



Applied nutritional investigation

The association of sleep duration and cardiometabolic risk factors in a national sample of children and adolescents: The CASPIAN III Study

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ABSTRACT

Objectives: Although sleep duration is one of the most important health-related factors, its association with risk factors for chronic diseases has not been completely clarified, especially among children and adolescents. The aim of this study was to evaluate the association between sleep duration and CVD risk factors among a nationally representative sample of Iranian children and adolescents.

Methods: This cross-sectional national study was performed on a representative sample of 5528 Iranian students, ages 10 to 18 y living in central cities of 27 provinces of Iran. Physical examinations and laboratory tests were performed using standard protocols. To determine the association between sleep duration and cardiometabolic risk factors, multivariable logistic regression was used and odds ratios (OR; with 95% confidence intervals) are reported.

Results: The mean \pm SD age was not significantly different among boys (14.69 ± 2.45 y) and girls (14.7 ± 2.38 y). In a crude model, boys who slept > 8 h and 5 to 8 h had lower OR for abdominal obesity compared with those who had slept < 5 h in a crude model (ORs, 0.70, 0.80, 1.0, respectively; $P = 0.008$). A similar result was observed in an age-adjusted model for the prevalence of abdominal obesity (ORs, 0.69, 0.76, 1.0, respectively; $P = 0.011$). Girls who had slept > 8 h per day had lower OR for high serum low-density lipoprotein levels compared with those who slept < 5 h per day ($P = 0.002$). These differences remained significant even in the fully adjusted model for all the confounding variables ($P = 0.008$). Moreover, among boys ages 10 to 14 y, longer sleep duration increased the risk for high total cholesterol in all models.

Conclusion: Shorter sleep duration increased the risk for some cardiometabolic risk factors among adolescents. The clinical significance of our findings should be determined in longitudinal studies.

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Introduction

Currently, cardiovascular diseases (CVDs) are identified as the most common cause of death, worldwide. CVDs are the most common cause of morbidity, mortality, and disability with high health care costs both in developed and developing countries [1]. The trend for overweight and obesity, which predicts CVDs among Iranian children and adolescents, doubled from 1993 to

1999. Studies have indicated that about 80% of obese children are suffering from at least one confirmed CV risk factor [2]. Prospective and retrospective studies have shown that roots of CV risk factors including diabetes, dyslipidemia, hypertension, and overweight can be influenced by lifestyle-related factors in childhood [1,3,4].

Dietary habits, physical activity status, smoking, and sleep duration are considered CV risk factors [5]. Little sleep, lack of sleep, and changes in sleep duration are common problems seen in both adults and children in different countries [6,7]. The effective factors on this alteration contain changing lifestyle

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because of rapid urbanization, modernization, technological transformation, and limited outdoor activities [8]. Increases in households with two wage earners and elevated prevalence of shift work are responsible factors for a greater reduction in mean sleep time among adults [6]. Time spent watching television, listening to radio, and on the Internet are effective factors on these changes in children [6]. Children and adolescents are especially vulnerable because their habits and lifestyles form during this critical period [9]. Epidemiologic studies on children and adolescents have reported a consistent and strong relationship of sleep deprivation to both concurrent and future obesity [10]. Other studies have indicated that obese children have dyslipidemia, higher blood pressure, higher fasting glucose and insulin, and higher atherosclerotic risk factors [11]. Studies have shown that children ages 3.6 to 18.5 y with less sleep duration have more insulin resistance than children with normal sleep times [12]. A cross-sectional study found an inverse association between sleep duration and waist circumference (WC) in Chinese school children and adolescents ages 6 to 20 y [9]. Reverse relationships of sleep duration and hypertension, which was reported among adults, also was shown among boys ages 11 to 14 y [13]. Evidence has indicated that less sleep duration enhances the risk for hypercholesterolemia. Wish et al recently showed that shorter sleep duration resulted in increase hypercholesterolemia in a longitudinal study of adolescents [9]. To our knowledge, there is limited information about effects of sleep duration on lipid profiles of children and adolescents.

Studies in adults have indicated a relationship between sleep duration and metabolic syndrome and diabetes. However, few studies found this association in children and adolescents [14, 15]. The population of Iran is extremely young. More than 32% of the population is under age 15 y and 12% are school aged [8]. Conducting studies among the young population therefore is important in this country. According to our knowledge, there is no published data from Iran that provides information about the relationship between sleep duration and CV risk factors, especially in children. Therefore, this study was conducted to examine the association between sleep duration and CV risk factors in Iranian school-aged (10–18 y) children.

Methods

This study was conducted using the data from a longitudinal national project, CASPIAN III (Childhood and Adolescence Surveillance and Prevention of Adult Non-communicable disease). This nationwide health survey was a school-based program that was conducted in 27 Iranian provinces. The comprehensive methodology of this study has been published previously [16], and here we just briefly describe it. The present study included 5528 students ages 10 to 18 y. They were recruited by multistage random cluster sampling from urban and rural areas of 27 provincial counties in Iran. We randomly selected eligible schools for our study from a list of schools that were stratified according to the information bank of the Ministry of Education. Students also were selected randomly from each school. Those students who had any chronic disease or who were taking medications were not included in this study. This project was approved by the ethnic committees and other relevant national regulatory organizations. Written informed consent and oral assent was obtained from parents and students, respectively.

Assessment of anthropometric measurements

We measured the weights of students while they were wearing light clothes twice. Measurements were done without wearing shoes by digital scale and averaged to the nearest 0.2 kg. Height was measured in duplicate without shoes while the students were standing and the shoulders were in normal position. Body mass index (BMI) was calculated as weight (in kg) divided by squared of height (in m²). WC was measured at a level midway between the lower rib margin and iliac crest to the nearest 0.5 cm. The maximum level of hip without any pressure to the body surface to the nearest 0.5 cm was considered for measuring hip circumference.

Assessment of biomarkers

Students were requested to attend the nearest health center to their school. All students were instructed to fast for 12 h before blood sampling. The ante-cubital vein was considered for blood sampling. This was done between 08:00 and 09:30. All blood samples were centrifuged for 10 min at 3000 g within 30 min of venipuncture. We measured lipid profile levels including high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) by auto-analyzers. HDL-C was measured after precipitation of non-HDL-C with dextran sulphate-magnesium chloride. Low-density lipoprotein cholesterol (LDL-C) in serum samples with TG \leq 400 mg/dL was calculated according to Friedwald equation. Fasting blood sugar (FBS) and total cholesterol (TC) were measured by auto-analyzer. Central Provincial Laboratory, which follows the standards of the National Reference Laboratory, a World Health Organization (WHO)-Collaborating center in Tehran, was used for analyzing. The Data and Safety Monitoring Board of the project has taken into account for different levels of quality control. A comprehensive operation manual was provided for the study team. Based on standard guidelines, a group of external evaluation and supervisor nominated by two collaborating ministries checked the performance of the personnel, and monitored and calibrated equipment.

Assessment of blood pressure

For measuring systolic and diastolic blood pressure (SBP; DBP) we requested patients to be seated and calm, with appropriate cuff sizes according to arm size. BPs were measured twice after more than 5 min rest. A trained technician measured SBP by defining the clear of the first sound (first Korotkoff phase) and DBP by defining as disappearance of sound (fifth Korotkoff phase). We recorded the average of two measurements.

Assessment of sociodemographic variables and sleep duration

The questionnaire was prepared based on the WHO STEPwise approach to NCD (Tools ver 9.5) and WHO Global School Health Survey (GSHS). The questions concerning the sociodemographic characteristics and sleep duration were included in the "parents' questionnaire," which was added to the previously mentioned questionnaires [17]. The validity and reliability of all questionnaires have been confirmed in the first survey of this surveillance system. Trained health care providers questioned parents about variables related to sociodemographic issues and past history (e.g. maternal education, child's birth weight, family dietary habits, family history of obesity and other chronic disease, as well as sleep duration). Sleep duration was categorized in three groups from <5 h, 5 to 8 h, and >8 h.

Definition of risk factors

The Centers for Disease Control and Prevention (CDC) provided the BMI percentiles for the classification of the children and adolescents as underweight, normal, overweight, and obese [18]. To evaluate the pattern of physical activity, three indicators were used including:

1. hours of physical education at school;
2. hours of watching television at home; and
3. hours spent on sports club training.

By using principle component analysis, we obtained a score for physical activity. The mean of this score is presented in Table 1.

Abnormal serum lipids were defined as a TC, LDL-C and or TG higher than the level corresponding to the age and gender-specific 95th percentile, also HDL-C lower than age and gender-specific 5th percentile [19]. High fasting blood sugar (FBS) was determined with levels \geq 100 mg/dL [20]. The average SBP or DBP above the 90th percentile for that age and gender after adjusting for weight and height was considered elevated BP [21].

Statistical analysis

General characteristics of boys and girls were analyzed separately by sleep duration categories including <5 h, 5 to 8 h and >8 h. We performed one-way analysis of variance (ANOVA) with post hoc tests to evaluate significant differences in general features (e.g., age, BMI, WC, weight, and height) according to sleep duration categories. We reported χ^2 test for significant differences of qualitative characteristics as a percentage according to sleep duration. ANOVA was used to report the mean serum lipid profile and glucose levels as well as BP with 95% confidence interval (CI) according to different sleep duration groups. To determine the association between several risk factors of CVD and sleep duration, multivariable logistic regression and odds ratios (OR; with 95% CI) was used. *P* for trend was reported to investigate dose-response correlation between CVD risk

Table 1
Demographic characteristics and physical activity of participants according to sleep duration categories,* all of the ages 10–18 y: The CASPIAN–III Study

	Sleep duration (h/d)			P-value [†]
	<5	5–8	>8	
Boys				
Age (y)	14.4 ± 2.2	14.9 ± 2.4	14.6 ± 2.5	0.450
Weight (kg)	45.8 ± 12.7	46.7 ± 13.6	45.5 ± 13.1	0.137
WC (cm)	69.6 ± 14.3	67.7 ± 11.9	67.4 ± 26.3	0.314
BMI (kg/m ²)	19.2 ± 4.2	19.9 ± 4.3	19.5 ± 4.0	0.015
Physical activity (%)	0.5 ± 0.8	0.6 ± 1.1	−0.3 ± 0.8	0.000
Family history				
Diabetes (%)	5.5	26.9	67.6	0.010
Obesity (%)	4.3	27.7	68	0.475
Hyperlipidemia (%)	5.2	28.7	66.1	0.002
Hypertension (%)	6.0	27.7	66.2	0.000
Osteoporosis (%)	5.4	30.1	64.5	0.004
Girls				
Age (y)	14.4 ± 2.1	14.9 ± 2.3	14.6 ± 2.4	0.269
Weight (kg)	46.2 ± 15.2	49.4 ± 16.2	47.6 ± 16.9	0.351
WC (cm)	69.0 ± 10.6	70.1 ± 11.5	69.7 ± 22.6	0.981
BMI (kg/m ²)	18.9 ± 3.7	19.5 ± 4.0	19.1 ± 4.1	0.263
Physical activity (%)	0.4 ± 0.9	0.6 ± 1.2	−0.3 ± 0.7	0.000
Family history				
Diabetes (%)	6.2	27.2	66.6	0.001
Obesity (%)	5.7	25.1	69.2	0.119
Hyperlipidemia (%)	7.3	25.4	67.3	0.001
Hypertension (%)	5.7	26.7	67.6	0.020
Osteoporosis (%)	7.2	31.3	61.6	0.000

ANOVA, analysis of variance; BMI, body mass index; WC, waist circumference

* Sleep duration.

† P-value are resulted from ANOVA.

factors and sleep duration. In multivariable logistic regression, we used a crude model without adjustment and three additionally adjusted models for several potential confounders. Statistical analysis was performed using the SPSS for Windows software (version 16.0. SPSS, Chicago, IL), and $P < 0.05$ was considered statistically significant.

Table 2
Demographic characteristics and physical activity of participants according to sleep duration categories,* ages 10–14 y: The CASPIAN–III Study

	Sleep duration (h/d)			P-value [†]
	<5	5–8	>8	
Boys				
Age (y)	12.6 ± 1.1	12.4 ± 1.2	12.3 ± 1.2	0.070
Weight (kg)	39.4 ± 12.2	37.9 ± 11.1	37.6 ± 11.4	0.257
WC (cm)	67.0 ± 17.2	63.4 ± 11.4	62.8 ± 17.3	0.056
BMI (kg/m ²)	18.6 ± 4.3	18.0 ± 3.7	17.9 ± 3.9	0.214
Physical activity (%)	0.6 ± 0.9	0.6 ± 1.1	−0.3 ± 0.8	0.000
Family history				
Diabetes (%)	7.6	24.3	68.1	0.048
Obesity (%)	4.9	25.2	69.9	0.519
Hyperlipidemia (%)	7.6	25.1	67.3	0.002
Hypertension (%)	8.3	23.3	68.4	0.000
Osteoporosis (%)	7.9	26.6	65.5	0.037
Girls				
Age (y)	12.7 ± 0.9	12.6 ± 1.1	12.5 ± 1.2	0.015
Weight (kg)	36.8 ± 9.5	38.3 ± 11.5	36.5 ± 10.4	0.074
WC (cm)	64.3 ± 9.2	65.8 ± 11.3	64.5 ± 22.9	0.580
BMI (kg/m ²)	17.5 ± 2.9	18.3 ± 3.8	17.5 ± 3.6	0.083
Physical activity (%)	0.4 ± 0.9	0.6 ± 1.3	−0.4 ± 0.7	0.000
Family history				
Diabetes (%)	6.2	25.2	68.6	0.136
Obesity (%)	6.0	21.9	72.1	0.537
Hyperlipidemia (%)	7.7	21.9	70.3	0.020
Hypertension (%)	6.2	23.6	70.3	0.369
Osteoporosis (%)	6.1	27.3	66.7	0.051

ANOVA, analysis of variance; BMI, body mass index; WC, waist circumference

* Sleep duration.

† P-value are resulted from ANOVA.

Table 3
Demographic characteristics and physical activity of participants according to sleep duration categories,* ages 14–18 years: The CASPIAN–III Study

	Sleep duration (h/d)			P-value [†]
	<5	5–8	>8	
Boys				
Age (y)	16.3 ± 1.2	16.7 ± 1.2	16.7 ± 1.2	0.210
Weight (kg)	52.2 ± 9.7	53.3 ± 11.4	52.5 ± 10.3	0.521
WC (cm)	72.2 ± 10.0	71.0 ± 11.1	71.5 ± 31.7	0.933
BMI (kg/m ²)	21.2 ± 3.7	21.3 ± 4.1	20.8 ± 3.6	0.047
Physical activity (%)	0.4 ± 0.7	0.6 ± 1.1	−0.2 ± 0.8	0.000
Family history				
Diabetes (%)	3.7	29.9	67.1	0.095
Obesity (%)	3.8	29.9	66.3	0.242
Hyperlipidemia (%)	3.1	31.7	65.1	0.026
Hypertension (%)	4.1	31.6	64.4	0.025
Osteoporosis (%)	3.1	33.3	63.6	0.031
Girls				
Age (y)	16.5 ± 1.1	16.7 ± 1.1	16.7 ± 1.2	0.808
Weight (kg)	57.4 ± 13.1	58.1 ± 13.9	58.1 ± 15.0	0.819
WC (cm)	74.7 ± 9.3	73.4 ± 10.6	74.6 ± 21.2	0.435
BMI (kg/m ²)	20.7 ± 3.7	20.5 ± 3.8	20.6 ± 4.1	0.863
Physical activity (%)	0.4 ± 1.0	0.6 ± 1.2	−0.3 ± 0.7	0.000
Family history				
Diabetes (%)	6.3	29.0	64.8	0.017
Obesity (%)	5.5	27.5	67.0	0.331
Hyperlipidemia (%)	6.8	28.8	64.4	0.015
Hypertension (%)	5.3	29.6	65.2	0.150
Osteoporosis (%)	8.5	35.3	56.2	0.000

ANOVA, analysis of variance; BMI, body mass index; WC, waist circumference

* Sleep duration.

† P-value are resulted from ANOVA.

Results

Table 1 presents the characteristics of the study participants ages 10 to 18 y by sleep duration categories. The mean ± SD age was 14.69 ± 2.45 y for boys and 14.7 ± 2.38 y for girls. No significant differences were observed between the age of participants in sleep duration categories in either group of girls and boys. In the boys group, those who slept < 5 h had lower BMI compared with participants in other two groups. However, significant differences were not observed among girls. In both groups, subjects who slept 5 to 8 h were more active. Moreover, a family history of diabetes, hypertension, high blood lipid levels, and osteoporosis among those who slept longer were higher in both sexes.

The characteristics of the study participants ages 10 to 14 and 14 to 18 y by sleep duration categories are shown in the Tables 2 and 3, respectively. Among participants ages 10 to 14 y in both genders, participants who slept 5 to 8 h were more active. Among boys ages 10 to 14 y, longer sleep duration was associated with higher frequency of positive family history for diabetes, hypertension, high blood lipid levels, and osteoporosis. For girls aged 10 to 14 y, sleep duration > 8 h was associated with younger age and greater family history of high blood lipid levels. For boys ages 14 to 18 y, sleep duration > 8 h was related to lower BMI, greater family history of hypertension, high blood lipid levels, osteoporosis, and lower physical activity. For girls ages 14 to 18 y, sleeping > 8 h was associated with lower physical activity levels, as well as with higher frequency of positive family history for diabetes, high blood lipid levels, and osteoporosis.

Clinical characteristics of all participants by age and sleep duration categories are shown in Table 4. For all participants (10–18 y), mean serum TC was positively associated with sleep duration only among boys. Moreover, in this group, those who

Table 5 (Continued)

	Sleep duration (h/d)		P-value [†]
	5–8/<5	8>/<5	
Elevated FBS			
Model I [‡]	1.73 (0.71–4.17)	1.97 (0.83–4.67)	0.672
Model II [§]	1.73 (0.72–4.18)	1.97 (0.83–4.67)	0.678
Model III	2.03 (0.83–4.94)	2.01 (0.84–4.83)	0.588
Model IV [¶]	2.03 (0.83–4.94)	2.01 (0.84–4.83)	0.592
Elevated blood pressure			
Model I [‡]	1.29 (0.38–4.34)	1.26 (0.38–4.14)	0.624
Model II [§]	1.32 (0.39–4.47)	1.37 (0.41–4.54)	0.790
Model III	1.22 (0.36–4.17)	1.38 (0.41–4.64)	0.561
Model IV [¶]	1.18 (0.34–4.10)	1.45 (0.43–4.94)	0.559

BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; FBS, fasting blood sugar; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TC, total cholesterol; TG, triglyceride

CVD abnormalities following criteria, according to Adult Treatment Panel III. Criteria modified for children and adolescents: overweight: BMI; 85th–95th; obesity, BMI > 95th; low HDL: < 50 mg/dL (except in boys 15–19 y old, that cut-off was < 45 mg/dL); high LDL: > 110 mg/dL; high TG: ≥ 100 mg/dL; high TC: > 200 mg/dL; elevated FBS > 100 mg/dL; high blood pressure: > 95th (adjusted by age, sex, height)

* Sleep duration.

† P-value are resulted from ANOVA.

‡ Without adjustment (crude model).

§ Adjusted for age.

|| Additionally adjusted for other characteristics including socioeconomic status, parents' educations, family history of chronic disease, sedentary lifestyle.

¶ Additionally adjusted for BMI in all abnormalities except for overweight and obesity.

(OR, 0.70; 95% CI, 0.39–1.28 and OR, 0.80; 95% CI, 0.43–1.50, respectively). Moreover, a similar result was observed in an age-adjusted model (OR, 0.69; 95% CI, 0.38–1.25 and OR, 0.76; 95% CI, 0.41–1.42, respectively).

Further adjustment for other potentially confounding variables such as socioeconomic status, parents' educations, family history of chronic disease, sedentary lifestyle, and BMI attenuated these associations. Hypercholesterolemia (LDL > 110 mg/dL) was significantly associated with sleep duration categories only in the girls' group. Table 5 shows that participants who sleep >8 h compared with those who slept <5 h had lower OR for high LDL in a crude model (OR, 0.46; 95% CI, 0.13–1.61). OR for high LDL after control for age (model 4) was 0.44 (95% CI, 0.13–1.56) and for other confounding variable (model 5, 6) were 0.76 (95% CI, 0.20–2.88) and 0.76 (95% CI, 0.20–2.89), respectively. Conversely, compared with participants who slept <5 h, those who slept 5 to 8 h had significantly higher LDL levels in all models (crude and adjusted).

Tables 6 and 7 show sex-specific and multivariable OR of CV risk factors by categories of sleep duration and age. We found that for boys ages 10 to 14 y, there was significant inverse association ($P < 0.05$) between sleep duration and abdominal obesity in a crude model. These results changed substantially after adjustment for potential confounders (Table 6). Furthermore, in this group, longer sleep duration increased the risk for high TC in all models (crude and adjusted). For girls ages 10 to 14 y, participants who slept 5 to 8 h had higher risk for high LDL compared with those who slept <5 h in a crude model ($P < 0.05$). Conversely, participants who slept >8 h compared with those who slept <5 h had significantly lower LDL levels in a crude model. Among boys ages 14 to 18 y, there was a positive association between sleep duration and risk for abdominal obesity only in a crude model (Table 7). The girls ages 14 to 18 y who slept 5 to 8 h and >8 h had lower risk for hypercholesterolemia (LDL > 110 mg/dL) than those who slept <5 h in a crude model (Table 7).

Discussion

This study evaluated the relationship between sleep duration and CVD risk factors among school-aged children. Our findings indicate a relationship between decreased sleep duration and elevated LDL-C among all girls ages 6 to 18 y. Even after adjusting for other potential confounding variables, this association remained significant. This association also remained significant among girls ages 10 to 14 y in a crude model. Moreover, the girls ages 14 to 18 y who slept 5 to 8 h and >8 h had lower risk for hypercholesterolemia (LDL > 110 mg/dL) than those who slept <5 h in a crude model. In the current study, those (10–18 y) with sleep duration <5 h had higher risk for abdominal obesity among boys both in crude and age-adjusted models. However, this association attenuated after additional adjusting for other potential confounders. We also found a similar association among boys ages 10 to 14 y only in a crude model. In contrary, long sleep duration was associated with higher risk for abdominal obesity among boys ages 14 to 18 y. Among boys ages 10 to 14 years, longer sleep duration increased the risk for high TC in all models. To the best of our knowledge, this is the first study that evaluated the association between sleep duration and CVD risk factors among a nationally representative sample of children in developing countries.

Some findings of the present study are similar to published papers [14,15] and some are different from previous studies that have examined the relationship between sleep duration and obesity, as well as CVD risk factors among children and adolescents [22–27]. We did not find a significant relationship between short sleep duration and obesity in the present study. The results of a study among American obese children and adolescents have reported that sleep duration does not predict the development of adiposity, and metabolic syndrome [12]. However, several epidemiologic studies have reported that short sleep duration is potentially related to obesity in both adults and children [7,10,28,29]. The majority of cross-sectional studies have indicated that short sleep duration (generally <6 h per night) enhances the risk for obesity in both adults and children in different countries [30]. For example, a cross-sectional study among young Iranian female students recently reported that sleep deprivation is related to lower diet quality indices and a higher rate of general and central obesity [31]. Moreover, some prospective studies have reported that short sleep duration increases weight gain and obesity risk among girls and boys [7]. However, one study has shown that long sleep duration (generally >8 h per night) is directly related to obesity [30].

The results of the present study showed that short sleep duration independent of high BMI and other confounding variables significantly increased LDL-C levels in girls. In accordance with our findings, experimental results showed that sleep deprivation elevated TC and LDL-C levels [32]. Gangwisch et al [33] reported that short sleep duration increases the risk for hypercholesterolemia in a longitudinal study among adolescents. Additionally, one study with Chinese youths has demonstrated that for each 1-h decrease in average sleep time, the risk for elevated LDL-C was 14% [9].

Several mechanisms have been proposed to explain the association of short sleep duration with obesity and hypercholesterolemia. Sleep deprivation may be associated with up-regulation of appetite, energy-expenditure reduction, increasing time spent eating, and adiposity [31,34]. This association may be mediated by decreasing levels of leptin, increasing levels of ghrelin, an increased ratio of ghrelin to leptin, low levels of testosterone, and high amounts of evening cortisol levels [35–39].

Table 6
Odds ratio (95% CIs) for cardiovascular risk factors by categories of sleep duration* ages 10–14 y: The CASPIAN–III Study

	Sleep duration (h/d)		P-value [†]
	5–8/<5	8>/<5	
Boys			
Overweight			
Model I [‡]	0.77 (0.44–1.34)	0.70 (0.41–1.19)	0.209
Model III [§]	1.23 (0.51–2.97)	1.01 (0.42–2.43)	0.534
Generalized obesity			
Model I [‡]	0.77 (0.44–1.34)	0.70 (0.41–1.19)	0.316
Model III [§]	1.23 (0.51–2.97)	1.01 (0.42–2.43)	0.372
Abdominal obesity			
Model I [‡]	0.85 (0.42–1.74)	0.75 (0.38–1.46)	0.022
Model III [§]	1.64 (0.47–5.72)	1.12 (0.32–3.85)	0.259
Model IV	0.39 (0.10–1.54)	0.42 (0.11–1.61)	0.570
High LDL			
Model I [‡]	0.45 (0.24–0.84)	0.42 (0.23–0.75)	0.368
Model III [§]	0.62 (0.23–1.63)	0.54 (0.21–1.39)	0.884
Model IV	0.39 (0.10–1.54)	0.42 (0.11–1.61)	0.877
High TC			
Model I [‡]	4.94 (0.00)	1.34 (0.00)	0.001
Model III [§]	4.11 (0.00)	1.10 (0.00)	0.014
Model IV	4.08 (0.00)	1.12 (0.00)	0.013
High TG			
Model I [‡]	0.79 (0.31–2.05)	0.94 (0.39–2.28)	0.752
Model III [§]	1.47 (0.32–6.73)	1.54 (0.35–6.81)	0.652
Model IV	1.68 (0.34–8.21)	1.81 (0.38–8.74)	0.528
Elevated FBS			
Model I [‡]	1.52 (0.57–4.05)	1.69 (0.66–4.33)	0.282
Model III [§]	2.96 (0.38–22.99)	3.37 (0.44–25.67)	0.290
Model IV	2.94 (0.38–22.85)	3.67 (0.44–25.67)	0.285
Elevated blood pressure			
Model I [‡]	0.65 (0.17–2.45)	0.74 (0.22–2.52)	0.903
Model III [§]	3.55 (0.00)	4.42 (0.00)	0.381
Model IV	3.42 (0.00)	4.46 (0.00)	0.347
Girls			
Overweight			
Model I [‡]	1.74 (0.79–3.81)	1.48 (0.70–3.16)	0.867
Model III [§]	1.40 (0.45–4.32)	0.98 (0.32–2.96)	0.333
General obesity			
Model I [‡]	1.74 (0.79–3.81)	1.48 (0.70–3.16)	0.706
Model III [§]	1.40 (0.45–4.32)	0.98 (0.32–2.96)	0.111
Abdominal obesity			
Model I [‡]	1.53 (0.58–4.08)	1.21 (0.47–3.09)	0.131
Model III [§]	1.56 (0.34–7.15)	0.80 (0.18–3.59)	0.200
Model IV	1.29 (0.36–4.62)	1.17 (0.34–4.09)	0.979
High LDL			
Model I [‡]	1.46 (0.78–2.74)	0.99 (0.54–1.81)	0.007
Model III [§]	1.54 (0.55–4.25)	0.99 (0.36–2.72)	0.224
Model IV	1.29 (0.36–4.62)	1.17 (0.34–4.09)	0.200
High TC			
Model I [‡]	2.85 (0.65–12.46)	2.88 (0.69–12.10)	0.266
Model III [§]	2.33 (0.29–18.52)	2.14 (0.27–16.60)	0.762
Model IV	2.24 (0.28–17.93)	2.23 (0.28–17.48)	0.630
High TG			
Model I [‡]	0.96 (0.37–2.45)	0.59 (0.24–1.45)	0.070
Model III [§]	1.22 (0.26–5.71)	0.62 (0.13–2.90)	0.146
Model IV	0.91 (0.18–4.49)	0.71 (0.15–3.47)	0.537
Elevated FBS			
Model I [‡]	0.86 (0.43–1.70)	0.91 (0.48–1.73)	0.999
Model III [§]	1.37 (0.44–4.27)	1.01 (0.33–3.07)	0.423
Model IV	1.30 (0.42–4.08)	0.92 (0.30–2.83)	0.323
Elevated blood pressure			
Model I [‡]	0.62 (0.19–2.02)	0.60 (0.20–1.78)	0.487
Model III [§]	0.63 (0.07–5.45)	0.97 (0.12–7.87)	0.539
Model IV	0.57 (0.06–4.96)	0.94 (0.12–7.64)	0.481

ANOVA, analysis of variance; BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; FBS, fasting blood sugar; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TC, total cholesterol; TG, triglyceride

CVD abnormalities following criteria, according to Adult Treatment Panel III. Criteria modified for children and adolescents: overweight: BMI; 85th–95th; obesity, BMI > 95th; low HDL: < 50 mg/dL (except in boys 15–19 y old, that cut-off was < 45 mg/dL); high LDL: > 110 mg/dL; high TG: ≥ 100 mg/dL; high TC: > 200 mg/dL; elevated FBS > 100 mg/dL; high blood pressure: > 95th (adjusted by age, sex, height)

* Sleep duration.

[†] P-value are resulted from ANOVA.

[‡] Without adjustment (crude model).

[§] Additionally adjusted for other characteristics including socioeconomic status, parents' educations, family history of chronic disease, sedentary lifestyle.

^{||} Additionally adjusted for BMI in all abnormalities except for overweight and obesity.

Table 7

Odds ratio (95% CIs) for cardiovascular risk factors by categories of sleep duration* ages 14–18 y: The CASPIAN-III Study

	Sleep duration (h/d)		P-value [†]
	5–8/<5	8>/<5	
Boys			
Overweight			
Model I [‡]	1.27 (0.67–2.40)	0.98 (0.53–1.83)	0.216
Model III [§]	1.51 (0.57–4.03)	1.24 (0.47–3.28)	0.554
Generalized obesity			
Model I [‡]	1.27 (0.67–2.40)	0.98 (0.53–1.83)	0.288
Model III [§]	1.51 (0.57–4.03)	1.24 (0.47–3.28)	0.490
Abdominal obesity			
Model I [‡]	1.82 (0.70–4.72)	1.25 (0.49–3.20)	0.025
Model III [§]	2.01 (0.46–8.78)	1.50 (0.34–6.48)	0.512
Model IV	0.42 (0.15–1.14)	0.50 (0.19–1.34)	0.853
High LDL			
Model I [‡]	0.67 (0.38–1.17)	0.55 (0.32–0.94)	0.215
Model III [§]	0.83 (0.36–1.89)	0.77 (0.34–1.74)	0.678
Model IV	0.42 (0.15–1.14)	0.50 (0.19–1.34)	0.676
High TC			
Model I [‡]	1.60 (0.47–5.45)	1.54 (0.47–5.08)	0.722
Model III [§]	2.67 (0.35–20.63)	2.72 (0.35–20.80)	0.546
Model IV	2.65 (0.34–20.45)	2.71 (0.35–20.78)	0.536
High TG			
Model I [‡]	0.57 (0.26–1.26)	0.47 (0.22–1.00)	0.066
Model III [§]	0.81 (0.23–2.88)	0.81 (0.23–2.85)	0.869
Model IV	0.76 (0.21–2.73)	0.76 (0.21–2.70)	0.817
Elevated FBS			
Model I [‡]	3.98 (0.94–16.87)	3.22 (0.77–13.48)	0.834
Model III [§]	2.26 (0.51–9.95)	1.57 (0.36–6.84)	0.343
Model IV	2.26 (0.51–9.93)	1.59 (0.36–6.94)	0.389
Elevated blood pressure			
Model I [‡]	0.51 (0.22–1.18)	0.47 (0.21–1.04)	0.151
Model III [§]	0.87 (0.25–3.06)	0.77 (0.22–2.66)	0.585
Model IV	0.78 (0.22–2.77)	0.72 (0.21–2.53)	0.638
Girls			
Overweight			
Model I [‡]	0.66 (0.33–1.31)	0.83 (0.43–1.59)	0.591
Model III [§]	0.63 (0.25–1.57)	0.62 (0.26–1.52)	0.515
General obesity			
Model I [‡]	0.66 (0.33–1.31)	0.83 (0.43–1.59)	0.345
Model III [§]	0.63 (0.25–1.57)	0.62 (0.26–1.52)	0.943
Abdominal obesity			
Model I [‡]	0.85 (0.32–2.29)	1.11 (0.43–2.86)	0.655
Model III [§]	1.37 (0.30–6.16)	1.25 (0.28–5.52)	0.376
Model IV	0.78 (0.21–2.80)	1.46 (0.42–5.06)	0.119
High LDL			
Model I [‡]	0.59 (0.31–1.14)	0.76 (0.41–1.41)	0.045
Model III [§]	0.80 (0.31–2.08)	1.03 (0.40–2.63)	0.897
Model IV	0.78 (0.21–2.80)	1.46 (0.42–5.06)	0.866
High TC			
Model I [‡]	0.56 (0.15–2.07)	0.59 (0.17–2.03)	0.676
Model III [§]	0.49 (0.10–2.45)	0.54 (0.11–2.61)	0.773
Model IV	0.42 (0.08–2.13)	0.44 (0.09–2.13)	0.610
High TG			
Model I [‡]	0.76 (0.30–1.92)	0.75 (0.31–1.83)	0.673
Model III [§]	0.47 (0.16–1.37)	0.50 (0.17–1.44)	0.544
Model IV	0.37 (0.12–1.12)	0.41 (0.14–1.23)	0.521
Elevated FBS			
Model I [‡]	0.98 (0.44–2.22)	1.20 (0.55–2.62)	0.281
Model III [§]	2.81 (0.64–12.42)	3.29 (0.75–14.44)	0.144
Model IV	2.84 (0.64–12.55)	3.32 (0.76–14.56)	0.143
Elevated blood pressure			
Model I [‡]	1.14 (0.46–2.82)	1.08 (0.45–2.58)	0.932
Model III [§]	1.48 (0.33–6.62)	1.54 (0.35–6.79)	0.670
Model IV	1.51 (0.33–6.96)	1.66 (0.37–7.51)	0.531

ANOVA, analysis of variance; BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; FBS, fasting blood sugar; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TC, total cholesterol; TG, triglyceride

CVD abnormalities following criteria, according to Adult Treatment Panel III. Criteria modified for children and adolescents: overweight: BMI 85th–95th; obesity, BMI > 95th; low HDL: < 50 mg/dL (except in boys 15–19 y old, that cut-off was < 45 mg/dL); high LDL: >110 mg/dL; high TG: ≥ 100 mg/dL; high TC: > 200 mg/dL; elevated FBS > 100 mg/dL; high blood pressure: > 95th (adjusted by age, sex, height)

* Sleep duration.

† P-value are resulted from regression.

‡ Without adjustment (crude model).

§ Additionally adjusted for other characteristics including socioeconomic status, parents' educations, family history of chronic disease, sedentary lifestyle.

|| Additionally adjusted for BMI in all abnormalities except for overweight and obesity.

Additionally, dysregulation of the autonomic nervous system may play a role in the association between sleep deprivation and obesity, which is mediated by elevation of hypothalamic–pituitary–adrenal (HPA) axis activity and activated systemic inflammatory processes [35]. Short sleep duration usually is related to less physical activity and more stress, which can increase the catecholamine-induced lipolysis, TC, and LDL levels [32]. Observed association between sleep restriction and higher LDL-C levels among girls may be related to sex discrepancy in factors associated with the risk factors for high cholesterol [32].

Our study has some limitations and notable strengths. To our knowledge, it is the first study conducted to evaluate the association of sleep duration and CVD risk factors in Iran. The strengths of our study include a large, nationally representative sample size and consideration of the role of potential confounders in the data analysis. Moreover, our study participants included both girls and boys who were analyzed separately. There were several limitations in this study. First, the cross-sectional nature of the study, which limited us from getting information on the causal relationship of sleep duration and CVD risk factors. Second, sleep duration was self-reported by participants, which may include misclassification. However, studies have reported a good correlation between self-reported sleep duration and quantitative sleep assessment with actigraphy. Third, we measured only the quantity of sleep but not the quality such as the presence or absence of sleep apnea, which is associated with increased risk for CVDs. It is possible that despite adequate sleep duration, poor quality of sleep is associated with detrimental effects on obesity and other CVD risk factors. Our findings may propose new attitudes into the relationship between lifestyle and health. The promotion of lifestyle modification in early life to get enough sleep and to avoid habitual sleep restriction can help improve present and future health status.

Conclusion

Our observation showed that short sleep duration was independently associated with high LDL-C among children and adolescents. Moreover, a positive association was found between sleep duration and mean TC among boys ages 10 to 14 y. More prospective studies with the purpose of assessing long-term exposure (e.g., repeated actigraphy) and better control for confounding variables such as sleep disturbance are required. If our findings are supported by future prospective studies, avoiding unsuitable sleep duration can be considered a primary preventive program against development of chronic diseases, notably CVDs in adulthood.

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