Night sleep duration and risk of each lipid profile abnormality in a Chinese population: a prospective cohort study

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Research

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Abstract

Background To explore the associations between sleep duration and abnormalities in each serum lipid level in a Chinese population. Methods A prospective study was conducted with 33,817 participants from the general Chinese population. Sleep duration was categorized as ≤5, 6, 7, 8 or ≥9 h. Each lipid profile abnormality was defined according to the Chinese Guidelines for the Prevention and Treatment of Dyslipidemia in Adults. The Cox proportional hazards model was used to assess these associations. Results Compared with a seven-hour sleep duration, longer sleep duration was significantly associated with high low-density lipoprotein cholesterol (LDL-C) (HR: 1.18; 95% CI: 1.10–1.17) in fully adjusted models. And a longer sleep duration was significantly associated with low high-density lipoprotein cholesterol (HDL-C) levels (HR: 1.22; 95% CI: 1.19–1.35). In subgroup analyses, the positive association between long sleep duration and high LDL-C and low HDL-C levels in men and in the different age groups were more pronounced than the association in women. No significant interactions were observed in the association between sleep duration and each abnormal serum lipid level by sex/age in the study population(p- interaction>0.05). Conclusions These findings suggest that longer sleep duration is associated with high LDL-C and low HDL-C levels among the Chinese population.

Introduction

Shorter or longer sleep durations have been reported to be associated with a higher risk of diabetes mellitus[1–3], obesity[4, 5], and hypertension[6, 7], and shorter or longer sleep durations have also been suggested to be related to cardiovascular disease (CVD)[8, 9] and atherosclerosis[10]. It has been increasingly recognized that sleeping habits, along with other lifestyle habits, such as eating, exercising, smoking, and drinking, are potential risk factors for cardiovascular outcomes. In fact, the mechanisms underlying these relationships are still unclear. In recent years, several studies have examined the links between sleep duration and risk factors for CVD, including lipids[11–21]. Dyslipidemia, such as high levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) and low levels of high-density lipoprotein cholesterol (HDL-C), increases the risk of CVD[22, 23], and hence remains an important issue in the field of health promotion and disease prevention. To prevent CVD, it is important to identify and change lifestyle habits that are associated with serum TG, HDL-C or LDL-C levels.

Increasing evidence from laboratory and epidemiological studies has suggested that shorter or longer sleep durations might be associated with serum lipid profiles[11–21]. However, the findings have been inconsistent. Cross-sectional associations have been found between short sleep durations and lower HDL-C levels in adult American women with type 2 diabetes[19] and in adult Japanese women from the general population[11]. Short sleep durations were found to be associated with the highest cholesterol levels among the cholesterol levels that were associated with all other sleep duration categories in cross-sectional analyses of the general populations from Norway[20] and the United States[21]. In contrast, longer sleep durations were found to be associated with low HDL-C levels among a Korean adult population from the Korean National Health and Nutrition Examination Survey[16]. A survey from the Coronary Artery Risk Development Study involving 503 black and white adults aged 32–51 years showed that longer sleep duration was associated with increased future TC levels and TG levels but not with HDL-C[15]. However, a common limitation to these published studies is that parameters related to snoring status, as well as a large sample cohort study, were lacking. Little is known about the longitudinal relationships. Based on this background, we hypothesized that sleep duration was linked to dyslipidemia in the general population in China. We conducted this study in a Chinese population from the Kailuan Study, aiming to explore the association between sleep duration and each lipid profile abnormality stratified by age and gender.

Methods

Study design and participants

The Kailuan study was a prospective cohort study involving 101,510 participants (men: 81,110; women: 20,400, aged 18–98 years) in the Kailuan community from June 2006 to October 2007[24]. Of the 101,510 people who participated in the survey, 33,817 adults were included in the analysis after excluding 62,739 subjects who had a history of dyslipidemia and 4954 subjects with missing information about sleep duration or serum lipid profiles from the baseline examination or the follow-up visits. The health interview survey was performed using self-administered structured questionnaires to obtain information on sociodemographic characteristics, health status, and health behaviors. Before the study, all doctors and nurses received rigorous unified training.

Assessment of sleep duration

Sleep duration data were collected through a self-reported answer to the question "How many hours of sleep have you had on an average at night in the preceding 3 months?" We divided sleep durations into five groups according to the responses: ≤5 hours, 6 hours, 7 hours, 8 hours, and ≥9 hours. Additionally, participants were asked to answer "yes" or "no" to the question "Do you generally snore when you sleep?"[24, 25].

Assessment of potential covariates

The clinical examination consisted of a medical history, a physical examination, anthropometric measurements, and self-administered questionnaires on lifestyle characteristics [24, 26], such as sleep duration, regular leisure-time physical activity, smoking habits, and daily alcohol consumption. Body mass index (BMI) was calculated as the weight (kg) divided by the square of height (meters^2). Blood pressure was measured three times using a standardized sphygmomanometer while the participant was in a seated position. We used the average of the three measurements.

We measured TG, TC, LDL-C, HDL-C, fasting blood glucose (FBG), and high-sensitivity C-reactive protein (h-CRP) levels. All blood samples were analyzed by using a Hitachi 747 autoanalyzer (Hitachi; Tokyo, Japan)[24]. Diabetes was defined as having a history of diabetes, the use of glucose-lowering agents, or a fasting blood glucose ≥7 mmol/l. Hypertension was defined as a systolic blood pressure ≥140 mmHg, a diastolic blood pressure ≥90 mmHg, a history of hypertension and/or the use of antihypertensive agents.

Diagnosis of each lipid profile abnormality

Each lipid profile abnormality was defined according to the Chinese Guidelines on the Prevention and Treatment of Dyslipidemia in Adults[27]. Specifically, abnormal TC was defined as TC >5.18 mmol/L, abnormal TG were defined as TG >1.70 mmol/L, abnormal LDL-C was defined as LDL-C >3.37 mmol/L, and abnormal HDL-C was defined as HDL-C <1.04 mmol/L.

Statistical analyses

We described continuous variables by their means ± standard deviations and compared groups using a one-way analysis of variance (ANOVA). Categorical variables were described as percentages and were compared using the Chi-square test. The Cox proportional hazards model was used to estimate the hazard ratio (HR) for the incidence of each lipid profile abnormality in relation to sleep duration and baseline covariates. The follow up of each subject continued until the diagnosis of lipid profile abnormalities at one of the routine medical examinations that occurred every 2 years or until the fourth follow-up examination that was conducted between 1 June 2014 and 31 December 2015, whichever came first. For all models, the adequacy of the Cox proportional hazards model was assessed. Model 1 was adjusted for age and sex. Model 2 further adjusted for the level of education, smoking, alcohol consumption, physical activity, and snoring. Model 3 further adjusted for hypertension, diabetes mellitus, BMI, and h-CRP. To investigate whether age/sex acted as an effect modifier in the relationship between sleep duration and dyslipidemia, we ran a regression model with an interaction term between age/sex and sleep duration. Because 11 hospitals participated in the study, we used a Cox proportional hazards model with a sandwich covariance matrix as a random effect to account for the potential confounding effect of multiple hospitals participating in the study[24]. The statistical analysis was performed using SAS 9.4. All statistical tests were two-sided, and the significance level was set at 0.05.

Results

The baseline characteristics of the study participants by sleep duration are shown in Table 1. The mean age of the participants was 49.11 years (range 20–97 years), and 76.27% were men. The percentages of participants who reported sleeping for ≤ 5 h, 6 h, 7 h, 8 h, and ≥ 9 h per night were 6.05%, 15.73%, 16.39%, 60.08%, and 1.74%, respectively. Age, sex, education, smoking, alcohol consumption, physical activity, hypertension, diabetes mellitus, and snoring symptoms differed between the sleep duration categories.

Table 1
Baseline characteristics according to sleep duration

Variable	Sleep duration(hours)					p value
	≤ 5 (n = 2046)	6.0 (n = 5321)	7 (n = 5544)	8.0 (n = 20318)	≥ 9 (n = 588)	
Questionnaire-based data						
Age, years	54.14 ± 12.30	51.38 ± 12.44	48.59 ± 12.92	48.24 ± 12.61	46.13 ± 14.96	< 0.001
Male, n (%)	1718(83.97)	4451(83.65)	4335(78.19)	14866(73.17)	422(71.77)	< 0.001
Female, n (%)	328(16.03)	870(16.35)	1209(21.81)	5452(26.83)	166(28.23)	< 0.001
High school or above, n (%)	383(18.72)	1320(24.81)	1914(34.52)	3612(17.78)	220(37.41)	< 0.001
Current smoker, n (%)	1130(55.23)	2971(55.84)	2904(52.38)	4821(23.73)	295(50.17)	< 0.001
Current alcohol consumption, n (%)	1114(54.45)	3126(58.75)	3134(56.53)	5030(24.76)	300(51.02)	< 0.001
Physically active, n (%)	548(26.78)	1206(22.66)	1175(21.19)	1626(8.00)	109(18.54)	< 0.001
Snoring, n (%)	449(21.95)	963(18.10)	879(15.95)	1244(6.12)	86(14.63)	< 0.001
Exam-based data						
Hypertension, n (%)	798(39.00)	1826(34.32)	1739(31.37)	7397(36.41)	162(27.55)	< 0.001
Systolic blood pressure, mmHg	128.64 ± 20.50	127.25 ± 20.03	125.16 ± 19.73	126.90 ± 20.17	123.06 ± 20.97	< 0.001
Diastolic blood pressure, mmHg	81.69 ± 11.27	81.11 ± 10.06	80.28 ± 10.94	81.84 ± 11.46	78.75 ± 11.46	< 0.001
Diabetes mellitus, n (%)	145(7.09)	311(5.84)	286(5.16)	999(4.92)	38(6.46)	< 0.001
Fasting blood glucose, mmol/L	5.27 ± 1.36	5.21 ± 1.30	5.19 ± 1.20	5.22 ± 1.26	5.23 ± 1.42	0.915
Total cholesterol, mmol/L	4.36 ± 0.58	4.36 ± 0.57	4.34 ± 0.57	4.38 ± 0.57	4.28 ± 0.62	< 0.001
Triglycerides, mmol/L	0.98 ± 0.34	0.98 ± 0.33	0.97 ± 0.34	1.01 ± 0.33	0.95 ± 0.35	< 0.001
Low-density lipoprotein, mmol/L	2.14 ± 0.64	2.13 ± 0.64	2.14 ± 0.61	2.07 ± 0.68	2.00 ± 0.66	< 0.001
High-density lipoprotein, mmol/L	1.55 ± 0.36	1.55 ± 0.33	1.52 ± 0.32	1.55 ± 0.31	1.53 ± 0.32	< 0.001
Body mass index, kg/m^2	24.11 ± 3.35	24.23 ± 3.32	24.25 ± 3.37	24.28 ± 3.36	24.02 ± 3.52	0.082
High sensitivity C-reactive protein, mg/L	0.66(0.27–1.80)	0.64(0.24–1.70)	0.70(0.29–1.70)	0.65(0.22–1.87)	0.80(0.27-2.00)	0.088

Table 2 shows the characteristics of the study participants by dyslipidemia status. Participants with dyslipidemia were more likely to be men, have less than a high school education, be current smokers, be current drinkers, have a higher h-CRP level, have a higher blood pressure, have a higher prevalence of snoring symptoms, be overweight and have diabetes.

Table 2
Comparisons between patients with and without dyslipidemia in the Kailuan study.

Variable	Dyslipidemia	Without dyslipidemia	p value
No.	n = 22548	n = 11269	
Questionnaire-based data			
Age, years	48.43 ± 12.22	50.48 ± 13.93	< 0.001
Male, n (%)	17126(75.95)	8666(76.90)	0.054
Female, n (%)	5422(24.05)	2603(23.10)	
High school or above, n (%)	5107(22.65)	2342(20.78)	< 0.001
Current smoker, n (%)	8336(36.97)	3785(33.59)	< 0.001
Current alcohol consumption, n (%)	8779(38.93)	3925(34.83)	< 0.001
Physically active, n (%)	3072(13.62)	1592(14.13)	0.210
Snoring, n (%)	2535(11.24)	1086(9.64)	< 0.001
Exam-based data			
Hypertension, n (%)	8009(35.52)	3913(34.72)	0.151
Systolic blood pressure, mmHg	126.76 ± 19.83	126.62 ± 20.72	< 0.001
Diastolic blood pressure, mmHg	81.66 ± 11.25	80.90 ± 11.40	< 0.001
Diabetes mellitus, n (%)	1251(5.55)	528(4.69)	< 0.001
Fasting blood glucose, mmol/L	5.25 ± 1.28	5.15 ± 1.24	< 0.001
Total cholesterol, mmol/L	4.42 ± 0.56	4.26 ± 0.57	< 0.001
Triglycerides, mmol/L	1.04 ± 0.33	0.91 ± 0.32	< 0.001
Low-density lipoprotein, mmol/L	2.13 ± 0.67	2.03 ± 0.65	< 0.001
High-density lipoprotein, mmol/L	1.53 ± 0.32	1.57 ± 0.32	< 0.001
Body mass index, kg/m^2	24.52 ± 3.34	23.73 ± 3.33	< 0.001
High sensitivity C-reactive protein, mg/L	0.70(0.26–1.90)	0.59(0.20–1.59)	< 0.001

The association between sleep duration and each abnormal serum lipid level was examined using multivariate Cox regression models (Table 3). Compared with a seven-hour sleep duration, longer sleep duration (≥ 8 hours) was significantly associated with high LDL-C (HR: 1.18; 95% CI: 1.10–1.17) in adjusted models. After adjusting for covariates, a longer sleep duration (≥ 9 hours) was significantly associated with low HDL-C levels (HR: 1.22; 95% CI: 1.19–1.35).

Table 3
Models for associations between sleep duration and each abnormal serum lipid level.

		Model 1	**Model 2**	**Model 3**
Sleep duration (h)	n(incidence)	HR(95% CI)	HR(95% CI)	HR(95% CI)
TC				
≤ 5	900(43.99)	0.96(0.89–1.03)	0.96(0.89–1.03)	0.97(0.90–1.05)
6.0	2410(45.29)	1.00(0.95–1.06)	1.00(0.95–1.06)	1.01(0.95–1.07)
7.0	2517(45.40)	reference	reference	reference
8.0	8818(43.40)	0.94(0.90–0.98)	0.99(0.94–1.04)	0.99(0.95–1.04)
≥ 9	236(40.14)	0.91(0.79–1.04)	0.91(0.80–1.04)	0.92(0.80–1.05)
TG				
≤ 5	543(26.54)	0.96(0.87–1.06)	0.95(0.86–1.05)	0.98(0.89–1.08)
6.0	1592(29.92)	1.04(0.97–1.11)	1.02(0.96–1.10)	1.03(0.96–1.10)
7.0	1679(30.28)	reference	reference	reference
8.0	6058(29.82)	1.01(0.95–1.06)	1.04(0.99–1.11)	1.04(0.98–1.10)
≥ 9	184(31.29)	1.05(0.90–1.22)	1.05(0.90–1.23)	1.05(0.90–1.23)
LDL-C				
≤ 5	408(19.94)	0.99(0.89–1.12)	0.99(0.88–1.11)	1.00(0.90–1.13)
6.0	1060(19.92)	1.01(0.93–1.10)	1.01(0.93–1.10)	1.01(0.93–1.10)
7.0	1083(19.53)	reference	reference	reference
8.0	4369(21.50)	1.15(1.07–1.23)	1.18(1.10–1.27)	1.18(1.10–1.27)
≥ 9	105(17.86)	1.00(0.82–1.22)	0.99(0.81–1.22)	1.00(0.82–1.22)
HDL-C				
≤ 5	1497(73.17)	0.99(0.94–1.05)	1.00(0.94–1.06)	1.00(0.94–1.07)
6.0	3744(70.36)	1.01(0.96–1.06)	1.01(0.97–1.06)	1.01(0.97–1.06)
7.0	3748(67.60)	reference	reference	reference
8.0	13980(68.81)	1.10(1.07–1.15)	1.10(1.06–1.15)	1.10(1.06–1.14)
≥ 9	423(71.94)	1.25(1.13–1.39)	1.25(1.13–1.38)	1.22(1.10–1.35)
Dyslipidemia				
≤ 5	1323(64.66)	0.95(0.89–1.01)	0.95(0.89–1.01)	0.97(0.91–1.03)
6.0	3634(68.30)	1.04(0.99–1.09)	1.04(0.99–1.09)	1.05(1.00-1.10)
7.0	3782(68.22)	reference	reference	reference
8.0	13420(66.05)	0.96(0.92–0.99)	1.01(0.97–1.05)	1.01(0.97–1.05)
≥ 9	389(66.16)	0.97(0.87–1.07)	0.97(0.87–1.08)	0.96(0.87–1.07)

Model 1 adjusted for age and sex.

Model 2 further adjusted for level of education, smoking, alcohol, physical activity, and snoring. Model 3 further adjusted for hypertension, diabetes mellitus, body mass index, and high-sensitivity C-reactive protein.

Table 4 shows the association between sleep duration and each abnormal serum lipid level stratified by sex and age. Among men, there was a positive association between long sleep duration and high LDL-C levels (HR: 1.14; 95% CI: 1.06–1.24) and low HDL-C levels (HR: 1.20; 95% CI: 1.07–1.35) in the fully adjusted model. Conversely, among women, there was a nonsignificant positive association between long sleep duration and high LDL-C and low HDL-C levels in the fully adjusted model. Positive associations between long sleep duration and high LDL-C levels and low HDL-C levels were found in participants aged < 60 years and participants aged ≥ 60 years. However, no significant interactions were observed in the association between sleep duration and each abnormal serum lipid level by sex/age in the study population (P for the interaction > 0.05).

Table 4
Model 3 to determine associations between sleep duration and abnormal serum lipid levels stratified by gender and age.

	Women		**Men**		**Age < 60**		**Age ≥ 60**	
Sleep duration (h)	n(incidence)	HR (95% CI)	n(incidence)	HR (95% CI)	n(incidence)	HR (95% CI)	n(incidence)	HR (95% CI)
TC								
≤ 5	198(60.37)	1.08(0.91–1.27)	702(40.86)	0.94(0.86–1.02)	689(48.18)	1.01(0.93–1.11)	211(34.25)	0.88(0.75–1.05)
6.0	493(56.67)	1.07(0.95–1.21)	1917(43.07)	0.99(0.93–1.06)	1972(47.82)	1.03(0.97–1.09)	438(36.59)	0.94(0.82–1.08)
7.0	620(51.28)	reference	1897(43.76)	reference	2124(46.57)	reference	393(39.98)	reference
8.0	2688(49.30)	0.98(0.89–1.07)	6130(41.24)	1.01(0.96–1.07)	7576(44.52)	0.99(0.94–1.05)	1242(37.62)	1.06(0.93–1.19)
≥ 9	77(46.39)	1.03(0.81–1.32)	159(37.68)	0.90(0.77–1.06)	202(41.74)	0.96(0.82–1.11)	34(32.69)	0.89(0.62–1.26)
TG								
≤ 5	99(30.18)	1.12(0.89–1.41)	444(25.84)	0.95(0.85–1.05)	464(32.45)	1.04(0.94–1.15)	79(12.82)	0.73(0.56–0.96)
6.0	266(30.57)	1.15(0.97–1.36)	1326(29.79)	1.01(0.93–1.09)	1382(33.51)	1.04(0.96–1.12)	210(17.54)	0.99(0.81–1.21)
7.0	305(25.23)	reference	1374(31.70)	reference	1499(32.87)	reference	180(18.31)	reference
8.0	1480(27.15)	1.08(0.95–1.23)	4578(30.80)	1.05(0.98–1.12)	5489(32.31)	1.05(0.99–1.11)	560(16.96)	1.00(0.84–1.20)
≥ 9	46(27.71)	1.17(0.84–1.62)	138(32.70)	1.07(0.90–1.28)	164(33.88)	1.07(0.90–1.26)	20(19.23)	1.12(0.70–1.78)
LDL-C								
≤ 5	88(26.83)	1.30(1.01–1.66)	320(18.63)	0.94(0.83–1.07)	316(22.10)	1.10(0.97–1.26)	92(14.94)	0.81(0.63–1.04)
6.0	195(22.41)	1.08(0.89–1.32)	865(19.43)	0.99(0.90–1.09)	848(20.56)	1.04(0.95–1.14)	212(17.71)	0.91(0.75–1.12)
7.0	233(19.27)	reference	850(19.61)	reference	891(19.54)	reference	192(19.53)	reference
8.0	1289(23.64)	1.31(1.13–1.52)	3080(20.72)	1.14(1.06–1.24)	3709(21.80)	1.19(1.10–1.28)	660(19.99)	1.21(1.02–1.44)
≥ 9	32(19.28)	1.27(0.87–1.84)	73(17.30)	0.94(0.74–1.19)	89(18.39)	1.05(0.84–1.30)	16(15.38)	0.83(0.50–1.39)
HDL-C								
≤ 5	205(62.50)	0.96(0.82–1.13)	1292(75.20)	1.01(0.95–1.08)	987(69.02)	1.00(0.93–1.08)	510(82.79)	1.01(0.90–1.13)
6.0	518(59.54)	0.99(0.88–1.11)	3226(72.48)	1.02(0.97–1.07)	2789(67.63)	1.02(0.97–1.08)	955(79.78)	0.96(0.88–1.06)
7.0	699(57.82)	reference	3049(70.33)	reference	2963(64.96)	reference	785(79.86)	reference
8.0	3200(58.69)	1.05(0.96–1.15)	10780(72.51)	1.10(1.05–1.15)	11248(66.10)	1.10(1.05–1.14)	2732(82.76)	1.10(1.02–1.20)
≥ 9	99(59.64)	1.22(0.98–1.52)	324(76.78)	1.20(1.07–1.35)	333(68.80)	1.22(1.09–1.37)	90(86.54)	1.08(0.86–1.34)
Dyslipidemia								
≤ 5	249(75.91)	1.13(0.98–1.30)	1074(62.51)	0.94(0.87–1.01)	994(69.51)	1.01(0.94–1.09)	329(53.41)	0.88(0.76-1.00)

Model 3 adjusted for age, sex, education, smoking, alcohol, physical activity, snoring, hypertension, diabetes mellitus, body mass index, and C-reactive protein.

	Women		Men		Age < 60		Age ≥ 60	
6.0	633(72.76)	1.13(1.01–1.25)	3001(67.42)	1.03(0.98–1.09)	2953(71.61)	1.07(1.01–1.12)	681(56.89)	0.98(0.88–1.10)
7.0	807(66.75)	reference	2975(68.63)	reference	3196(70.07)	reference	586(59.61)	reference
8.0	3619(66.38)	1.02(0.94–1.11)	9801(65.93)	1.02(0.98–1.07)	11513(67.66)	1.00(0.96–1.05)	1907(57.77)	1.10(0.99–1.21)
≥ 9	114(68.67)	1.13(0.92–1.38)	275(65.17)	0.94(0.83–1.07)	333(68.80)	1.00(0.89–1.12)	56(53.85)	0.91(0.69–1.20)

Model 3 adjusted for age, sex, education, smoking, alcohol, physical activity, snoring, hypertension, diabetes mellitus, body mass index, and C-reactive protein.

Discussion

These prospective data demonstrated that moderate and/or long sleep durations were associated with an increased risk of future low HDL-C and high LDL-C levels. These associations were observed independent of age, gender, BMI, h-CRP, smoking habits, alcohol consumption, physical activity, snoring, education, diabetes mellitus, and hypertension. However, no associations were revealed between sleep duration and the risk of future high TG or TC levels.

Several studies have reported the associations between serum lipid profiles and sleep duration, but the results of these studies have been inconsistent[11–13, 18, 20, 28]. A National Health and Nutrition Survey in Japan showed a U-shaped relationship between sleep duration and a high level of TG and between sleep duration and a low level of HDL-C[11]. Consistent with this finding, a study from the China Health and Nutrition Survey (2009) involving 8574 adults showed that both shorter and longer sleep durations were associated with higher risks of abnormal serum lipid profiles[18]. However, two cross-sectional studies conducted in the USA and Japan showed that self-reported sleep duration was not associated with hypercholesterolemia[20, 28]. On the other hand, the Kansai Healthcare Study in Japan reported that moderate and/or long sleep durations decreased the risk of future low HDL-C and high triglyceride levels[12]. Additionally, Juliana C. Chan et al. concluded that longer sleep duration was associated with lower risks of high TC and LDL-C in children and adolescents[13]. Our prospective findings suggest that longer sleep duration is associated with low HDL-C levels and high LDL-C levels among the Chinese population. Differences in the age distribution of the study population, lifestyle, socioeconomic status, incomplete control for confounding factors, or different categorization of sleep duration may explain the inconclusive associations thus far.

Several studies have found sex differences in the associations between sleep duration and abnormal serum lipid levels[17, 18]. Results from the National Health Interview Survey 2008 found gender differences in the association between sleep duration and hypercholesterolemia, with a positive association found between sleep duration ≤ 5 h and hypercholesterolemia in women, and an inverse association found between sleep duration ≥ 8 h and hypercholesterolemia in men[17]. Data from the China Health and Nutrition Survey (2009) showed that both shorter and longer sleep durations were associated with higher risks of abnormal serum lipid profiles in women but not in men[18]. However, the present study found no significant interaction between sleep duration and sex with respect to abnormal serum lipid levels, and in the present study, the sleep duration pattern was shown to be different between men and women. However, it is unclear whether these differences induce sex differences in the association between sleep duration and abnormal serum lipid levels, or not. Additionally, the current study found no significant interaction between sleep duration and age with respect to abnormal serum lipid levels. Further research is needed to investigate the association between sleep duration and serum lipid levels.

Moreover, it is not easy to explain the biological mechanism responsible for the association between long sleep duration and a high LDL-C or a low HDL-C level. First, several previous studies have shown that prolonged sleep duration may be associated with obesity[29], hypertension[6], diabetes and glucose intolerance[30, 31]. Second, sleep duration and abnormal lipids often share some common risk factors, such as smoking [32], alcohol consumption[33], and lower socioeconomic status [34]. Third, inflammation is one of the most important biological pathways because long sleep durations can lead to increased levels of inflammatory markers. We also found that the level of h-CRP in participants with dyslipidemia was higher than in those without dyslipidemia.

The major strengths of the current study include its large sample size and the availability of information on potential confounders and mediators. However, this study has some limitations. First, objective data could not be used for the evaluation of sleep duration. Therefore, the bias due to the use of self-reported data on sleep duration in this study remains to be resolved. The use of more precise measures of sleep-related variables, such as polysomnographies, may reduce variability, but these techniques are logistically unfeasible to perform in large epidemiological studies such as ours. Second, data on Chinese midday naps and sleep quality were not collected in the current study. Participants with sleep apnea were not excluded, which is associated with a high risk of dyslipidemia[35]. However, we adjusted for snoring status as an alternative confounder. Third, lipid-lowering therapy was an important factor influencing lipid levels, but we did not consider lipid-lowering medications when adjusting the confounding factors. Other limitations include possible bias arising from loss to follow up and missing data on baseline risk variables. Finally, we only investigated employees of the Kailuan Coal Company, and most of them were men. Therefore, the results may not be applicable to the general population.

Conclusions

In conclusion, the results of this study indicate that a long sleep duration is associated with a high serum LDL-C level or a low HDL−C level among the Chinese population. The amount of sleep might play a key role in the risk of future lipid profile abnormalities. Behavioral interventions could include assistance with implementing sleep hygiene practices and modifying maladaptive sleep habits. Further research is needed to investigate the biological and behavioral links between long sleep duration and high cholesterol and to explore the efficacy of sleep interventions for the treatment and prevention of dyslipidemia.

Abbreviations

LDL-C: low-density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; CVD: cardiovascular disease ; TC: total cholesterol; TG: triglyceride ; BMI : body mass index; FBG: fasting blood glucose; h-CRP: high-sensitivity C-reactive protein; HR: hazard ratio.

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Kailuan General Hospital in compliance with the Declaration of Helsinki, and all participants provided written informed consent.

Consent for publication

Not applicable

Availability of data and material

The datasets used and/or analysed during the current study are not available.

Competing interests The authors declare that they have no competing interests.**Funding**No funding was received.

Author Contributions

S.W. and X.W.conceived and designed this study, X.L. directed dataanalysis, X.L. and Q.S. writing the paper. W.Z. prepared the database and reviewed thepaper. X.W.and S.W. conducted the quality assurance, reviewed and edited the paper. All authorsreviewed the manuscript.

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