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Association between self-reported sleep duration and serum lipid profile in a middle-aged and elderly population in Taiwan: A community-based, cross-sectional study

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Association between sleep duration and serum lipid profile

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ABSTRACT

Objectives: The association between sleep duration and serum lipid profile in the middle-aged and elderly is unclear. The aim of this study was to investigate and evaluate the relationships between sleep duration and levels of serum total cholesterol (TC), LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), and triglycerides (TG) in these populations. Design: Cross-sectional observational study.

Setting: Community-based investigation in Guishan township of northern Taiwan. Participants: A total of 400 community-dwelling middle-aged and elderly individuals were enrolled. All participants underwent a baseline assessment in 2014, which included anthropometrics, blood samples, and self-administered questionnaires. Participants were divided into three groups based on their sleep duration.

Outcome measures: Multivariate logistic regression was used to obtain odds ratios (ORs) and 95% confidence intervals (CIs) to assess the relationship between sleep duration and lipid profiles.

Results: Participant mean age was 64.5 years old and 35.3% were men. Subjects with longer (>7 hours) and shorter (<6 hours) nightly sleep duration had a higher prevalence of low HDL-C levels (HDL \leq 40 mg/dL) than those with moderate sleep duration (6-7 hours). Multivariate logistic regression revealed that, compared to individuals with sleep duration of 6-7 hours, the odds of having low HDL-C were 3.68 (95% CI = 1.59-8.49) greater for

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individuals with sleep duration of <6 hours and 2.89 (95% CI = 1.10-7.61) greater for

individuals with sleep duration of >7 hours.

Conclusions: There was a U-shaped relationship between sleep duration and HDL-C levels.

Sleep duration over 7 hours or less than 6 hours increased the risk of low serum HDL-C

levels.

Keywords: Cholesterol/Lipid, Family Health, Metabolism, Prevention, Sleep Disorders.

Strengths and limitations of this study:

- The results of the study show that sleep duration of 6-7 hours per night reduced the risk of abnormal serum lipid profiles.
- This is the first study to explore the associations between sleep duration and lipid profiles in middle-aged and elderly Taiwanese population.
- We could not determine a causal relationship in our findings because this was a cross-sectional study.
- Lifestyle questionnaires were self-reported, which may limit the accuracy of measurements.

1	INTRODUCTION
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2	Several studies have identified that longer or shorter sleep duration may increase the
3	mortality and morbidity risks from diabetes mellitus ¹⁻³ , obesity ⁴ , hypertension ⁵ and coronary
4	heart disease ⁶ . Some studies have shown a U-shaped association between sleep duration and
5	major morbidities ³⁷⁸ . Dyslipidemia, such as high levels of total cholesterol (TC), low density
6	lipoprotein cholesterol (LDL-C), and triglycerides (TG) and low levels of high density
7	lipoprotein cholesterol (HDL-C), increases the risk of cardiovascular disease ⁹¹⁰ . There is a
8	lack of consensus on possible significant associations between sleep duration and serum lipid
9	profiles, and there have been few studies on this issue in the Taiwanese population. Therefore,
10	we investigated the relationship between sleep duration and levels of serum TC, LDL-C,
11	HDL-C, and TG in a community-based study in Taiwan.
12	MATERIALS AND METHODS
12 13	MATERIALS AND METHODS The present study was an observational, cross-sectional study. We enrolled 400
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20	levels, creatinine levels, and uric acid levels. The self-administered questionnaire included
21	items related to smoking, drinking, exercise, sleep, and other lifestyle habits.
22	Definitions and variables
23	Based on the NCEP (National Cholesterol Education Program) ATP III guidelines, we set
24	different cutoff points for each item of the serum lipid profiles (LDL-C: 130 mg/dL; TC: 200
25	mg/dL; HDL-C: 40 mg/dL; and TG: 150 mg/dL). Hyperlipidemia was defined as having
26	LDL-C, TC, or TG levels above the cutoff point; or use of lipid lowering agents.
27	The questionnaire about sleep included the question: "What was your daily average sleep
28	duration during the past month (not counting the times lying on the bed without sleeping)?".
29	We divided sleep duration into three groups: less than 6 hours (<6 hours), greater than or
30	equal to 6 hours but less than or equal to 7 hours (6-7 hours), and more than 7 hours (>7
31	hours). Hypertension was defined as self-reported hypertension or the use of anti-hypertensive
32	drugs. Diabetes was defined as self-reported diabetes mellitus or the use of oral anti-diabetic
33	drugs or insulin. Alcohol drinking was defined as drinking alcohol greater than or equal to 2
34	days per week. Regular exercise was defined as jogging, doing gymnastics, mountain
35	climbing, doing qigong, or other exercises on a habitual basis.
36	The institutional review board (IRB) approved the study and all patients provided written
37	informed consent.
38	

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39 Statistical analysis

40	Adequate blood samples and complete questionnaires were collected from all
41	participants. After data were collected, statistical analysis was conducted using the PASW
42	21.0 statistical package (SPSS Inc., Chicago, USA). A <i>p</i> -value <0.05 was considered
43	statistically significant. Our statistical analysis included multiple steps. First, characteristics
44	were compared across hyperlipidemia and sleep duration sub-groups using t-tests, one-way
45	ANOVA, and chi-square tests. Homogeneity examination was performed with ANOVA, and
46	heterogeneous variables were analyzed with the Brown-Forsythe test. Second, we measured
47	correlations between serum lipid profiles and related risk factors. Third, logistic regression
48	analyses were conducted to assess the relationship between sleep duration and risk of high
49	LDL-C, high TC, low HDL-C, and high TG levels; additional regression models were
50	adjusted for relevant covariates.
51	RESULTS
52	The characteristics of study subjects with and without hyperlipidemia are shown in Table
53	1. Among the included participants, 260 (65%) had hyperlipidemia. Body mass index, waist
54	circumference, waist-to-height ratio, AST levels, fasting plasma glucose levels, and uric acid
55	levels were significantly higher in the hyperlipidemia group.

Variables	No hyperlipidemia (n=140)	Hyperlipidemia (n=260)	p-value
Sex (male)	57 (40.7)	84 (32.3)	0.101
Age (years)	65.4 ± 8.4	64.0 ± 8.4	0.072
Current smoking	9 (6.4)	34 (13.1)	0.043
Alcohol drinking	20 (14.3)	55 (21.2)	0.107
Regular exercise	116 (83.2)	211 (81.2)	0.681
Hypertension	64 (45.7)	137 (52.7)	0.209
Diabetes	28 (19.7)	56 (21.5)	0.238
LDL-C (mg/dL)	97.6 ± 17.7	129.6 ± 32.6	< 0.001
TC (mg/dL)	169.8 ± 21.1	211.9 ± 33.2	< 0.001
HDL-C (mg/dL)	55.4 ± 12.8	53.9 ± 14.5	0.161
TG (mg/dL)	84.5 ± 29.3	142.3 ± 71.6	< 0.001
Sleep (hours)	-6.2 ± 1.2	6.2 ± 1.3	0.276
SBP (mmHg)	128.0 ± 16.0	130.2 ± 17.0	0.267
DBP (mmHg)	76.1 ± 11.3	77.1 ± 12.2	0.592
BMI (kg/m ²)	23.7 ± 3.4	25.0 ± 3.6	0.001
WC (cm)	82.9 ± 8.5	86.3 ± 10.1	0.001
WHtR	0.5 ± 0.1	0.5 ± 0.1	0.003
ALT (mg/dL)	20.3 ± 8.9	23.9 ± 14.6	0.032
Creatinine (mg/dL)	0.8 ± 0.6	0.8 ± 0.3	0.408
FPG (mg/dL)	92.2 ± 19.1	98.4 ± 28.5	0.028
Uric acid (mg/dL)	5.4 ± 1.3	5.9 ± 1.4	0.001

57 Data expressed as mean \pm SD for continuous variables and n (%) for categorical variables.

58 Abbreviations: TC = total cholesterol; TG = triglyceride; LDL-C = low density lipoprotein;

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59 HDL-C = high density lipoprotein; Sleep = sleep duration; ALT = alanine transaminase; WC

60 = waist circumference; WHtR = waist to height ratio; BMI = body mass index; FPG = fasting

61 plasma glucose; SBP = systolic blood pressure; DBP = diastolic blood pressure.

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The characteristics of study subjects categorized by sleep duration are shown in Table 2. There were 100 participants in the ≤ 6 hours group, 255 in the 6-7 hours group, and 45 in the >7 hours group. Low HDL-C levels significantly higher in the >7 hours group (31.1%) than other two groups (9.8% for the 6-7 hours group and 13% for the <6 hours group); post-hoc tests also showed the difference was significant.

Variable	<6 hours (n = 100)	6-7 hours (n =	>7 hours (n =	p-value
		255)	45)	
Sex (male)	30 (30)	88 (34.5)	23 (51.1)	0.044
Age (years)	65.2 ± 7.6	64.1 ± 8.3	65.1 ± 10.6	0.530
High LDL-C	33 (33)	87 (34.1)	15 (33.3)	0.978
High TC	49 (49)	125 (49.0)	21 (47.7)	0.941
Low HDL-C	13 (13)	25 (9.8)	14 (31.1)	0.001
High TG	22 (22)	58 (22.7)	14 (31.1)	0.437
Alcohol drinking	15 (15)	52 (20.4)	11 (24.4)	0.346
Current Smoking	10 (10)	27 (10.6)	6 (13.3)	0.828
Regular exercise	80 (80)	209 (81.9)	39 (86.4)	0.642
Hypertension	47 (47)	125 (49.0)	29 (64.4)	0.122
Diabetes	25 (25)	45 (17.6)	9 (20.0)	0.293
Dyslipidemia	69 (69)	161 (63.1)	30 (66.7)	0.564
SBP (mmHg)	129.9 ± 16.5	128.6 ± 16.7	133.3 ± 25.2	0.216
DBP (mmHg)	76.4 ± 13.0	76.6 ± 11.8	78.9 ± 9.13	0.436
BMI (kg/m ²)	24.3 ± 3.9	24.6 ± 3.4	25.1 ± 3.6	0.394
WC (cm)	84.7 ± 10.4	84.8 ± 9.6	87.4 ± 8.6	0.235
WHtR	0.5 ± 0.1	0.6 ± 0.1	0.6 ± 0.1	0.417
ALT (mg/dL)	22.5 ± 14.0	22.3 ± 11.94	22.7 ± 15.9	0.530
Creatinine (mg/dL)	0.8 ± 0.3	0.8 ± 0.3	1.0 ± 0.9	0.920
FPG (mg/dL)	98.2 ± 28.2	94.8 ± 19.3	100.3 ± 45.1	0.519
Uric acid (mg/dL)	5.6 ± 1.3	5.8 ± 1.4	6.1 ± 1.4	0.101

Abbreviations: High TC = total cholesterol $\geq 200 \text{ (mg/d)}$; High TG = triglyceride $\geq 150 \text{ (mg/d)}$;

70 High LDL-C = low density lipoprotein \geq 130 (mg/d); Low HDL-C = high density

71 lipoprotein \leq 40 (mg/d); ALT = alanine transaminase; WC = waist circumference; WHtR =

72 waist to height ratio; BMI = body mass index; FPG = fasting plasma glucose; SBP = systolic

73 blood pressure; DBP = diastolic blood pressure.

74	Figures 1 show comparisons of serum lipid profiles by sleep duration for two age groups,
75	age equal to or greater than 65 years old (elderly group) and all others (middle age group).
76	Abnormal total cholesterol levels had highest prevalence (around 50%) among the four items;
77	there were no significant differences by sleep duration (Figure 1A). The average prevalence of
78	high LDL-C level was about one-third, and prevalence gradually increased with longer sleep
79	duration in middle age group but decreased with longer sleep duration in the elderly group
80	(Figure 1B). There was a U-shaped (or J-shaped) distribution in the association of low HDL-C
81	levels with sleep duration; both age groups showed significantly higher prevalence of low
82	HDL-C if sleep duration was <6 hours or >7 hours (Figure 1D). Abnormal triglyceride levels
83	by sleep duration in the middle age group also appeared U-shaped (Figure 1C).
84	Spearman's correlation test showed low correlation among the serum lipid profile
85	variables, except HDL-C with sex and waist circumference (Table 3). However, HDL-C was
86	the only variable of the serum lipid profile significantly correlated with sleep duration.

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	TC	LDL	HDL	
Sex	0.247*	0.159*	0.365*	
Age (years)	-0.098*	-0.089	-0.072	
Current smoking	0.053	0.047	-0.133*	
Alcohol	0.089	0.076	0.016	
Exercise	-0.052	-0.037	-0.089	
BMI (kg/m ²)	0.007	0.38	-0.263*	
FPG (mg/dL)	-0.126	-0.127*	-0.255*	
WC (cm)	-0.04	0.005	-0.362*	
Sleep (hours)	-0.04	0.007	-0.122*	
*P-value <0.05				
Data are correlation coef	ficients.			
Abbreviations: WC=wai	st circumferenc	e; BMI=body ma	ass index; FPG=f	asting
glucose; Sleep= sleep du	ration.			

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93	Multivariable logistic regression model results are shown in Table 4. Model 1 assessed
94	the crude odds ratio of lipid profiles by sleep duration; Model 2 adjusted for age; Model 3
95	adjusted for age and waist circumference; and Model 4 adjusted for age, waist circumference,
96	and other traditional factors that influence lipid levels, including sex, alcohol drinking,
97	exercise, and smoking. The 6–7-hour sleep duration group was set as the reference category.
98	The p-value of the odds ratio were all above 0.05 (95% confidence interval including 1) when
99	the outcome was high TC levels, high LDL-C levels, or high TG levels; the odds ratios were
100	all significant in Models 1 through 4 when the dependent variable was low HDL-C levels.

		Sleep <6 hours	Sleep 6-7 hours	Sleep >7 hour
		(n=100)	(n=255)	(n=45)
		Tota	l cholesterol	
	Model 1	1.02 (0.54-1.92)	1.00	1.10 (0.54-2.22
	Model 2	0.99 (0.52-1.88)	1.00	1.10 (0.52-2.24
	Model 3	1.01 (0.53-1.93)	1.00	1.13 (0.56-2.3)
	Model 4	0.90 (0.45-1.80)	1.00	0.95 (0.44-2.02
			LDL-C	
	Model 1	1.04 (0.53-2.03)	1.00	0.99 (0.47-2.08
	Model 2	1.02 (0.52-2.00)	1.00	0.99 (0.47-2.10
	Model 3	1.05 (0.53-2.07)	1.00	1.03 (0.49-2.19
	Model 4	1.001(0.50-2.01)	1.00	0.94 (0.43-2.04
			HDL-C	
	Model 1	3.81 (1.80-8.05)*	1.00	3.02 (1.28-7.14
	Model 2	3.75 (1.77 <mark>-7.93)</mark> *	1.00	3.02 (1.28-7.16
	Model 3	3.62 (1.63-8.00)*	1.00	2.93 (1.17-7.34
	Model 4	3.68 (1.59-8.49)*	1.00	2.89 (1.10-7.61
		Tr	iglyceride	
	Model 1	0.65 (0.33-1.31)	1.00	0.63 (0.28-1.37
	Model 2	0.65 (0.33-1.31)	1.00	0.63 (0.27-1.38
	Model 3	0.72 (0.35-1.46)	1.00	0.69 (0.31-1.35
	Model 4	0.64 (0.31-1.34)	1.00	0.60 (0.26-1.39
102	Data are expressed	as odds ratio (OR) and 9	95% confidence interva	al (CI))
103	*P-value < 0.05			
104	†Model 1: unadjust	ed; Model 2: multiple lo	gistic regression adjus	ted for age; Model
105	multiple logistic reg	gression adjusted for age	and waist circumferen	nce; Model 4: mult
106	logistic regression a	adjusted for age, sex, wa	ist circumference, alco	hol drinking, exerc
107	smoking.			
108	Abbreviations: TC=	total cholesterol; TG=tr	iglyceride; LDL-C=lov	w density lipoprote
109	HDL-C= high dens	ity lipoprotein.		
			12	

DISCUSSION

111	In our study, traditional risk factors, such as high body mass index, high waist
112	circumference, high fasting plasma glucose levels, and high uric acid levels, were
113	significantly associated with abnormal lipid values. Elevated AST levels may be associated
114	with non-alcoholic liver disease. We found a U-shaped association between sleep duration and
115	low HDL-C levels, and logistic regression models showed that sleep duration was
116	significantly associated with low HDL-C levels regardless of adjustment for potential
117	confounders.
118	Several studies have reported associations between serum lipid profiles and sleep
119	duration, but the results of these studies have been inconsistent ¹¹⁻¹⁶ . Choi et al. ¹¹ collected
120	data from 4,222 Korean participants over the age of 60 and found a U-shaped association
121	between low HDL-C levels and high triglyceride levels, which were similar to the results of
122	our study. Both short and long sleep durations were related to an increased risk of metabolic
123	syndrome and sleep duration of 7 hours demonstrated the lowest prevalence of metabolic
124	syndrome in this study. Hall et al. ¹² reported that sleep duration was independently associated
125	with three components of metabolic syndrome: abdominal obesity, elevated serum glucose
126	levels, and elevated triglyceride levels; the optimal sleep duration for preventing metabolic
127	syndrome determined by this study was 7 to 8 hours per night. Bjorvatn et al. ¹³ demonstrated
128	that cholesterol levels, triglyceride levels, and blood pressure were higher in subjects with short

129	sleep duration. Another study ¹⁴ revealed that HDL-C levels decreased with short and long
130	sleep duration among normotensive, but not hypertensive, women.
131	The logistic regression models of low HDL-C level adjusted for age, sex, waist
132	circumference, alcohol drinking, exercise, and smoking. To our knowledge, the prevalence of
133	dyslipidemia is proportional to age and different in men and women. The association between
134	waist circumference and dyslipidemia has been confirmed in some studies ^{17 18} . Lifestyle
135	factors have been shown to influence serum lipid or lipoprotein levels. For example, smoking
136	decreases HDL-C levels and increases TG levels, whereas alcohol consumption increases the
137	levels of both. Exercise increases HDL-C levels and decreases TG levels. In addition, alcohol
138	consumption is reported to decrease LDL-C levels ^{19 20} . Even after adjusting for these
139	potential confounding factors, our logistic regression models showed significant differences
140	in odds of low HDL-C levels between sleep duration groups.
141	The influence that shorter sleep duration has on body weight and dyslipidemia has become
142	clearer in recent years. Sleep restriction is associated with hormone imbalance; it reduces leptin
143	(an appetite suppressant) and elevates ghrelin levels (an appetite stimulant) ⁴²¹ , which may
144	contribute to increased body weight and lead to dyslipidemia. The biochemical mechanism for
145	the relationship between longer sleeper and dyslipidemia has not been clearly confirmed; some
146	studies showed prolonged sleep duration may be associated with glucose intolerance and
147	diabetes ¹²² . Physical performance, such as reduced energy consumption due to increased time

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> in bed, may affect obesity; one study explained this relationship by showing that longer sleep duration was related to less exercise 6 . Some studies have reported that the relative risk of mortality and morbidity was lowest when sleep duration was between 7-8 hours per night ^{3 6-8}, but other research has reported that 6-7 hours is more optimal ¹⁵. The cutoff point of our study was set at 6-7 hours; we found the volunteers in our study generally woke up early regardless of the amount of time they slept, and they often had spontaneous arousal from nocturnal sleep. This may be explained by physiological age-related changes in circadian modulation, homeostatic factors, cardiopulmonary function, and endocrine function ^{23 24}; or be due to underlying chronic diseases. In an attempt to recommend the optimal sleep duration for lowering morbidity based on epidemiological data, it is inferred that the optimal sleep duration will vary for different populations¹⁵. Limitations of this study should be mentioned. First, we could not determine a causal relationship in our findings because this was a cross-sectional study. A cohort study is required for determining more causal relationships, which we aim to complete in the future. Second, selection bias might exist because the volunteer participant selection was limited to one township, and participants were not selected randomly or with a stratified method from the population. This could limit the generalizability of these results. In addition, our sample size was relatively small so sampling bias should be considered. Third, we did not group the

167	population by underlying disease, such as diabetes or cerebrovascular disease, due to limited
168	sample size; the proper cutoff for lipid profiles may differ according to underlying diseases.
169	Furthermore, the HDL-C cutoff point differs by sex; in metabolic syndrome, it is set as 40
170	mg/dL for men and 50 mg/dL for women. The influence of sleep duration on lipid metabolism
171	may also differ between men and women; for example, a study by Kaneita et al. ¹⁵ of 1,666
172	men and 2,329 women aged 20 years or older from Japan showed that HDL-C levels had a
173	U-shaped association with sleep duration in women but not men. Finally, the present study
174	estimated sleep duration only by a self-reported questionnaire, a more systematically biased
175	estimate than measured sleep duration.
176	CONCLUSIONS
177	In our study of a Taiwanese population, sleep duration of 6-7 hours per night reduced the
178	risk of abnormal serum lipid profiles. Sleep duration over 7 hours or less than 6 hours may
179	increase the risk of low serum HDL-C levels, although adjustment for risk factors attenuated
180	this relationship. Inappropriate sleep duration might be a potential risk factor for low HDL-C
181	levels, and adequate sleep duration may improve low HDL-C status. Lifestyle interventions,

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- including exercise and abstaining from alcohol and smoking, should be initiated early in
- 183 high-risk groups.

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Contributors

PL was involved in writing of the manuscript, collection and analyzed the data. KTC and YAL were involved in collection data. HHC and JYC contributed conceived, designed and performed the experiments, collected and analyzed the data, revising it critically for important intellectual content and final approval of the version to be submitted.

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 - Competing interests None declared.
 - Ethics approval The study was approved by Chang-Gung Medical Foundation Institutional
- Review Board (102-2304B), and written informed consent was given by all the participants
- before enrollment.
- Data sharing statement No additional data are available.

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Figure1 Prevalence of serum lipid profiles by sleep duration between two age groups (middle age: 50-65 years old; elderly: \geq 65 years old)

Figure1 Prevalence of serum lipid profiles by sleep duration between two age groups (middle age: 50-65 years old; elderly: ≥65 years old)

256x200mm (150 x 150 DPI)

STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of cross-sectional studies

Section/Topic	ltem #	Recommendation	Reported on page #
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract	1-2
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	2-3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4
Objectives	Objectives 3 State specific objectives, including any prespecified hypotheses		4
Methods			
Study design	4	Present key elements of study design early in the paper	4
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data 4 collection	
Participants	6	6 (<i>a</i>) Give the eligibility criteria, and the sources and methods of selection of participants NA	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	5
Data sources/	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe 4-5	
measurement	easurement comparability of assessment methods if there is more than one group		
Bias	9	Describe any efforts to address potential sources of bias	NA
Study size	10	Explain how the study size was arrived at	4
Quantitative variables	ititative variables 11 Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why		6
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	6
		(b) Describe any methods used to examine subgroups and interactions	NA
		(c) Explain how missing data were addressed	NA
		(d) If applicable, describe analytical methods taking account of sampling strategy	NA

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		(e) Describe any sensitivity analyses	NA
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility,	NA
		confirmed eligible, included in the study, completing follow-up, and analysed	
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	NA
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	7-9
		(b) Indicate number of participants with missing data for each variable of interest	NA
Outcome data	15*	Report numbers of outcome events or summary measures	10-12
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence	12-13
		interval). Make clear which confounders were adjusted for and why they were included	
		(b) Report category boundaries when continuous variables were categorized	NA
		(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	NA
Discussion			
Key results	18	Summarise key results with reference to study objectives	14
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	16-17
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	14-17
Generalisability	21	Discuss the generalisability (external validity) of the study results	NA
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on	18
		which the present article is based	

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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Association between self-reported sleep duration and serum lipid profile in a middle-aged and elderly population in Taiwan: A community-based, cross-sectional study

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Keywords:	Cholesterol/Lipid, Family Health, Metabolism, Prevention, Sleep Disorders

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elderly population in Taiwan: A community-based, cross-sectional study

• Running title

Association between sleep duration and serum lipid profile

Author names and affiliations

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ABSTRACT

Objectives: The association between sleep duration and serum lipid profile in the middle-aged and elderly is unclear. The aim of this study was to investigate and evaluate the relationships between sleep duration and levels of serum total cholesterol (TC), LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), and triglycerides (TG) in these populations. **Design:** Cross-sectional observational study.

Setting: Community-based investigation in Guishan township of northern Taiwan.

Participants: A total of 400 community-dwelling middle-aged and elderly individuals were

enrolled. All participants underwent a baseline assessment in 2014, which included

anthropometrics, blood samples, and self-administered questionnaires. Participants were classified into three groups based on their sleep duration.

Outcome measures: Multivariate logistic regression was used to obtain odds ratios (ORs) and 95% confidence intervals (CIs) to assess the relationship between sleep duration and lipid profiles.

Results: Participant mean age was 64.5 years old and 35.3% were men. Subjects with longer (>7 hours) and shorter (<6 hours) nightly sleep duration had a higher prevalence of low HDL-C levels (HDL <40 mg/dL) than those with moderate sleep duration (6-7 hours).

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Multivariate logistic regression revealed that, compared to individuals with sleep duration of 6-7 hours, the odds of having low HDL-C were 3.68 (95% CI = 1.59-8.49) greater for individuals with sleep duration of <6 hours and 2.89 (95% CI = 1.10-7.61) greater for individuals with sleep duration of >7 hours.

Conclusions: There was a U-shaped relationship between sleep duration and HDL-C levels. Sleep duration over 7 hours or less than 6 hours increased the risk of low serum HDL-C levels.

Keywords: Cholesterol/Lipid, Family Health, Metabolism, Prevention, Sleep Disorders.

Strengths and limitations of this study:

- The results of the study show that sleep duration of 6-7 hours per night reduced the risk of abnormal serum lipid profiles.
- This is the first study to explore the associations between sleep duration and lipid profiles in middle-aged and elderly Taiwanese population.
- We could not determine a causal relationship in our findings because this was a cross-sectional study.
- Lifestyle questionnaires were self-reported, which may limit the accuracy of measurements.

INTRODUCTION

2	As we known, the most aged adult suffers lipid profile disorder. ¹ Moreover, the different
3	levels in percentage of lipid disorders may have association between demography variables
4	(i.e. as age and gender). ¹ A study also found significant association between high prevalence
5	of dyslipidemia and risk factors, such as increasing age, smoking status, hypertension,
6	diabetes, and body mass index. ² Several studies have identified that longer or shorter sleep
7	duration may increase the mortality and morbidity risks from diabetes mellitus $^{3-5}$, obesity 6 ,
8	hypertension ⁷ and coronary heart disease ⁸ . Some studies have shown a U-shaped association
9	between sleep duration and major morbidities ^{5 9 10} . Cardiovascular disease has been a leading
10	cause of death worldwide. ¹¹ Dyslipidemia, such as high levels of total cholesterol (TC), low
11	density lipoprotein cholesterol (LDL-C), and triglycerides (TG) and low levels of high density
12	lipoprotein cholesterol (HDL-C), increases the risk of cardiovascular disease ¹² , and hence
13	remains an important issue in the field of health promotion and disease prevention. Previous
14	studies show age-associated alterations in the level, composition, and function of lipid profiles,
15	including an inverted U-shaped quadratic trajectory for TC, LDL-C, and TG ^{13 14} , and a
16	decrease anti-oxidative ability. ¹⁵ Other than age, factors that have been suggested to be related
17	to lipid profile levels include BMI, body composition, diet, and cardiorespiratory fitness. ^{13 14}
18	Though some study suggests that sleep deprivation may bring changes to plasma lipid

19 ¹⁶, there is a lack of consensus on possible significant associations between sleep duration and

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20	serum lipid profiles, and even fewer data available in the Taiwanese population ¹⁷ . Therefore,
21	we investigated the relationship between sleep duration and levels of serum TC, LDL-C,
22	HDL-C, and TG in a community-based study in Taiwan.
23	MATERIALS AND METHODS
24	Study participants
25	The present study was an observational, cross-sectional study. We enrolled 400
26	participants, including 141 men and 259 women. The inclusion criteria included (1) the
27	residents aged over 50 years old; (2) the residents living in Guishan township. 12 people aged
28	younger than 50 years old were excluded from the study. Subjects were excluded if (1) the
29	residents could not complete the full examinations or missing data for age, sex,
30	anthropometric values and blood test results; (2) the residents were functionally dependent; (3)
31	the residents were unable to adequately communicate with the interviewers; (4) the residents
32	had major illness recently; (5) the residents with known sleep disorders.
33	Data collection
34	Data collection comprised two parts: a physical status examination and a
35	self-administered questionnaire. For the physical status examination, height, weight,
36	abdominal circumference, blood pressure, and blood samples were collected for all
37	participants. Blood samples included the following items: lipid profiles (TC, LDL-C, HDL-C,

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38	and TG), aspartate aminotransferase (AST) levels, fasting plasma glucose (FPG) levels,			
39	creatinine levels, and uric acid levels. The self-administered questionnaire included items			
40	related to smoking, drinking, exercise, sleep, and other lifestyle habits. All participants were			
41	performed in the same day recorded from March 2014 to August 2014.			
42	Definitions and variables			
43	Based on the NCEP (National Cholesterol Education Program) ATP III guidelines, we set			
44	different cutoff points for each item of the serum lipid profiles (LDL-C: 130 mg/dL; TC: 200			
45	mg/dL; HDL-C: 40 mg/dL; and TG: 150 mg/dL). Hyperlipidemia was defined as having			
46	LDL-C, TC, or TG levels above the cutoff point.			
47	Applied from Pittsburgh Sleep Quality Index(PSQI) ¹⁷ , the questionnaire about sleep			
48	included the question: "What was your daily average sleep duration during the past month			
49	(not counting the times lying on the bed without sleeping)?". We divided sleep duration into			
50	three groups: less than 6 hours (<6 hours), greater than or equal to 6 hours but less than or			
51	equal to 7 hours (6-7 hours), and more than 7 hours (>7 hours). Hypertension was defined as			
52	self-reported hypertension or the use of anti-hypertensive drugs. Diabetes was defined as			
53	self-reported diabetes mellitus or the use of oral anti-diabetic drugs or insulin. Alcohol			
54	drinking was defined as drinking alcohol greater than or equal to 2 days per week. Regular			
55	exercise was defined as exercise times were greater or equal to 2 days per week. Current			
56	smoking was defined as literal meaning.			

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57	The institutional review board (IRB) approved the study and all patients provided written
58	informed consent.
59	Statistical analysis
60	Adequate blood samples and complete questionnaires were collected from all
61	participants. After data were collected, statistical analysis was conducted using the SPSS 21.0
62	statistical package (SPSS Inc., Chicago, USA). A <i>p</i> -value <0.05 was considered statistically
63	significant. Our statistical analysis included multiple steps. First, characteristics were
64	compared across hyperlipidemia and sleep duration sub-groups using t-tests, one-way
65	ANOVA, and chi-square tests. Homogeneity examination was performed with ANOVA, and
66	heterogeneous variables were analyzed with the Brown-Forsythe test. Second, we measured
67	correlations between serum lipid profiles and related risk factors. Third, logistic regression
68	analyses were conducted to assess the relationship between sleep duration and risk of high
69	LDL-C, high TC, low HDL-C, and high TG levels; additional regression models were
70	adjusted for relevant covariates.
71	RESULTS
72	The characteristics of study subjects with and without hyperlipidemia are shown in Table
73	1. Among the included participants, 260 (65%) had hyperlipidemia. Body mass index, waist
74	circumference, waist-to-height ratio, AST levels, fasting plasma glucose levels, and uric acid
75	levels were significantly higher in the hyperlipidemia group.
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Variables	No hyperlipidemia (n=140)	Hyperlipidemia (n=260)	p-value
Sex (male)	57 (40.7)	84 (32.3)	0.101
Age (years)	65.4 ± 8.4	64.0 ± 8.4	0.072
Current smoking	9 (6.4)	34 (13.1)	0.043
Alcohol drinking	20 (14.3)	55 (21.2)	0.107
Regular exercise	116 (83.2)	211 (81.2)	0.681
Hypertension	64 (45.7)	137 (52.7)	0.209
Diabetes	28 (19.7)	56 (21.5)	0.238
LDL-C (mg/dL)	97.6 ± 17.7	129.6 ± 32.6	< 0.001
TC (mg/dL)	169.8 ± 21.1	211.9 ± 33.2	< 0.001
HDL-C (mg/dL)	55.4 ± 12.8	53.9 ± 14.5	0.161
TG (mg/dL)	84.5 ± 29.3	142.3 ± 71.6	< 0.001
Sleep (hours)	6.2 ± 1.2	6.2 ± 1.3	0.276
SBP (mmHg)	128.0 ± 16.0	130.2 ± 17.0	0.267
DBP (mmHg)	76.1 ± 11.3	77.1 ± 12.2	0.592
BMI (kg/m ²)	23.7 ± 3.4	25.0 ± 3.6	0.001
WC (cm)	82.9 ± 8.5	86.3 ± 10.1	0.001
WHtR	0.52 ± 0.05	0.54 ± 0.06	0.003
ALT (mg/dL)	20.3 ± 8.9	23.9 ± 14.6	0.032
Creatinine (mg/dL)	0.8 ± 0.6	0.8 ± 0.3	0.408
FPG (mg/dL)	92.2 ± 19.1	98.4 ± 28.5	0.028
Uric acid (mg/dL)	5.4 ± 1.3	5.9 ± 1.4	0.001

Data expressed as mean \pm SD for continuous variables and n (%) for categorical variables.

Abbreviations: TC = total cholesterol; TG = triglyceride; LDL-C = low density lipoprotein;

HDL-C = high density lipoprotein; Sleep = sleep duration; ALT = alanine transaminase; WC

= waist circumference; WHtR = waist to height ratio; BMI = body mass index; FPG = fasting

plasma glucose; SBP = systolic blood pressure; DBP = diastolic blood pressure.
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The characteristics of study subjects categorized by sleep duration are shown in Table 2. There were 100 participants in the ≤ 6 hours group, 255 in the 6-7 hours group, and 45 in the >7 hours group. Low HDL-C levels significantly higher in the >7 hours group (31.1%) than other two groups (9.8% for the 6-7 hours group and 13% for the <6 hours group); post-hoc tests also showed the difference was significant (significant difference between <6 hours group and >7 hours group(p value = 0.009) \cdot 6-7 hours and >7 hours(p value=0.001). ΪΩΠΜ^Δ

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Variable	<6 hours (n = 100)	6-7 hours (n =	>7 hours (n =	p-value
		255)	45)	
Sex (male)	30 (30)	88 (34.5)	23 (51.1)*†	0.044
Age (years)	65.2 ± 7.6	64.1 ± 8.3	65.1 ± 10.6	0.530
LDL-C (mg/dL)	115.8 ± 30.4	119.2 ± 31.3	119.6 ± 40.0	0.643
TC (mg/dL)	197.2 ± 34.9	197.0 ± 34.2	198.0 ± 45.4	0.984
HDL-C (mg/dL)	57.4 ± 15.8	53.9 ± 12.5	51.2 ± 16.4	0.025
TG (mg/dL)	120.4 ± 61.8	119.8 ± 62.7	138.5 ± 88.6	0.207
High LDL-C	33 (33)	87 (34.1)	15 (33.3)	0.978
High TC	49 (49)	125 (49.0)	21 (47.7)	0.941
Low HDL-C	13 (13)	25 (9.8)	14 (31.1)**	0.001
High TG	22 (22)	58 (22.7)	14 (31.1)	0.437
Alcohol drinking	15 (15)	52 (20.4)	11 (24.4)	0.346
Current Smoking	10 (10)	27 (10.6)	6 (13.3)	0.828
Regular exercise	80 (80)	209 (81.9)	39 (86.4)	0.642
Hypertension	47 (47)	125 (49.0)	29 (64.4)	0.122
Diabetes	25 (25)	45 (17.6)	9 (20.0)	0.293
Hyperlipidemia	69 (69)	161 (63.1)	30 (66.7)	0.564
SBP (mmHg)	129.9 ± 16.5	128.6 ± 16.7	133.3 ± 25.2	0.216
DBP (mmHg)	76.4 ± 13.0	76.6 ± 11.8	78.9 ± 9.13	0.436
BMI (kg/m ²)	24.3 ± 3.9	24.6 ± 3.4	25.1 ± 3.6	0.394
WC (cm)	84.7 ± 10.4	84.8 ± 9.6	87.4 ± 8.6	0.235
WHtR	0.5 ± 0.1	0.6 ± 0.1	0.6 ± 0.1	0.417
ALT (mg/dL)	22.5 ± 14.0	22.3 ± 11.94	22.7 ± 15.9	0.530
Creatinine (mg/dL)	0.8 ± 0.3	0.8 ± 0.3	1.0 ± 0.9	0.920
FPG (mg/dL)	98.2 ± 28.2	94.8 ± 19.3	100.3 ± 45.1	0.519
Uric acid (mg/dL)	5.6 ± 1.3	5.8 ± 1.4	6.1 ± 1.4	0.101



p-value < 0.05 compared with <6 hours group, [†] p-value < 0.05 compared with 6-7 hours group.

Abbreviations: High TC = total cholesterol $\geq 200 \text{ (mg/d)}$; High TG = triglyceride $\geq 150 \text{ (mg/d)}$;

High LDL-C = low density lipoprotein \geq 130 (mg/d); Low HDL-C = high density

lipoprotein \leq 40 (mg/d); ALT = alanine transaminase; WC = waist circumference; WHtR =

waist to height ratio; BMI = body mass index; FPG = fasting plasma glucose; SBP = systolic

1 2 3 4 5 6 7 8 9 10 11 2 3 4 5 6 7 8 9 10 11 2 3 4 5 6 7 8 9 10 11 2 3 4 5 6 7 8 9 0 11 2 2 3 2 4 5 6 7 28 9 031 22 3 4 5 6 7 28 9 031 22 3 4 5 6 7 8 9 0 11 12 3 4 5 6 7 8 9 0 11 12 3 4 5 6 7 8 9 0 11 12 3 3 4 5 6 7 8 9 0 11 12 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 1 12 2 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 8 9 0 1 2 2 3 4 5 6 7 8 9 0 31 2 3 3 4 5 6 7 7 8 9 0 1 2 2 3 4 5 6 7 7 8 9 0 1 2 2 3 4 5 6 7 7 8 9 0 1 2 2 3 4 5 6 7 7 8 9 0 1 2 2 3 4 5 6 5 7 8 9 0 1 2 2 3 4 5 5 6 7 7 8 9 0 1 2 3 3 4 5 5 6 7 7 8 9 0 1 2 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5	96	blood pressure; DBP = diastolic blood pressure
55 56 57 58 59 60		11
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97	Figures 1 show comparisons of serum lipid profiles by sleep duration for two age groups,
98	age equal to or greater than 65 years old (elderly group, 168 people) and all others (middle
99	age group, 232 people). Abnormal total cholesterol levels had highest prevalence (around
100	50%) among the four items; there were no significant differences by sleep duration (Figure
101	1A). The average prevalence of high LDL-C level was about one-third, and prevalence
102	gradually increased with longer sleep duration in middle age group but decreased with longer
103	sleep duration in the elderly group (Figure 1B). There was a U-shaped (or J-shaped)
104	distribution in the association of low HDL-C levels with sleep duration; both age groups
105	showed significantly higher prevalence of low HDL-C if sleep duration was <6 hours or >7
106	hours (Figure 1C). Abnormal triglyceride levels by sleep duration in the middle age group
107	also appeared U-shaped (Figure 1D).

Multivariable logistic regression model results are shown in Table 3. Model 1 assessed the crude odds ratio of lipid profiles by sleep duration; Model 2 adjusted for age; Model 3 adjusted for age and waist circumference; and Model 4 adjusted for age, waist circumference, and other traditional factors that influence lipid levels, including sex, alcohol drinking, exercise, and smoking. The 6–7-hour sleep duration group was set as the reference category. The p-value of the odds ratio were all above 0.05 (95% confidence interval including 1) when the outcome was high TC levels, high LDL-C levels, or high TG levels; the odds ratios were all significant in Models 1 through 4 when the dependent variable was low HDL-C levels.

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(n=100) $(n=255)$ $(n=45)$ Total cholesterol Model 1 1.02 (0.54-1.92) 1.00 1.10 (0.54-2.22) Model 2 0.99 (0.52-1.88) 1.00 1.10 (0.52-2.24) Model 3 1.01 (0.53-1.93) 1.00 0.95 (0.44-2.02) Model 4 0.90 (0.45-1.80) 1.00 0.99 (0.47-2.08) Model 1 1.04 (0.53-2.03) 1.00 0.99 (0.47-2.10) Model 2 1.02 (0.52-2.00) 1.00 0.99 (0.47-2.10) Model 3 1.05 (0.53-2.07) 1.00 1.03 (0.49-2.19) Model 4 1.001(0.50-2.01) 1.00 0.94 (0.43-2.04) HDL-C Model 1 3.81 (1.80-8.05)* 1.00 3.02 (1.28-7.14) Model 2 3.75 (1.77-7.93)* 1.00 3.02 (1.28-7.14) Model 3 3.62 (1.63-8.00)* 1.00 2.89 (1.10-7.61) Triglyceride Model 3 3.62 (1.63-8.00)* 1.00 2.89 (1.10-7.61) 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37) Model 3 0.72 (0.35-1.46) 1.00			Sleep <6 hours	Sleep 6-7 hours	Sleep >7 hours
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Model 3 1.05 (0.53-2.07) 1.00 1.03 (0.49-2.19) Model 4 1.001(0.50-2.01) 1.00 0.94 (0.43-2.04) HDL-C HDL-C Model 1 3.81 (1.80-8.05)* 1.00 3.02 (1.28-7.14)* Model 2 3.75 (1.77-7.93)* 1.00 3.02 (1.28-7.16)* Model 3 3.62 (1.63-8.00)* 1.00 2.93 (1.17-7.34)* Model 4 3.68 (1.59-8.49)* 1.00 2.89 (1.10-7.61)* Triglyceride Model 1 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37) Model 2 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37) Model 3 0.72 (0.35-1.46) 1.00 0.69 (0.31-1.35) Model 3 0.72 (0.35-1.46) 1.00 0.69 (0.26-1.39) Model 4 0.64 (0.31-1.34) 1.00 0.60 (0.26-1.39) Model 1: unadjusted; Model 2: multiple logistic regression adjusted for age; Model 3 1.01 1.02 1.02 Model 1: unadjusted; Model 2: multiple logistic regression adjusted for age and waist circumference; Model 4: multiple 1.00 1.00 1.00 1.00 1.00 1.00 1.00 1.00 1.00 1.00 1.00 <t< td=""><td></td><td>Model 2</td><td>1.02 (0.52-2.00)</td><td>1.00</td><td>0.99 (0.47-2.10)</td></t<>		Model 2	1.02 (0.52-2.00)	1.00	0.99 (0.47-2.10)
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Model 3 $3.62 (1.63-8.00)^*$ 1.00 $2.93 (1.17-7.34)^2$ Model 4 $3.68 (1.59-8.49)^*$ 1.00 $2.89 (1.10-7.61)^2$ Triglyceride Model 1 $0.65 (0.33-1.31)$ 1.00 $0.63 (0.28-1.37)$ Model 2 $0.65 (0.33-1.31)$ 1.00 $0.63 (0.27-1.38)$ Model 3 $0.72 (0.35-1.46)$ 1.00 $0.69 (0.31-1.35)$ Model 4 $0.64 (0.31-1.34)$ 1.00 $0.60 (0.26-1.39)$ 117 Data are expressed as odds ratio (OR) and 95% confidence interval (CI)) *P-value < 0.05 *P-value < 0.05 * * *Podel 1: unadjusted; Model 2: multiple logistic regression adjusted for age; Model 3 multiple logistic regression adjusted for age and waist circumference; Model 4: multiple logistic regression adjusted for age, sex, waist circumference, alcohol drinking, exerci smoking. * Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL -C= high density lipoprotein		Model 2	3.75 (1.77-7.93)*	1.00	3.02 (1.28-7.16)*
Model 4 $3.68 (1.59-8.49)^*$ 1.00 $2.89 (1.10-7.61)^*$ TriglycerideModel 1 $0.65 (0.33-1.31)$ 1.00 $0.63 (0.28-1.37)$ Model 2Model 2 $0.65 (0.33-1.31)$ 1.00 $0.63 (0.27-1.38)$ Model 3Model 3 $0.72 (0.35-1.46)$ 1.00 $0.69 (0.31-1.35)$ Model 4Model 4 $0.64 (0.31-1.34)$ 1.00 $0.60 (0.26-1.39)$ 117Data are expressed as odds ratio (OR) and 95% confidence interval (CI))118*P-value <0.05		Model 3	3.62 (1.63-8.00)*	1.00	2.93 (1.17-7.34)*
$\begin{tabular}{ c c c c c } \hline Triglyceride \\ \hline Model 1 & 0.65 & (0.33-1.31) & 1.00 & 0.63 & (0.28-1.37) \\ \hline Model 2 & 0.65 & (0.33-1.31) & 1.00 & 0.63 & (0.27-1.38) \\ \hline Model 3 & 0.72 & (0.35-1.46) & 1.00 & 0.69 & (0.31-1.35) \\ \hline Model 4 & 0.64 & (0.31-1.34) & 1.00 & 0.60 & (0.26-1.39) \\ \hline Data are expressed as odds ratio (OR) and 95% confidence interval (CI)) \\ *P-value < 0.05 \\ *P-value < 0.05 \\ *Model 1: unadjusted; Model 2: multiple logistic regression adjusted for age; Model 3 \\ multiple logistic regression adjusted for age and waist circumference; Model 4: multiple logistic regression adjusted for age, sex, waist circumference; Model 4: multiple logistic regression adjusted for age, sex, waist circumference, alcohol drinking, exerci smoking. \\ \hline Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein \\ \hline HDL -C= high density lipoprotein \\ \hline \end{tabular}$		Model 4	3.68 (1.59-8.49)*	1.00	2.89 (1.10-7.61)*
Model 1 $0.65 (0.33-1.31)$ 1.00 $0.63 (0.28-1.37)$ Model 2 $0.65 (0.33-1.31)$ 1.00 $0.63 (0.27-1.38)$ Model 3 $0.72 (0.35-1.46)$ 1.00 $0.69 (0.31-1.35)$ Model 4 $0.64 (0.31-1.34)$ 1.00 $0.60 (0.26-1.39)$ 117Data are expressed as odds ratio (OR) and 95% confidence interval (CI))*P-value < 0.05 *Model 1: unadjusted; Model 2: multiple logistic regression adjusted for age; Model 3multiple logistic regression adjusted for age and waist circumference; Model 4: multiplelogistic regression adjusted for age, sex, waist circumference, alcohol drinking, exercismoking.Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoproteinHDL -C= high density lipoprotein			Tr	iglyceride	
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Model 30.72 (0.35-1.46)1.000.69 (0.31-1.35)Model 40.64 (0.31-1.34)1.000.60 (0.26-1.39)117Data are expressed as odds ratio (OR) and 95% confidence interval (CI))*P-value <0.05		Model 2	0.65 (0.33-1.31)	1.00	0.63 (0.27-1.38)
Model 40.64 (0.31-1.34)1.000.60 (0.26-1.39)117Data are expressed as odds ratio (OR) and 95% confidence interval (CI))118*P-value <0.05		Model 3	0.72 (0.35-1.46)	1.00	0.69 (0.31-1.35)
 Data are expressed as odds ratio (OR) and 95% confidence interval (CI)) *P-value <0.05 †Model 1: unadjusted; Model 2: multiple logistic regression adjusted for age; Model 3 multiple logistic regression adjusted for age and waist circumference; Model 4: multiple logistic regression adjusted for age, sex, waist circumference, alcohol drinking, exerci smoking. Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL -C= high density lipoprotein 		Model 4	0.64 (0.31-1.34)	1.00	0.60 (0.26-1.39)
 *P-value <0.05 †Model 1: unadjusted; Model 2: multiple logistic regression adjusted for age; Model 3 multiple logistic regression adjusted for age and waist circumference; Model 4: multiple logistic regression adjusted for age, sex, waist circumference, alcohol drinking, exerci smoking. Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL-C= high density lipoprotein 	117	Data are expressed	as odds ratio (OR) and 9	5% confidence interva	al (CI))
 †Model 1: unadjusted; Model 2: multiple logistic regression adjusted for age; Model 3 multiple logistic regression adjusted for age and waist circumference; Model 4: multiple logistic regression adjusted for age, sex, waist circumference, alcohol drinking, exerci smoking. Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL-C= high density lipoprotein 	118	*P-value < 0.05			
 multiple logistic regression adjusted for age and waist circumference; Model 4: multiple logistic regression adjusted for age, sex, waist circumference, alcohol drinking, exerci smoking. Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL-C= high density lipoprotein 	119	†Model 1: unadjust	ed; Model 2: multiple lo	gistic regression adjus	ted for age; Model 3:
 logistic regression adjusted for age, sex, waist circumference, alcohol drinking, exerci smoking. Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL-C= high density lipoprotein 	120	multiple logistic rea	gression adjusted for age	and waist circumferen	nce; Model 4: multiple
 smoking. Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL-C= high density lipoprotein 	121	logistic regression a	adjusted for age, sex, wa	ist circumference, alco	bhol drinking, exercise
 Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low density lipoprotein HDL-C= high density lipoprotein 	122	smoking.			
HDL- C = high density linoprotein	123	Abbreviations: TC=	total cholesterol; TG=tr	iglyceride; LDL-C=lo	w density lipoprotein;
	124	HDL-C= high dens	ity lipoprotein.		
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DISCUSSION

126	In our study, traditional risk factors, such as high body mass index, high waist
127	circumference, high fasting plasma glucose levels, and high uric acid levels, were
128	significantly associated with abnormal lipid values. Elevated AST levels may be associated
129	with non-alcoholic liver disease. We found a U-shaped association between sleep duration and
130	low HDL-C levels, and logistic regression models showed that sleep duration was
131	significantly associated with low HDL-C levels regardless of adjustment for potential
132	confounders.
133	Several studies have reported associations between serum lipid profiles and sleep
134	duration, but the results of these studies have been inconsistent ¹⁸⁻²³ . Choi et al. ¹⁸ collected
135	data from 4,222 Korean participants over the age of 60 and found a U-shaped association
136	between low HDL-C levels and high triglyceride levels, which were similar to the results of
137	our study. Both short and long sleep durations were related to an increased risk of metabolic
138	syndrome and sleep duration of 7 hours demonstrated the lowest prevalence of metabolic
139	syndrome in this study. Hall et al. ¹⁹ reported that sleep duration was independently associated
140	with three components of metabolic syndrome: abdominal obesity, elevated serum glucose
141	levels, and elevated triglyceride levels; the optimal sleep duration for preventing metabolic
142	syndrome determined by this study was 7 to 8 hours per night. Bjorvatn et al. ²⁰ demonstrated
143	that cholesterol levels, triglyceride levels, and blood pressure were higher in subjects with short

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144	sleep duration. Another study ²¹ revealed that HDL-C levels decreased with short and long
145	sleep duration among normotensive, but not hypertensive, women.
146	The logistic regression models of low HDL-C level adjusted for age, sex, waist
147	circumference, alcohol drinking, exercise, and smoking. To our knowledge, the prevalence of
148	dyslipidemia is proportional to age and different in men and women. The association between
149	waist circumference and dyslipidemia has been confirmed in some studies ^{24 25} . Lifestyle
150	factors have been shown to influence serum lipid or lipoprotein levels. For example, smoking
151	decreases HDL-C levels and increases TG levels, whereas alcohol consumption increases the
152	levels of both ²⁶⁻²⁸ . Exercise increases HDL-C levels and decreases TG levels ^{29 30} . In addition,
153	alcohol consumption is reported to decrease LDL-C levels ^{31 32} . Even after adjusting for these
154	potential confounding factors, our logistic regression models showed significant differences
155	in odds of low HDL-C levels between sleep duration groups.
156	The influence that shorter sleep duration has on body weight and dyslipidemia has become
157	clearer in recent years. Sleep restriction is associated with hormone imbalance; it reduces leptin
158	(an appetite suppressant) and elevates ghrelin levels (an appetite stimulant) ^{6 33} , which may
159	contribute to increased body weight and lead to dyslipidemia. The biochemical mechanism for
160	the relationship between longer sleeper and dyslipidemia has not been clearly confirmed; some
161	studies showed prolonged sleep duration may be associated with glucose intolerance and
162	diabetes ^{3 34} . Physical performance, such as reduced energy consumption due to increased time

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163	in bed, may affect obesity; one study explained this relationship by showing that longer sleep
164	duration was related to less exercise ⁸ .
165	Some studies have reported that the relative risk of mortality and morbidity was lowest
166	when sleep duration was between 7-8 hours per night ^{5 8-10} , but other research has reported that
167	6-7 hours is more optimal ²² . The cutoff point of our study was set at 6-7 hours; we found the
168	volunteers in our study generally woke up early regardless of the amount of time they slept, and
169	they often had spontaneous arousal from nocturnal sleep. This may be explained by
170	physiological age-related changes in circadian modulation, homeostatic factors,
171	cardiopulmonary function, and endocrine function ^{35 36} ; or be due to underlying chronic
172	diseases. In an attempt to recommend the optimal sleep duration for lowering morbidity based
173	on epidemiological data, it is inferred that the optimal sleep duration will vary for different
174	populations ²² .
175	Limitations of this study should be mentioned. First, we could not determine a causal
176	relationship in our findings because this was a cross-sectional study. A cohort study is
177	required for determining more causal relationships, which we aim to complete in the future.
178	Second, selection bias might exist because the volunteer participant selection was limited to
179	one township, and participants were not selected randomly or with a stratified method from
180	the population. This could limit the generalizability of these results. In addition, our sample
181	size was relatively small so sampling bias should be considered. Third, we did not group the

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182	population by underlying disease, such as diabetes or cerebrovascular disease, due to limited
183	sample size; the proper cutoff for lipid profiles may differ according to underlying diseases.
184	Furthermore, the HDL-C cutoff point differs by sex; in metabolic syndrome, it is set as 40
185	mg/dL for men and 50 mg/dL for women. The influence of sleep duration on lipid metabolism
186	may also differ between men and women; for example, a study by Kaneita et al. ²² of 1,666
187	men and 2,329 women aged 20 years or older from Japan showed that HDL-C levels had a
188	U-shaped association with sleep duration in women but not men. Fourth, glycosylated
189	hemoglobin might be a more appropriate measurement of diabetes instead of self-reported
190	diabetes mellitus or the use of oral anti-diabetic drugs or insulin. Fifth, as a majority of other
191	similar studies in the literature covering examining effects of sleep duration on serum lipids,
192	diet is not factored for as a covariate. It may be explained by daily life diet is a diversified
193	condition so that diet is difficult merging into one variable and bringing to the regression model
194	for adjustment. Sixth, lipid-lowering therapy was an important factor influencing lipid levels,
195	but we did not considering about lipid-lowering medication while formulating the
196	questionnaire. Finally, the present study estimated sleep duration only by a self-reported
197	questionnaire, a more systematically biased estimate than measured sleep duration.
198	CONCLUSIONS
199	In our study of a Taiwanese population, sleep duration of 6-7 hours per night reduced the

200 risk of abnormal serum lipid profiles. Sleep duration over 7 hours or less than 6 hours may

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201	increase the risk of low serum HDL-C levels, although adjustment for risk factors attenuated
202	this relationship. Inappropriate sleep duration might be a potential risk factor for low HDL-C
203	levels, and adequate sleep duration may improve low HDL-C status. Lifestyle interventions,
204	including exercise and abstaining from alcohol and smoking, should be initiated early in
205	high-risk groups.

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206 Contributors

PL was involved in writing of the manuscript, collection and analyzed the data. KTC and
YAL were involved in collection data. IST provided statistical advice. HHC and JYC
contributed conceived, designed and performed the experiments, collected and analyzed the
data, revising it critically for important intellectual content and final approval of the version to
be submitted.

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- **Competing interests** None declared.
- 215 Ethics approval The study was approved by Chang-Gung Medical Foundation Institutional
- 216 Review Board (102-2304B), and written informed consent was given by all the participants
- 217 before enrollment.
- **Data sharing statement** No additional data are available.

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STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of cross-sectional studies

Section/Topic	ltem #	Recommendation	Reported on page #
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract	1-3
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	2-3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4-5
Objectives	3	State specific objectives, including any prespecified hypotheses	4-5
Methods			
Study design	4	Present key elements of study design early in the paper	5
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5
Participants	6	(a) Give the eligibility criteria, and the sources and methods of selection of participants	x
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	5-7
Data sources/	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe	5-6
measurement		comparability of assessment methods if there is more than one group	
Bias	9	Describe any efforts to address potential sources of bias	X
Study size	10	Explain how the study size was arrived at	5-6
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	6-7
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	7
		(b) Describe any methods used to examine subgroups and interactions	х
		(c) Explain how missing data were addressed	х
		(d) If applicable, describe analytical methods taking account of sampling strategy	Х

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		(e) Describe any sensitivity analyses	х
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility,	Х
		confirmed eligible, included in the study, completing follow-up, and analysed	
		(b) Give reasons for non-participation at each stage	х
		(c) Consider use of a flow diagram	Х
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	7-12
		(b) Indicate number of participants with missing data for each variable of interest	х
Outcome data	15*	Report numbers of outcome events or summary measures	7-14
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence	13-14
		interval). Make clear which confounders were adjusted for and why they were included	
		(b) Report category boundaries when continuous variables were categorized	Х
		(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	Х
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	х
Discussion			
Key results	18	Summarise key results with reference to study objectives	15
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and	17-18
		magnitude of any potential bias	
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from	15-18
		similar studies, and other relevant evidence	
Generalisability	21	Discuss the generalisability (external validity) of the study results	Х
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on	19
		which the present article is based	

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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Association between self-reported sleep duration and serum lipid profile in a middle-aged and elderly population in Taiwan: A community-based, cross-sectional study

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Primary Subject Heading :	General practice / Family practice
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• Article Title

Association between self-reported sleep duration and serum lipid profile in a middle-aged and

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elderly population in Taiwan: A community-based, cross-sectional study

• Running title

Association between sleep duration and serum lipid profile

Author names and affiliations

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ABSTRACT

Objectives: The association between sleep duration and serum lipid profile in the middle-aged and the elderly is unclear. The aim of this study was to investigate and evaluate the relationships between sleep duration and levels of serum total cholesterol (TC), LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), and triglycerides (TG) in these populations. **Design:** Cross-sectional observational study.

Setting: Community-based investigation in Guishan Township of northern Taiwan. Participants: A total of 400 community-dwelling middle-aged and elderly individuals were

enrolled. All participants underwent a baseline assessment in 2014, which included anthropometrics, blood samples, and self-administered questionnaires. Participants were

classified into three groups based on their sleep duration.

Outcome measures: Multivariate logistic regression was used to obtain odds ratios (ORs) and 95% confidence intervals (CIs) to assess the relationship between sleep duration and lipid profiles.

Results: Participant mean age was 64.5 years and 35.3% were men. Subjects with longer (>7 hours) and shorter (<6 hours) nightly sleep duration had a higher prevalence of low HDL-C levels (HDL <40 mg/dL) than those with moderate sleep duration (6-7 hours). Multivariate

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logistic regression revealed that, compared to individuals with sleep duration of 6-7 hours, the odds of having low HDL-C were 3.68 (95% CI = 1.59-8.49) greater for individuals with sleep duration of <6 hours and 2.89 (95% CI = 1.10-7.61) greater for individuals with sleep duration of >7 hours.

Conclusions: There was a U-shaped relationship between sleep duration and HDL-C levels. Sleep duration over 7 hours or less than 6 hours increased the risk of low serum HDL-C levels.

Keywords: Cholesterol/Lipid, Family Health, Metabolism, Prevention, Sleep, Sleep Duration.

Strengths and limitations of this study:

- This is the first study to explore the associations between sleep duration and lipid profiles in middle-aged and elderly Taiwanese population.
- We could not determine a causal relationship in our findings because this was a cross-sectional study.
- Lifestyle questionnaires were self-reported, which may limit the accuracy of measurements.

INTRODUCTION

2	As known, most older adults experience lipid profile disorders. ¹ Moreover, the different
3	levels in the percentage of lipid disorders may have an association with demographic factors
4	(such as age and sex). ¹ A study also found a significant association between the high
5	prevalence of dyslipidemia and risk factors, such as increasing age, smoking status,
6	hypertension, diabetes, and body mass index. ² Several studies have identified that longer or
7	shorter sleep duration may increase the mortality and morbidity risks from diabetes
8	mellitus, ³⁻⁵ obesity, ⁶ hypertension, ⁷ and coronary heart disease. ⁸ Some studies have shown a
9	U-shaped association between sleep duration and major morbidities. ⁵⁹¹⁰ Cardiovascular
10	disease has been a leading cause of death worldwide. ¹¹ Dyslipidemia, such as high levels of
11	total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG)
12	and low levels of high-density lipoprotein cholesterol (HDL-C), increases the risk of
13	cardiovascular disease, ¹² and hence remains an important issue in the field of health
14	promotion and disease prevention. Previous studies have shown age-associated alterations in
15	the level, composition, and function of lipid profiles, including an inverted U-shaped
16	quadratic trajectory for TC, LDL-C, and TG, ^{13 14} and a decrease in anti-oxidative ability. ¹⁵
17	Other than age, factors that have been suggested to be related to lipid profile levels include
18	body mass index (BMI), body composition, diet, and cardiorespiratory fitness. ^{13 14}

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Although some studies suggest that sleep deprivation may result in changes to plasma lipid levels,¹⁶ there is a lack of consensus on the possible associations that may exist between sleep duration and serum lipid profiles, and even fewer data are available in the Taiwanese population.¹⁷ Therefore, we investigated the relationship between sleep duration and levels of serum TC, LDL-C, HDL-C, and TG in a community-based study in Taiwan. **MATERIALS AND METHODS Study participants** The present study was an observational, cross-sectional study. We enrolled 400 participants, including 141 men and 259 women. The inclusion criteria included (1) residents aged over 50 years, and (2) the residents living in Guishan Township. Twelve people aged younger than 50 years old were excluded from the study. Subjects were excluded if (1) they could not complete the full examinations or had missing data for age, sex, anthropometric values and blood test results; (2) were functionally dependent; (3) were unable to adequately communicate with the interviewers; (4) had major illnesses recently; or (5) had known sleep disorders. **Data collection** Data collection comprised two parts: a physical status examination and a

36 self-administered questionnaire. For the physical status examination, height, weight,

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37	abdominal circumference, blood pressure, and blood samples were collected for all
38	participants. Blood samples included lipid profiles (TC, LDL-C, HDL-C, and TG), aspartate
39	aminotransferase (AST) levels, fasting plasma glucose (FPG) levels, creatinine levels, and
40	uric acid levels. The self-administered questionnaire included items related to smoking,
41	drinking, exercise, sleep, and other lifestyle habits. Details from all the participants were
42	obtained on the same day while the data collection was performed from March to August
43	2014.
44	Definitions and variables
45	Based on the National Cholesterol Education Program (NCEP) Adult Treatment Panel
46	(ATP) III guidelines, we set different cutoff points for each item of the serum lipid profiles
47	(LDL-C: 130 mg/dL; TC: 200 mg/dL; HDL-C: 40 mg/dL; and TG: 150 mg/dL).
48	Hyperlipidemia was defined as having LDL-C, TC, or TG levels above the cutoff points.
49	Adapted from the Pittsburgh Sleep Quality Index (PSQI), ¹⁷ the questionnaire about sleep
50	included the question: "What was your daily average sleep duration during the past month
51	(not counting the times lying on the bed without sleeping)?". We divided sleep duration into
52	three groups: less than 6 hours (<6 hours), greater than or equal to 6 hours but less than or
53	equal to 7 hours (6-7 hours), and more than 7 hours (>7 hours). Hypertension was defined as a
54	self-reported hypertension or the use of anti-hypertensive drugs. Diabetes was defined as a
55	self-reported diabetes mellitus or the use of oral anti-diabetic drugs or insulin. Alcohol
	6

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drinking was defined as drinking alcohol on greater than or equal to 2 days per week. Regular	shed as
exercise was defined as exercising greater or equal to 2 days per week. Current smoking was	10.1136
defined as the literal meaning.	/bmjope Protecte
The institutional review board (IRB) approved the study and all patients provided written	n-2017-()d by co
informed consent.)15964 o pyright,
Statistical analysis	includir
	tober ng for
Adequate blood samples and complete questionnaires were collected from all	2017. uses
participants. After data were collected, statistical analysis was conducted using the SPSS 21.0	. Downlo Supe related
statistical package (SPSS Inc., Chicago, USA). A <i>p</i> -value <0.05 was considered statistically	baded fr erieur (A to text a
significant. Our statistical analysis included multiple steps. First, characteristics were	om http: \BES) . nd data
compared across hyperlipidemia and sleep duration sub-groups using t-tests, one-way	//bmjop mining,
ANOVA, and chi-square tests. Homogeneity examination was performed with ANOVA, and	en.bmj.c Al train
heterogeneous variables were analyzed with the Brown-Forsythe test. Second, we measured	ing, and
correlations between serum lipid profiles and related risk factors. Third, logistic regression	June 7, similar
analyses were conducted to assess the relationship between sleep duration and the risk of high	2025 at technol
LDL-C, high TC, low HDL-C, and high TG levels; additional regression models were	Agence ogies.
adjusted for relevant covariates.	Bibliog
RESULTS	raphique
The characteristics of study subjects with and without hyperlipidemia are shown in Table	e de l Ens
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> 1. Among the included participants, 260 (65%) had hyperlipidemia. Body mass index, waist

- circumference, waist-to-height ratio, AST levels, FPG levels, and uric acid levels were
- significantly higher in the hyperlipidemia group.

Variables	No hyperlipidemia (n=140)	Hyperlipidemia (n=260)	<i>p</i> -value
Sex (male)	57 (40.7)	84 (32.3)	0.101
Age (years)	65.4 ± 8.4	64.0 ± 8.4	0.072
Current smoking	9 (6.4)	34 (13.1)	0.043
Alcohol drinking	20 (14.3)	55 (21.2)	0.107
Regular exercise	116 (83.2)	211 (81.2)	0.681
Hypertension	64 (45.7)	137 (52.7)	0.209
Diabetes	28 (19.7)	56 (21.5)	0.238
LDL-C (mg/dL)	97.6 ± 17.7	129.6 ± 32.6	< 0.001
TC (mg/dL)	169.8 ± 21.1	211.9 ± 33.2	< 0.001
HDL-C (mg/dL)	55.4 ± 12.8	53.9 ± 14.5	0.161
TG (mg/dL)	84.5 ± 29.3	142.3 ± 71.6	< 0.001
Sleep (hours)	6.2 ± 1.2	6.2 ± 1.3	0.276
SBP (mmHg)	128.0 ± 16.0	130.2 ± 17.0	0.267
DBP (mmHg)	76.1 ± 11.3	77.1 ± 12.2	0.592
BMI (kg/m^2)	23.7 ± 3.4	25.0 ± 3.6	0.001
WC (cm)	82.9 ± 8.5	86.3 ± 10.1	0.001
WHtR	0.52 ± 0.05	0.54 ± 0.06	0.003
ALT (mg/dL)	20.3 ± 8.9	23.9 ± 14.6	0.032
Creatinine (mg/dL)	0.8 ± 0.6	0.8 ± 0.3	0.408
FPG (mg/dL)	92.2 ± 19.1	98.4 ± 28.5	0.028
Uric acid (mg/dL)	5.4 ± 1.3	5.9 ± 1.4	0.001

79 Data expressed as mean \pm SD for continuous variables and n (%) for categorical variables.

80 Abbreviations: TC = total cholesterol; TG = triglyceride; LDL-C = low-density lipoprotein;

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81 HDL-C = high-density lipoprotein; Sleep = sleep duration; ALT = alanine transaminase; WC

82 = waist circumference; WHtR = waist to height ratio; BMI = body mass index; FPG = fasting

83 plasma glucose; SBP = systolic blood pressure; DBP = diastolic blood pressure.

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> The characteristics of the study subjects categorized by sleep duration are shown in Table 2. There were 100, 255, and 45 participants in the <6 hours, 6-7 hours, and the >7 hours groups respectively. Low HDL-C levels were significantly higher in the >7 hours group (31.1%) than in the other two groups (9.8% for the 6-7 hours group and 13.0% for the <6 hours group). The post-hoc tests also showed that the difference was significant (significant difference between <6 hours and >7 hours groups (*p* value = 0.009), 6-7 hours and >7 hours (*p* value=0.001).

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Variable	<6 hours (n = 100)	6-7 hours (n =	>7 hours (n =	<i>p</i> -value
_		255)	45)	
Sex (male)	30 (30)	88 (34.5)	23 (51.1)*†	0.044
Age (years)	65.2 ± 7.6	64.1 ± 8.3	65.1 ± 10.6	0.530
LDL-C (mg/dL)	115.8 ± 30.4	119.2 ± 31.3	119.6 ± 40.0	0.643
TC (mg/dL)	197.2 ± 34.9	197.0 ± 34.2	198.0 ± 45.4	0.984
HDL-C (mg/dL)	57.4 ± 15.8	53.9 ± 12.5	51.2 ± 16.4	0.025
TG (mg/dL)	120.4 ± 61.8	119.8 ± 62.7	138.5 ± 88.6	0.207
High LDL-C	33 (33)	87 (34.1)	15 (33.3)	0.978
High TC	49 (49)	125 (49.0)	21 (47.7)	0.941
Low HDL-C	13 (13)	25 (9.8)	14 (31.1)**	0.001
High TG	22 (22)	58 (22.7)	14 (31.1)	0.437
Alcohol drinking	15 (15)	52 (20.4)	11 (24.4)	0.346
Current Smoking	10 (10)	27 (10.6)	6 (13.3)	0.828
Regular exercise	80 (80)	209 (81.9)	39 (86.4)	0.642
Hypertension	47 (47)	125 (49.0)	29 (64.4)	0.122
Diabetes	25 (25)	45 (17.6)	9 (20.0)	0.293
Hyperlipidemia	69 (69)	161 (63.1)	30 (66.7)	0.564
SBP (mmHg)	129.9 ± 16.5	128.6 ± 16.7	133.3 ± 25.2	0.216
DBP (mmHg)	76.4 ± 13.0	76.6 ± 11.8	78.9 ± 9.13	0.436
BMI (kg/m ²)	24.3 ± 3.9	24.6 ± 3.4	25.1 ± 3.6	0.394
WC (cm)	84.7 ± 10.4	84.8 ± 9.6	87.4 ± 8.6	0.235
WHtR	0.5 ± 0.1	0.6 ± 0.1	0.6 ± 0.1	0.417
ALT (mg/dL)	22.5 ± 14.0	22.3 ± 11.94	22.7 ± 15.9	0.530
Creatinine (mg/dL)	0.8 ± 0.3	0.8 ± 0.3	1.0 ± 0.9	0.920
FPG (mg/dL)	98.2 ± 28.2	94.8 ± 19.3	100.3 ± 45.1	0.519
Uric acid (mg/dL)	5.6 ± 1.3	5.8 ± 1.4	6.1 ± 1.4	0.101

95 group.

96 Abbreviations: High TC = total cholesterol \geq 200 (mg/d); High TG = triglyceride \geq 150 (mg/d);

97 High LDL-C = low-density lipoprotein ≥ 130 (mg/d); Low HDL-C = high-density

98 lipoprotein \leq 40 (mg/d); ALT = alanine transaminase; WC = waist circumference; WHtR =

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99 waist to height ratio; BMI = body mass index; FPG = fasting plasma glucose; SBP = systolic

100 blood pressure; DBP = diastolic blood pressure.

101	Figures 1 shows the comparisons of serum lipid profiles by sleep duration for two age
102	groups: equal to or greater than 65 years (elderly group, 168 people) and all others (middle
103	age group, 232 people). Abnormal total cholesterol prevalence levels were the highest (around
104	50%) among the four items with no significant differences by sleep duration (Figure 1A). The
105	average prevalence of high LDL-C was about one-third, and the prevalence gradually
106	increased with longer sleep duration in the middle age group but decreased with longer sleep
107	duration in the elderly group (Figure 1B). There was a U-shaped (or J-shaped) distribution in
108	the association of low HDL-C levels with sleep duration with both age groups showing
109	significantly higher prevalence of low HDL-C when the sleep duration was <6 hours or >7
110	hours (Figure 1C). Abnormal triglyceride levels by sleep duration in the middle age group
111	also appeared to have a U-shaped distribution (Figure 1D).

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Multivariable logistic regression model results are shown in Table 3. Model 1 assessed
the crude odds ratio of lipid profiles by sleep duration; Model 2 adjusted for age; Model 3
adjusted for age and waist circumference; and Model 4 adjusted for age, waist circumference,
and other traditional factors that influence lipid levels, including sex, alcohol drinking,
exercise, and smoking. The 6–7-hour sleep duration group was set as the reference category.
In a model where high TC levels, high LDL-C levels, or high TG levels were the outcome, no
significant association was found with sleep duration. However, significant association was
found in Models 1 through 4 when the dependent variable was low HDL-C levels (Table 3).


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$\begin{tabular}{ c c c c c c } \hline $(n=100)$ (n=255) (n=45)$ \\ \hline $Total cholesterol$ \\ \hline $Total cholesterol$ \\ \hline $Model 1$ 1.02 (0.54-1.92) 1.00 1.10 (0.54-2.22)$ \\ \hline $Model 2$ 0.99 (0.52-1.88) 1.00 1.10 (0.52-2.24)$ \\ \hline $Model 3$ 1.01 (0.53-1.93) 1.00 1.13 (0.56-2.33)$ \\ \hline $Model 4$ 0.90 (0.45-1.80) 1.00 0.95 (0.44-2.02)$ \\ \hline $LDL-C$ \\ \hline $Model 1$ 1.04 (0.53-2.03) 1.00 0.99 (0.47-2.06)$ \\ \hline $Model 2$ 1.02 (0.52-2.00) 1.00 0.99 (0.47-2.16)$ \\ \hline $Model 3$ 1.05 (0.53-2.07) 1.00 1.03 (0.49-2.19)$ \\ \hline $Model 4$ 1.001 (0.50-2.01) 1.00 0.94 (0.43-2.04)$ \\ \hline $HDL-C$ \\ \hline $Model 1$ 3.81 (1.80-8.05)* 1.00 3.02 (1.28-7.14)$ \\ \hline $Model 1$ 3.81 (1.80-8.05)* 1.00 3.02 (1.28-7.14)$ \\ \hline $Model 1$ 3.81 (1.63-8.00)* 1.00 2.93 (1.17-7.34)$ \\ \hline $Model 3$ 3.62 (1.63-8.00)* 1.00 2.89 (1.10-7.61)$ \\ \hline $Triglyceride$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.63 (0.28-1.37)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.66 (0.26-1.39)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.66 (0.26-1.39)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.66 (0.26-1.39)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.66 (0.26-1.39)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.66 (0.26-1.39)$ \\ \hline $Model 1$ 0.65 (0.33-1.31) 1.00 0.66 (0.26-1.39)$$			Sleep <6 hours	Sleep 6-7 hours	Sleep >7 hours
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 smoking. Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low-density lipoprote HDL-C= high-density lipoprotein. 	125	logistic regression a	adjusted for age, sex, wat	st circumference, alco	hol drinking, exerci
 Abbreviations: TC=total cholesterol; TG=triglyceride; LDL-C=low-density lipoprote HDL-C= high-density lipoprotein. 	126	smoking.			
128 HDL-C= high-density lipoprotein.	127	Abbreviations: TC=	total cholesterol; TG=tr	glyceride; LDL-C=lo	w-density lipoprotei
	128	HDL-C= high-dens	ity lipoprotein.		
				15	

DISCUSSION

130	In our study, traditional risk factors, such as high BMI, high waist circumference, high
131	FPG levels, and high uric acid levels, were significantly associated with abnormal lipid values.
132	Elevated AST levels may be associated with non-alcoholic liver disease. We found a
133	U-shaped association between sleep duration and low HDL-C levels, and logistic regression
134	models showed that sleep duration was significantly associated with low HDL-C levels
135	regardless of the adjustment for potential confounders.
136	Several studies have reported the associations between serum lipid profiles and sleep
137	duration, but the results of these studies have been inconsistent. ¹⁸⁻²³ Choi et al. ¹⁸ collected
138	data from 4,222 Korean participants over the age of 60 years and found a U-shaped
139	association between low HDL-C levels and high triglyceride levels, which were similar to the
140	results of our study. Both short and long sleep durations were related to an increased risk of
141	metabolic syndrome and sleep duration of 7 hours demonstrated the lowest prevalence of
142	metabolic syndrome in this study. Hall et al. ¹⁹ reported that sleep duration was independently
143	associated with three components of metabolic syndrome: abdominal obesity, elevated serum
144	glucose levels, and elevated triglyceride levels. The optimal sleep duration for preventing
145	metabolic syndrome determined by this study was 7 to 8 hours per night. Bjorvatn et al. ²⁰
146	demonstrated that cholesterol levels, triglyceride levels, and blood pressure were higher in
147	subjects with short sleep duration. Another study ²¹ revealed that HDL-C levels decreased with

148	short and long sleep duration among normotensive, but not hypertensive, women.
149	The logistic regression models, adjusted for age, sex, waist circumference, alcohol
150	drinking, exercise, and smoking, showed significant association with low HDL-C levels. To
151	our knowledge, the prevalence of dyslipidemia is proportional to age and different in men and
152	women. The association between waist circumference and dyslipidemia has been confirmed
153	in some studies. ^{24 25} Lifestyle factors have been shown to influence serum lipid or lipoprotein
154	levels. For example, smoking decreases HDL-C levels and increases TG levels, whereas
155	alcohol consumption increases the levels of both. ²⁶⁻²⁸ Exercise increases HDL-C levels and
156	decreases TG levels. ^{29 30} In addition, alcohol consumption is reported to decrease LDL-C
157	levels. ^{31 32} Even after adjusting for these potential confounding factors, our logistic regression
158	models showed significant differences in odds of low HDL-C levels between sleep duration
159	groups.
160	The influence that shorter sleep duration has on body weight and dyslipidemia has
161	become clearer in recent years. Sleep restriction is associated with hormone imbalance; it
162	reduces leptin (an appetite suppressant) and elevates ghrelin levels (an appetite stimulant), ^{6 33}
163	which may contribute to increased body weight and lead to dyslipidemia. The biochemical
164	mechanism for the relationship between longer sleep and dyslipidemia has not been clearly
165	confirmed; some studies showed that prolonged sleep duration may be associated with
166	glucose intolerance and diabetes. ^{3 34} Physical performance, such as reduced energy

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167	consumption due to increased time in bed, may affect obesity; one study explained this
168	relationship by showing that longer sleep duration was related to less exercise. ⁸
169	Some studies have reported that the relative risk of mortality and morbidity was the
170	lowest when sleep duration was 7-8 hours per night, ^{5 8-10} but other research has reported that
171	6-7 hours is more optimal. ²² The cutoff point of our study was set at 6-7 hours; we found the
172	volunteers in our study generally woke up early regardless of the amount of time they slept,
173	and they often had spontaneous arousal from nocturnal sleep. This may be explained by
174	physiological age-related changes in circadian modulation, homeostatic factors,
175	cardiopulmonary function, and endocrine function; ^{35 36} or be due to underlying chronic
176	diseases. In an attempt to recommend the optimal sleep duration for lowering morbidity based
177	on epidemiological data, it is inferred that the optimal sleep duration will vary for different
178	populations. ²²
179	Limitations of this study should be mentioned. First, since this was a cross-sectional
180	study, a cohort study is required for determining more causal relationships. Second, selection
181	bias might exist because the volunteer participant selection was limited to one township, and
182	participants were not selected randomly or with a stratified method from the population. This
183	could limit the generalizability of these results. In addition, our sample size was relatively
184	small; hence, sampling bias should be considered. Third, we did not group the population by
185	underlying diseases, such as diabetes or cerebrovascular disease, due to the limited sample

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186	size. The proper cutoff for lipid profiles may differ according to underlying diseases.
187	Furthermore, the HDL-C cutoff point differs by sex; in metabolic syndrome, it is set as 40
188	mg/dL for men and 50 mg/dL for women. The influence of sleep duration on lipid metabolism
189	may also differ between men and women; in a study by Kaneita et al., ²² among 1,666 men and
190	2,329 women aged 20 years or older from Japan, it was shown that HDL-C levels had a
191	U-shaped association with sleep duration in women but not in men. Fourth, glycosylated
192	hemoglobin might be a more appropriate measurement of diabetes instead of self-reported
193	diabetes mellitus or the use of oral anti-diabetic drugs or insulin. Fifth, similar to the majority
194	of studies in the literature examining the effects of sleep duration on serum lipids, diet was
195	not factored in as a covariate. It may be explained that daily life diet is a diversified condition.
196	Thus, it may be difficult to represent it with just one variable in order to adjust for it in the
197	regression model. Sixth, lipid-lowering therapy was an important factor influencing lipid
198	levels, but we did not consider lipid-lowering medications while formulating the
199	questionnaire. Finally, the present study estimated sleep duration only by a self-reported
200	questionnaire, which is a more systematically biased estimate than measured sleep duration.
201	CONCLUSIONS
202	In our study of a Taiwanese population, sleep duration of 6-7 hours per night reduced the
203	risk of abnormal serum lipid profiles. Sleep duration over 7 hours or less than 6 hours may
204	increase the risk of low serum HDL-C levels, although adjustment for risk factors attenuated

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this relationship. Inappropriate sleep duration might be a potential risk factor for low HDL-C levels, and adequate sleep duration may improve low HDL-C status. Lifestyle interventions, including exercise and abstaining from alcohol and smoking, should be initiated early in high-risk groups.

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209 Contributors
210 PL was involved in the data collection, analysis, and writing of the manuscript. KTC and
211 YAL were involved in the collection of data. IST provided statistical advice. HHC and JYC

contributed to the conceptualization; they designed, performed the experiments, collected and

- analyzed the data, as well as revising it critically for important intellectual content; and also
- approved the final version to be submitted.
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- 217 Competing interests None declared.
- 218 Ethics approval The study was approved by Chang-Gung Medical Foundation Institutional
- 219 Review Board (102-2304B), and written informed consent was given by all the participants

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- before enrollment.
 - **Data sharing statement** No additional data are available.

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332	Figure Legends
333	Figure1. Prevalence of serum lipid profiles by sleep duration between two age groups (middle
334	age: 50-65 years old; elderly: ≥65 years old)

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Figure1. Prevalence of serum lipid profiles by sleep duration between two age groups (middle age: 50-65 years old; elderly: ≥65 years old)

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STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of cross-sectional studies

Section/Topic	ltem #	Recommendation	Reported on page #
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract	1-3
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	2-3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4-5
Objectives	3	State specific objectives, including any prespecified hypotheses	4-5
Methods			
Study design	4	Present key elements of study design early in the paper	5
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5
Participants	6	(a) Give the eligibility criteria, and the sources and methods of selection of participants	X
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	5-7
Data sources/	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe	5-6
measurement		comparability of assessment methods if there is more than one group	
Bias	9	Describe any efforts to address potential sources of bias	X
Study size	10	Explain how the study size was arrived at	5-6
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	6-7
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	7
		(b) Describe any methods used to examine subgroups and interactions	X
		(c) Explain how missing data were addressed	Х
		(d) If applicable, describe analytical methods taking account of sampling strategy	Х

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Page	30	of	30
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		(e) Describe any sensitivity analyses	Х
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility,	х
		confirmed eligible, included in the study, completing follow-up, and analysed	
		(b) Give reasons for non-participation at each stage	х
		(c) Consider use of a flow diagram	х
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	7-12
		(b) Indicate number of participants with missing data for each variable of interest	х
Outcome data	15*	Report numbers of outcome events or summary measures	7-14
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence	13-14
		interval). Make clear which confounders were adjusted for and why they were included	
		(b) Report category boundaries when continuous variables were categorized	х
		(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	х
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	х
Discussion			
Key results	18	Summarise key results with reference to study objectives	15
Limitations	mitations 19 Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias		17-18
Interpretation	20 Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence		15-18
Generalisability	21	Discuss the generalisability (external validity) of the study results	x
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	19

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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