regression analyses were performed to test the association between categories of sleep duration and continuous measures of BMI and waist circumference at phase 5 (cross-sectional analyses), as well as the association between categories of sleep duration at phase 5 and changes in BMI and waist circumference between the two phases (prospective analyses), by modeling 4 dummy variables and using 7 hours of sleep as the reference. The coefficients estimate the adjusted mean difference in BMI and waist circumference or changes in BMI and waist circumference between each category and the reference. Covariates included: baseline age, sex, employment, as well as the baseline value of BMI or waist in prospective analyses to

account for the phenomenon of "regression to the mean" (33) (model 1); alcohol consumption, smoking, physical activity, and CVD drugs (model 2 + variables of model 1); the SF-36 mental and physical health scores, depressive symptoms, and use of hypnotics (model 3 + variables of model 2). Logistic regression analyses were also performed to examine the cross-sectional association between categories of sleep duration and prevalent obesity (BMI ≥ 30) at phase 5, as well as the prospective association between categories of sleep duration at phase 5 and incident obesity at phase 7 among participants who were non-obese (BMI < 30) at phase 5. Sensitivity analyses were performed by using the cut-off value for overweight (BMI ≥ 25) to assess the prevalence as well as the incidence of combined overweight and obesity. In these analyses, 7 hours of sleep was selected as the reference category. Subgroup analyses were also performed after exclusion of participants on medication for cardiovascular disease. All analyses were carried out using STATA 9.0.

RESULTS

Characteristics of participants at phase 5 ("baseline") are reported in table 1 by categories of sleep duration and sex. In general, participants of both sexes sleeping 5 hours or less had a poorer health status and lifestyle profile. For example, they had higher mean levels of BMI and waist circumference, were less likely to be physically active and more likely to be current smokers (men only); they were more likely to use CVD drugs (women only), more likely to report depressive symptoms, and had lower scores for mental and physical health than other categories.

Table 2 shows the cross-sectional association between categories of sleep duration and continuous measures of BMI and waist circumference at phase 5. Compared to 7 hours of sleep, participants sleeping 5 hours or less were characterized by higher levels of both BMI (β=+0.82 units; 95%CI 0.38 to 1.26) and waist circumference (β=+1.88 cm; 0.64 to 3.12). Moreover, there were consistent, significant inverse associations ($p < 0.001$) between duration of sleep (as a continuous variable) and both BMI (β = −0.36; 95% CI, −0.49 to −0.24) and waist circumference (β = −0.96; −1.32 to −0.60) (Appendix 1 on-line).

Table 3 shows the prospective association between categories of sleep duration at phase 5 and changes in BMI and waist circumference between the two phases. Compared to 7 hours of sleep, short duration of sleep (i.e. ≤5 hours) was not associated with significant changes in BMI (β=−0.06; −0.26 to 0.14) or waist circumference (β=+0.44; −0.23 to 1.12). Results were virtually identical without the inclusion of the baseline value of BMI (β=−0.06; −0.25 to 0.14) or waist circumference (β=+0.37; −0.31 to 1.05). When using duration of sleep as a continuous variable, no significant changes in BMI (β = 0.03; −0.03 to 0.08) or waist circumference (β = −0.08; −0.28 to 0.11) were observed between the two phases. Findings were not substantially changed after exclusion of participants on CVD drugs ($n = 639$) (Appendix 2 and Appendix 3 on-line).

Table 4 displays the odds ratios (and 95% CIs) of prevalent obesity ($n = 691$, 13.8%) across categories of sleep duration at phase 5, using 7 hours of sleep as the reference category. In the fully adjusted model, participants sleeping 5 hours or less had a significant increased risk of obesity (OR 1.65; 1.22 to 2.24) compared to those sleeping 7 hours, with a significant liner trend across categories of sleep duration ($p = 0.003$). Likewise, in sensitivity analyses participants sleeping 5 hours or less reported a significant increased combined prevalence of overweight and obesity (OR 1.41; 1.10 to 1.80) compared to those sleeping 7 hours, with a significant linear trend across categories of sleep duration ($p < 0.001$).

Table 5 displays the odds ratios (and 95% CIs) of incident obesity ($n = 246$, 6.5%) at phase 7 among participants who were non-obese (BMI < 30) at phase 5 ($n = 3,786$). No significant

associations were reported across categories of sleep duration, although the risk estimates of obesity were in the expected direction among participants sleeping less than 7 hours (OR 1.21; 0.89 to 1.64, for 6 hours; OR 1.05; 0.60 to 1.82, for 5 hours and less). Likewise, no significant associations were reported when using a different cut-off value (BMI ≥ 25 kg/m^2) for the combined incidence of overweight and obesity (OR 1.09; 0.83 to 1.41, for 6 hours; OR 1.28; 0.80 to 2.06, for 5 hours and less). After exclusion of participants on CVD drugs, findings were not substantially different (Appendix 4 on-line).

DISCUSSION

Cross-sectional findings from the Whitehall II cohort showed that shorter duration of sleep was significantly associated, in a linear fashion, with greater body weigh and risk of obesity, which is consistent with previous cross-sectional reports (14–22, 24). Specifically, the regression coefficient of −0.36 (−0.49 to −0.24) for the cross-sectional association between the number of hours of sleep and BMI is consistent with the magnitude of the association observed in a recently published cross-sectional analysis from a population-based sample in the US (21). Moreover, unlike previous investigations, which did not include measures of body fat distribution and central adiposity, in the cross-sectional analyses we also found a significant, inverse association between hours of sleep and waist circumference. Finally, individuals sleeping 5 hours or less had a 65% increased risk of obesity compared to those sleeping 7 hours, in fully adjusted models, which is consistent with pooled estimates reported in a meta-analysis of cross-sectional studies (34).

The prospective analyses, however, did not show a significant association between sleep duration and future changes in body weight or waist circumference, therefore not supporting a temporal relationship between short sleep duration and obesity, an essential criterion to infer causality. Indeed, most of the evidence so far comes from cross-sectional analyses (14–22, 24), which preclude the possibility of sorting out the bidirectional relationship between sleep duration and body weight. On the other hand, change models offer the possibility of controlling for all time-stable confounders. Obesity is often co-morbid with several medical conditions that in turn may impair the quality and duration of sleep (29). Findings from a few longitudinal investigations have been inconsistent (23–26). First, Hasler *et al* found that short sleep time was associated with subsequent obesity in subjects younger than 35 years but the association diminished in older participants (23). However, this study was based on a small sample (n = 496) with over-representation of cases with psychiatric disorders and, thus, with a limited generalizability. In addition, in this study the potential for reverse causality could not be excluded because the authors also found an association between earlier obesity and future short sleep duration. Second, Gangwisch *et al* reported inconsistent findings from cross-sectional and longitudinal analyses on the large US sample of the NHANES-I (24). Similar to our study, the authors found significant cross-sectional associations between short sleep duration and high body weight and obesity, whereas sleep duration at baseline was not associated with significant future changes in BMI over a mean follow-up of 8–10 years ($\beta = -0.053$, p =0.27), consistent with our findings. It should be noted, however, that this analysis was based on self-reported weights to compute BMI during the follow-up and did not include measures of body fat distribution. Third, a recently published report from the large sample of the Nurses' Health Study (n = 68,183) has shown that a habitual sleep time of less than 7 hours predicted a modest increase in weight over a mean follow-up of 16 years (26). In addition, the relative risks for incident obesity were 1.15 (1.04, 1.26) and 1.06 (1.01, 1.11) for women sleeping 5 and 6 hours, respectively, compared to those sleeping 7 hours, which resemble our point risk estimates. However, this analysis was based on self-reported measures of body weight, did not include measures of body fat distribution, and was limited to a large cohort of middle-aged women from the nursing profession, thus with a limited generalizability. Conversely, there were no significant

associations between sleep duration at baseline and weight gain (and incident type 2 diabetes) in a population-based study of 1,462 Swedish women followed for 32 years (25). It should be noted that in this study the authors did find significant, inverse associations between sleep duration and BMI and waist-to-hip ratio in cross-sectional analyses at baseline, which is consistent with our findings. In summary, our results are consistent in showing inverse cross-sectional relationships between short sleep and obesity but no prospective associations.

The biological mechanisms underlying the observed associations between short sleep duration and increased body weight are unclear, although the evidence from physiological studies suggests some possible explanations. For example, sleep deprivation has been found to influence hormones that control appetite. In a small study on 12 young, healthy men, an induced short-term sleep restriction was associated with a reduction in the levels of the anorexigenic hormone leptin on one hand and an increase in the levels of the orexigenic hormone ghrelin, and thus increased hunger (35). Similar associations were found in cross-sectional analyses from the Wisconsin Sleep Cohort Study, suggesting that these effects may persist with long-term sleep restriction (19). Glucose homeostasis is also affected by sleep deprivation since glucose uptake varies through different phases of sleep, and adverse effects of sleep deprivation have been reported on cortisol levels, glucose tolerance, and growth hormone secretion (36–38). Moreover, sleep deprivation has been associated with changes in levels of circulating catecholamines and alterations of neurovegetative responses (39). Recent findings also suggest that sleep may have powerful effects on the immune function and that inflammatory responses to sleep deprivation may represent one mechanism linking short sleep duration to obesity and other metabolic disorders (40–43). Finally, we cannot exclude that sleep deprivation may represent a risk marker for poorer health outcomes and impaired quality of life rather than a casual factor for obesity and other related diseases (11). Indeed, our descriptive analyses showing a poorer health status and lifestyle profile associated with shorter duration of sleep strongly suggest that other factors and co-morbidities could confound the associations between duration and quality of sleep and health outcomes.

There are some limitations in our study. First, the population 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 longitudinal 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 (44). Moreover, because outcomes were assessed prospectively, any misclassification of sleep duration would be non-differential with respect to weight gain, 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 future changes in body weight and fat distribution. However, other longitudinal analyses (23–25), but one (26), based on longer follow-ups, still failed to detect significant and consistent associations. The strengths of this study include the simultaneous inclusion of a number of covariates known to be related to both sleep habits and obesity. A further strength is that anthropometric measures of relative weight and body fat distribution were directly measured at both examinations and were not based on questionnaire like in previous longitudinal investigations, thus minimizing the potential of recall bias and misclassification that occurs when using self report alone.

In summary, consistent with previous investigations, this study demonstrates significant associations between short duration of sleep and greater body weight, central adiposity and risk of obesity, in cross-sectional analyses. However, prospective analyses do not support a

temporal relationship between short duration of sleep and gains in body weight or central adiposity, which is consistent with all (23–25) but one (26) previous longitudinal studies. While sustained sleep curtailment and ensuing excessive daytime sleepiness are undoubtedly cause for concern, our study suggests that short duration of sleep might represent a risk marker rather than a causative risk factor for obesity. Further prospective studies with objective assessment of long-term exposure (e.g., repeated actigraphy), more specific outcomes (including direct measures of adiposity) and better control for confounders are needed before causality can be determined.

Acknowledgments

The Whitehall II study has been supported by grants from the Medical Research Council; British Heart Foundation; Health and Safety Executive; Department of Health; National Heart Lung and Blood Institute (HL36310), US, NIH; National Institute on Aging (AG13196), US, NIH; Agency for Health Care Policy Research (HS06516); and the John D and Catherine T MacArthur Foundation Research Networks on Successful Midlife Development and Socio-economic Status and Health. J.E.F. is supported by the MRC (Grant number G8802774), M.J.S. by a grant from the British Heart Foundation, and M.G.M. by an MRC Research Professorship. We thank all participating Civil Service departments and their welfare, personnel, and establishment officers; the Occupational Health and Safety Agency; the Council of Civil Service Unions; all participating civil servants in the Whitehall II study; all members of the Whitehall II study team.

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Table 1

Baseline characteristics* of subjects by sleep duration category and by sex. The Whitehall II Study, London, England (Phase 5: 1997–1999, n=5,021)

	Sleep duration (hours)					p-value†
	≤5	6	7	8	≥ 9	
Men (n=3,619)						
No of subjects	238	1,193	1,619	532	37	
Age (yrs)	54.5(5.5)	54.6(5.7)	55.5(6.1)	57.4(6.2)	57.8(5.3)	0.001
Lowest employment (%)	10.1	4.8	3.4	3.4	5.4	<0.001
BMI (kg/m^2)	27.0(4.1)	26.4(3.5)	25.9(3.4)	25.4(3.3)	25.7(3.3)	0.001
Weight (Kg)	83.0(12.5)	82.2(12.2)	81.0(11.6)	79.1(11.1)	80.7(10.3)	0.001
Waist (cm)	95.0(11.6)	92.9(10.2)	91.6(9.7)	90.2(9.9)	91.5(8.5)	0.001
Weekly alcohol (units)	50.2(9.0)	52.1(6.9)	52.4(6.4)	51.9(6.8)	50.5(9.1)	0.03
Current smoking (%)	11.9	8.6	8.7	8.7	10.8	0.56
Physical activity (%)	41.6	49.9	49.7	50.6	35.1	0.06
CVD drugs (%)	19.0	14.1	14.2	18.1	24.3	0.03
SF-36 Mental (score)‡	46.9(12.4)	50.5(9.2)	52.5(8.2)	53.7(7.5)	52.3(8.5)	0.001
SF-36 Physical (score)§	49.6(9.7)	52.1(7.0)	52.3(6.6)	51.9(6.8)	50.1(8.9)	0.001
Depressive symptoms (%)	24.8	12.9	9.5	9.1	8.1	<0.001
Use of Hypnotics (%)	3.0	0.5	0.1	0.4	0	<0.001
Women (n=1,402)						
No of subjects	142	457	534	242	27	
Age (yrs)	56.4(6.0)	56.0(6.0)	55.7(6.2)	56.9(6.1)	55.0(5.9)	0.10
Lowest employment (%)	33.1	27.7	26.8	25.5	37.0	0.001
BMI (kg/m^2)	27.1(5.8)	26.2(5.1)	25.9(4.4)	26.4(5.0)	25.8(4.6)	0.31
Weight (Kg)	70.6(15.7)	69.7(14.0)	69.0(12.2)	69.8(13.2)	69.3(11.2)	0.31
Waist (cm)	82.3(14.3)	80.3(11.8)	80.1(11.2)	80.2(11.9)	81.7(12.2)	0.78
Weekly alcohol (units)	7.3(10.9)	8.0(8.6)	8.3(9.8)	8.3(10.1)	8.4(10.1)	0.06
Current smoking (%)	13.6	16.3	12.6	12.4	3.7	0.23
Physical activity (%)	25.4	33.5	35.0	38.0	22.2	0.08

	Sleep duration (hours)					p-value†
	≤5	6	7	8	≥ 9	
	Men (n=3,619)					
CVD drugs (%)	22.7	10.9	16.3	16.9	14.8	0.009
SF-36 Mental (score)**	42.5(13.4)	48.9(10.4)	50.6(9.2)	52.4(8.9)	46.6(15.8)	0.001
SF-36 Physical (score)††	45.9(12.8)	50.2(8.4)	49.9(8.9)	49.9(8.8)	45.1(10.4)	0.001
Depressive symptoms (%)	32.9	14.1	12.1	7.9	18.5	<0.001
Use of Hypnotics (%)	1.4	1.1	0.8	0.8	3.7	0.61

*Data are expressed as the mean (standard deviation) or as percentages.

†P-value for comparison across sleep duration categories using the chi-squared analysis for categorical variables and Kruskal Wallis test for continuous variables.

‡range 8.0 to 67.5

§range 8.4 to 68.7

**range 7.8 to 70.8

††range 11.5 to 69.5

Table 2

Cross-sectional relationships* of duration of sleep (hours) with BMI and waist circumference at phase 5 (1997–1999). The Whitehall II Study, London, England (n=5,021).

	≤5h (n=380)	6h (n=1,650)	7h (n=2,153)	8h (n=774)	≥9h (n=64)	p-value†	
	Coef. (95% CI)	Coef. (95% CI)	reference	Coef. (95% CI)	Coef. (95% CI)	Linear	Non-linear
BMI (Kg/m^2)							
Model 1	1.09‡ (0.66, 1.52)	0.41‡ (0.16, 0.66)	0	−0.28 (−0.6, 0.04)	−0.22 (−1.19, 0.75)	<0.001	0.92
Model 2	0.94‡ (0.51, 1.36)	0.44‡ (0.19, 0.68)	0	−0.27 (−0.59, 0.04)	−0.45 (−1.41, 0.52)	<0.001	0.55
Model 3	0.82‡ (0.38, 1.26)	0.43‡ (0.18, 0.68)	0	−0.25 (−0.57, 0.07)	−0.62 (−1.59, 0.35)	<0.001	0.84
Waist (Cm)							
Model 1	2.90‡ (1.69, 4.11)	0.97‡ (0.26, 1.68)	0	−1.17‡ (−2.1, −0.24)	0.25 (−2.48, 2.98)	<0.001	0.23
Model 2	2.43‡ (1.23, 3.62)	1.04‡ (0.35, 1.74)	0	−1.18‡ (−2.09, −0.27)	−0.73 (−3.43, 1.97)	<0.001	0.68
Model 3	1.88‡ (0.64, 3.12)	0.93‡ (0.23, 1.64)	0	−1.09‡ (−2.01, −0.18)	−1.38 (−4.06, 1.3)	<0.001	0.95

*Results are expressed as β coefficients and 95% confidence intervals (CI) compared to 7h of sleep category

†P-values for test of linear and nonlinear trends

‡Significant p-values ($p \leq 0.05$) for contrast of that specific category versus reference category

Model 1: adjusted for age, sex, employment

Model 2: Model 1 + alcohol consumption, smoking, physical activity and cardio-vascular drugs

Model 3: Model 2 + mental and physical scores (SF36), depressive symptoms and use of hypnotics

Table 3

Prospective relationships* of duration of sleep (hours) with changes in BMI and waist circumference between phase 5 (1997–1999) and phase 7 (2003–2004). The Whitehall II Study, London, England (n=4,378).

	≤5h (n=307)	6h (n=1,429)	7h (n=1,903)	8h (n=678)	≥9h (n=61)	p-value†	
	Coef. (95% CI)	Coef. (95% CI)	reference	Coef. (95% CI)	Coef. (95% CI)	Linear	Non-linear
BMI (Kg/m^2)							
Model 1	0.02 (−0.17, 0.21)	0.02 (−0.09, 0.13)	0	0.08 (−0.06, 0.22)	−0.20 (−0.59, 0.20)	0.89	0.72
Model 2	0.01 (−0.18, 0.20)	0.03 (−0.08, 0.13)	0	0.08 (−0.06, 0.21)	−0.20 (−0.60, 0.20)	0.95	0.81
Model 3	−0.06 (−0.26, 0.14)	0.00 (−0.11, 0.11)	0	0.09 (−0.05, 0.23)	−0.17 (−0.58, 0.24)	0.36	0.82
Waist (Cm)							
Model 1	0.56 (−0.08, 1.21)	0.14 (−0.22, 0.51)	0	0.14 (−0.33, 0.61)	−0.39 (−1.73, 0.96)	0.22	0.44
Model 2	0.58 (−0.07, 1.23)	0.16 (−0.20, 0.53)	0	0.11 (−0.37, 0.58)	−0.30 (−1.67, 1.07)	0.17	0.50
Model 3	0.44 (−0.23, 1.12)	0.12 (−0.26, 0.49)	0	0.15 (−0.33, 0.63)	−0.34 (−1.71, 1.03)	0.39	0.58

*Results are expressed as β coefficients and 95% confidence intervals (CI) compared to 7h of sleep category

†P-values for test of linear and nonlinear trends

Model 1: adjusted for age, sex, employment, and baseline value of BMI or waist

Model 2: Model 1 + alcohol consumption, smoking, physical activity and cardio-vascular drugs

Model 3: Model 2 + mental and physical scores (SF36), depressive symptoms and use of hypnotics

Table 4

Odds ratio (95% CI) of prevalent obesity and overweight + obesity across categories of sleep duration at phase 5 (1997–1999). The Whitehall II Study, London, England (n=5,021).

	≤5h (n=380)	6h (n=1,650)	7h (n=2,153)	8h (n=774)	≥9h (n=64)	p-value*	
Obesity (BMI ≥ 30)	**88 cases**	**238 cases**	**259 cases**	**97 cases**	**9 cases**	**Linear**	**Non-linear**
Model 1	2.02† (1.53, 2.66)	1.20 (0.99, 1.45)	1	1.01 (0.78, 1.30)	1.08 (0.53, 2.22)	<0.001	0.01
Model 2	1.88† (1.41, 2.49)	1.24† (1.02, 1.51)	1	1.03 (0.79, 1.33)	0.99 (0.48, 2.08)	<0.001	0.06
Model 3	1.65† (1.22, 2.24)	1.23† (1.01, 1.50)	1	1.02 (0.78, 1.33)	0.89 (0.42, 1.90)	0.003	0.30
Overweight + Obesity (BMI ≥ 25)	245 cases	1,004 cases	1,198 cases	392 cases	35 cases		
Model 1	1.45† (1.15, 1.82)	1.25† (1.10, 1.43)	1	0.80† (0.67, 0.94)	0.97 (0.59, 1.60)	<0.001	0.36
Model 2	1.38† (1.09, 1.74)	1.27† (1.11, 1.46)	1	0.80† (0.67, 0.95)	0.86 (0.51, 1.44)	<0.001	0.35
Model 3	1.41† (1.10, 1.80)	1.28† (1.12, 1.47)	1	0.82† (0.69, 0.98)	0.82 (0.48, 1.39)	<0.001	0.49

*P-values for test of linear and nonlinear trends

†Significant p-values ($p \leq 0.05$) for contrast of that specific category versus reference category

Model 1: adjusted for age, sex, employment

Model 2: Model 1 + alcohol consumption, smoking, physical activity and cardio-vascular drugs

Model 3: Model 2 + mental and physical scores (SF36), depressive symptoms and use of hypnotics

Table 5

Odds ratio (95% CI) of incident obesity (n=3,786)* and incident overweight + obesity (n=1,887)† at phase 7 (2003–2004) across categories of sleep duration at phase 5 (1997–1999). The Whitehall II Study, London, England.

	≤5h	6h	7h	8h	≥9h	p-value*	
Obesity (BMI ≥ 30)	**18 cases / 243 subjects**	**90 cases / 1,229 subjects**	**101 cases / 1,668 subjects**	**34 cases / 593 subjects**	**3 cases / 53 subjects**	**Linear**	**Non-linear**
Model 1	1.18 (0.70, 1.99)	1.22 (0.91, 1.64)	1	0.95 (0.64, 1.42)	0.88 (0.27, 2.88)	0.17	0.87
Model 2	1.14 (0.67, 1.93)	1.25 (0.93, 1.68)	1	0.90 (0.60, 1.36)	0.89 (0.27, 2.94)	0.12	0.72
Model 3	1.05 (0.60, 1.82)	1.21 (0.89, 1.64)	1	0.97 (0.64, 1.47)	0.91 (0.27, 3.01)	0.34	0.77
Overweight + Obesity (BMI ≥ 25)	31 cases / 114 subjects	132 cases / 567 subjects	185 cases / 847 subjects	61 cases / 331 subjects	7 cases / 28 subjects		
Model 1	1.33 (0.85, 2.08)	1.09 (0.85, 1.41)	1	0.82 (0.59, 1.13)	1.29 (0.54, 3.10)	0.07	0.74
Model 2	1.32 (0.84, 2.09)	1.10 (0.85, 1.42)	1	0.81 (0.59, 1.13)	1.23 (0.51, 2.97)	0.07	0.80
Model 3	1.28 (0.80, 2.06)	1.09 (0.83, 1.41)	1	0.84 (0.60, 1.17)	1.29 (0.53, 3.14)	0.13	0.77

*Number of non-obese participants at phase 5 (BMI < 30)

†Number of normal-weight participants at phase 5 (BMI < 25)

‡P-values for test of linear and non-linear trends

Model 1: adjusted for age, sex, employment

Model 2: Model 1 + alcohol consumption, smoking, physical activity and cardio-vascular drugs

Model 3: Model 2 + mental and physical scores (SF36), depressive symptoms and use of hypnotics