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Original Article

Poor sleep quality and lipid profile in a rural cohort (The Baependi Heart Study)

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ABSTRACT

Aim: To test the association between cardiometabolic risk factors and subjective sleep quality assessed by the Pittsburgh sleep quality index (PSQI), independent of obstructive sleep apnea (OSA) and sleep duration.

Methods: A total of 573 participants from the Baependi Heart Study, a rural cohort from Brazil, completed sleep questionnaires and underwent polygraphy for OSA evaluation. Multivariable linear regression analysis tested the association between cardiovascular risk factors (outcome variables) and sleep quality measured by PSQI, adjusting for OSA and other potential confounders (age, sex, race, salary/wage, education, marital status, alcohol intake, obesity, smoking, hypertension, and sleep duration).

Results: The sample mean age was 43 ± 16 years, 66% were female, and mean body mass index (BMI) was 26 ± 5 kg/m^2. Only 20% were classified as obese (BMI ≥30). Overall, 50% of participants reported poor sleep quality as defined by a PSQI score ≥5. A high PSQI score was significantly associated with higher very-low-density lipoprotein (VLDL) cholesterol levels (beta = 0.392, $p = 0.012$) and higher triglyceride levels (beta = 0.017, $p = 0.006$), even after adjustments, including the apnea–hypopnea index. Further adjustments accounting for marital status, alcohol intake, and medication use did not change these findings. No significant association was observed between PSQI scores and glucose or blood pressure. According to PSQI components, sleep disturbances (beta = 1.976, $p = 0.027$), sleep medication use (beta = 1.121, $p = 0.019$), and daytime dysfunction (beta = 1.290, $p = 0.024$) were significantly associated with higher VLDL serum levels. Only the daytime dysfunction domain of the PSQI components was significantly associated with higher triglyceride levels (beta = 0.066, $p = 0.004$).

Conclusion: Poorer lipid profile was independently associated with poor sleep quality, assessed by the PSQI questionnaire, regardless of a normal sleep duration and accounting for OSA and socio-economic status.

1. Introduction

Sleep disturbances have been associated with metabolic dysregulation and may contribute to weight gain, obesity, and diabetes [1–6]. Obstructive sleep apnea (OSA) is known to explain part of this association, but other factors besides OSA are thought to also play a role [7–13]. For example, poor sleep quality has been

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associated with obesity, diabetes and weight gain [8,9,11,12], but few studies have shown these associations in the absence of OSA [8,11]. In addition, there is still controversy about the association between poor sleep quality and the lipid profile, once a poorer lipid profile has been initially associated only with sleep duration, but not with self-reported sleep quality [13]. However, a worse lipid profile was also independently associated with poor subjective sleep quality scores [12].

The aim of the current study was to test whether there is an independent association between subjective sleep quality, assessed by the Pittsburgh sleep quality index (PSQI) questionnaire, and cardiometabolic factors, while adjusting for OSA in a sample from the general population.

2. Methods

2.1. Study cohort

The Baependi Heart Study, now in its 12th year, is a family-based cohort study aimed at evaluating genetic and environmental influences on cardiovascular risk factors [14,15]. Baependi is a small rural town in the state of Minas Gerais, Brazil, with very limited inward migration. At baseline, 1691 individuals across 95 families were evaluated. Cross-sectional data was collected for 2239 participants [15]. The current study analyzed a subset of participants at the second evaluation period (from 2010 to 2014). All participants in this subset had completed sleep questionnaires (the PSQI and Epworth sleepiness scale, ESS) ($N = 1801$), and underwent OSA evaluation by overnight polygraphy ($N = 573$). We only studied individuals with complete sleep questionnaire data and sleep polygraphy. Our sample size is contingent on the fact that the sleep polygraphy protocol was only performed in a subset of participants. There was no specific inclusion criteria for the polygraphy protocol and all individuals from the total sample were invited to participate in the sleep polygraphy protocol.

The study protocol was approved by the ethics committee of the Hospital das Clinicas, University of São Paulo, Brazil, and each subject provided informed written consent before participation.

2.2. Covariates

Anthropometric measures, such as height, weight, waist, hip, and neck circumferences, were measured following standardized procedures as previously described [16]. Body mass index (BMI) was calculated from height and weight (kg/m^2). Smoking status was defined by asking the question: 'Have you smoked cigarettes…?' Possible answers were: (1) yes, in the past, but not currently; (2) yes, and I still smoke; and (3) I have never smoked. The three choices were thus characterized as (1) former, (2) current, and (3) non-smoker, respectively [17]. Seated systolic blood pressure (SBP) and diastolic blood pressure (DBP) were assessed by one trained examiner. The mean out of three measures was used to describe blood pressure at rest. Medication use, alcohol use, salary, education level, and marital status were assessed by questionnaires.

2.3. Sleep questionnaires

The PSQI is a self-rated questionnaire which assesses sleep quality over the past month. Nineteen individual items generate seven component scores: subjective sleep quality, habitual sleep efficiency, sleep latency, sleep duration, sleep disturbances, use of sleeping medication, and daytime dysfunction. A global PSQI score equal to or greater than 5 has been proposed to distinguish between good and poor sleepers [18,19].

The ESS is a self-administered questionnaire that provides a measurement of daytime sleepiness. EES scores (the sum of eight item scores, 0–3) can range from 0 to 24. Excessive daytime sleepiness is defined as a score >10 [20].

2.4. Other sleep measures

The diagnosis of OSA was made based on a portable type III study (Stardust II; Philips, Eindhoven, Netherlands). The overnight sleep monitor was installed by a technician at the patient's home. Briefly, the portable monitor used pulse oximetry, airflow detection through nasal cannula pressure, position sensor, heart rate recorded by pulse oximetry, and respiratory effort (chest-strap piezoelectric detection). Apneas were classified as central (absence of airflow or ≥90% reduction of baseline for ≥10 s associated with the absence of thoracic and abdominal effort), obstructive (absence of airflow or ≥90% reduction of baseline for ≥10 s associated with the presence of thoracic and abdominal effort), or mixed (absence of airflow or ≥90% reduction of baseline for ≥10 s in the presence of thoracic and abdominal effort at the end of the event) [21,22]. Hypopneas were classified as a 30% or more drop in airflow for ≥10 s with a 4% decrease in oxygen desaturation [21,22]. The apnea–hypopnea index (AHI) was assessed by the number of respiratory events (apneas and hypopneas) divided by the total sleep recording time. The OSA severity was determined according to standard cut-offs as mild (AHI 5–14), moderate (AHI 15–29), or severe (AHI ≥30) [21,22]. Further details regarding the polygraphy exam were previously described [23].

Bedtime and sleep duration were self-reported on the PSQI. Sleep efficiency information was extracted from the PSQI questionnaire. Data from sleep duration of this cohort has been previously published [14].

2.5. Metabolic risk factors

Fasting blood glucose, glycated hemoglobin (HbA1c), total cholesterol, lipoprotein fractions, and triglycerides were assayed by standard techniques in 12-h fasting blood samples. Serum samples were stored at −80 °C prior to analysis. We also tested the cholesterol ratio (total cholesterol divided by high-density lipoprotein (HDL) cholesterol) [24,25].

2.6. Statistical analysis

Descriptive analyses are shown as mean ± standard deviation (SD) for continuous and percentage for categorical variables. Natural log transformation (ln) was used in cases of departure from normality. Because triglycerides showed high kurtosis, we log transformed this variable (ln) while running linear regression analysis. Multivariable linear regression analysis was used to test the independent relationship between cardiovascular risk factors as outcome variables (lipid/glucose blood levels and blood pressure) and sleep quality as a main predictor (assessed by the PSQI), adjusting for age, sex, race, salary (monthly), education level, smoking status (never, former, current), BMI, SBP, daytime sleepiness (by ESS-continuous score), AHI continuous score, sleep duration, and bedtime. Further adjustments were also made for lipid-lowering medication use, blood glucose levels, alcohol use, and marital status. We also ran a sensitivity analysis excluding those with OSA (AHI ≥15) in order to explore the association between cardiometabolic factors and sleep quality independent of OSA. Similarly, we performed a restricted analysis for participants who did not use lipid-lowering medication. Sleep quality by the PSQI was used as a continuous score, a dichotomous category (cut-off 5), and by its seven domains. The final sample size for the current analysis was 573

Table 1
Univariate analysis of all complete sleep questionnaires (full sample) and those who also underwent portable sleep study (overnight polygraphy; analytical sample).

Demographics	Full sample ($N = 1801$)	Analytical sample ($N = 573$)	p
Age (years)	45 ± 17	43 ± 16	**0.023**
Sex, male (%)	42	34	**<0.001**
Self-reported race (%)			
White	76	80	**0.005**
Black	6	4	
Mixed	13	16	
Smoking status (%)			
Never	66	72	**<0.001**
Former	22	20	
Current	12	8	
Alcohol use (%)			
None	70	73	0.260
<Once/month	6	7	
Once to 3 times/week	10	9	
Up to 3 times/month	11	9	
Daily	1	1	
Other	2	1	
Salary (%)			
Up to 1 minimum wage (MW)	17	15	0.280
From 1 to 5 MW	76	78	
From 5 to 10 MW	3.5	4.0	
From 10 to 20 MW	1.0	0.5	
>20 MW	0.5	0.5	
No answer	2.0	2.0	
Marital status (%)			
Married	62	62	0.122
Single	26	27	
Divorced	6	5	
Widowed	6	6	
Education level (%)			
Illiterate	3	2	0.564
High school	82	81	
Technical training	3	4	
Graduation	12	13	
BMI (kg/m^2)	26 ± 5	26 ± 5	0.740
Waist circumference (cm)	91 ± 12	91 ± 12	0.580
Hip circumference (cm)	98 ± 10	98 ± 10	0.670
Neck circumference (cm)	36 ± 8	36 ± 12	0.790
Medication use (%)			
Anti-hypertensive	28	28	0.700
Hypoglycemic	5	6	0.210
Lipid-lowering	8	11	**0.004**
Oral contraceptive	15	18	0.965
Post-menopausal status (%)	22	24	0.830
Cardiometabolic risk factors			
VLDL cholesterol (mg/dL)	25 ± 12	25 ± 11	0.190
LDL cholesterol (mg/dL)	125 ± 35	123 ± 35	0.093
HDL cholesterol (mg/dL)	47 ± 12	48 ± 11	0.490
Total cholesterol (mg/dL)	198 ± 41	195 ± 41	0.075
Cholesterol ratio	4.40 ± 1.30	4.30 ± 1.30	0.052
Triglycerides (mg/dL)[a]	131 ± 75	126 ± 60	0.190
Glucose (mg/dL)	93 ± 20	94 ± 19	0.106
HbA1c (%)	5.7 ± 0.8	5.7 ± 0.7	0.084
Systolic BP (mmHg)	123 ± 11	121 ± 11	**0.001**
Diastolic BP (mmHg)	78 ± 9	77 ± 8	**0.017**

Data are shown as mean ± standard deviation (SD) for continuous and percentage for categorical variables. p-Value is by independent t-test for continuous and normally distributed variables (the test is independent for each covariate) (p-value indicates comparison of those included in the analytic sample to those excluded) and by Chi-squared for categorical variables. BMI, body mass index; BP, blood pressure; cholesterol ratio, total cholesterol/HDL; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very-low-density lipoprotein.
Bold values represent statistical significance (p-value < 0.05).
[a] Since triglycerides value was skewed, the p-value is by Mann–Whitney, a non-parametric test. The median and interquartile range (IQR) (25th, 75th) for triglycerides in the full sample is 116 (85, 158) mg/dL and in the analytical sample is 112 (87, 154) mg/dL.

participants, who underwent overnight polygraphy and had completed sleep questionnaires. We show descriptive data from the full sample ($N = 1801$) and from those used in the present analysis ($N = 573$). In addition, we tested whether those included in our analytic sample differed significantly from those who did not participate in the polygraphy examinations using independent t-tests for continuous variables or Chi-squared tests for categorical variables. Missing data were excluded while running regression analysis. Data were analyzed by the IBM SPSS statistics software version 18. The alpha level of significance was set at <0.05.

3. Results

Table 1 shows descriptive data from the total and the analytical sample for demographics and cardiovascular risk factors. The analytical sample is predominantly female (66%), mean age was 43 ± 16 years, age range from 18 to 81 years old, and mean BMI of 26 ± 5 kg/m^2. Overweight (BMI between 25 and 30) participants were 30% and only 20% were classified as obese subjects (BMI ≥30). Most reported never having smoked and not using alcohol. Almost one-quarter of the women in the sample were post-menopausal and almost 30% of the sample used anti-hypertensive medication. In general, cardiovascular risk factors were within normal ranges. The analytical sample showed a similar distribution of variables (eg, demographic and cardiometabolic factors) compared to the full sample, except for age, sex, race, smoking status, and blood pressure levels. Table 2 shows sleep characteristics according to questionnaires and polygraphy. Overall, 50% classified their sleep quality as poor by the PSQI, and 34% reported excessive daytime sleepiness. OSA, by polygraphy, was shown in 18% of the study sample. Sleep efficiency was considered to be normal (>85%) and self-reported sleep duration indicated that the majority of them slept more than 6 h/night. Self-reported bedtime showed a mean bedtime of 22:40 h. The analytical sample showed a slightly higher percentage of participants with poor sleep quality by the PSQI compared to full sample participants (50 vs 46%, respectively). Likewise, the analytical sample showed higher percentage of participants with excessive daytime sleepiness compared to participants from full sample (34 vs 30%, respectively). After testing for independent associations between sleep quality and cardiovascular risk factors, only VLDL cholesterol and triglyceride levels remained significantly associated with higher PSQI scores (Table 3). To explore other potential confounding factors relating poor sleep quality with the lipid profile, we further adjusted for lipid-lowering medication use, fasting glucose levels, alcohol use, and marital status. Results remained similar (Supplementary material: Appendix A1). To test the association between sleep quality and cardiometabolic risk variables, independently of OSA, we also performed an analysis only for those with an AHI <15, that is, excluding those with moderate-to-severe OSA. The results remained statistically significant (Table 4). In addition, we also ran a restricted analysis excluding those taking lipid-lowering medication. The findings remained significant (Table 5).

Furthermore, we evaluated the PSQI by components as main predictors. The Supplementary Material shows all multiple regression analyses testing PSQI components' association with cardiovascular risk factors. According to PSQI components, we first modeled them as continuous scores, and then, if statistically significant, they were modeled within strata. Higher scores in the daytime dysfunction domain (showed overall and within strata) were significantly associated with higher ln (triglycerides) levels ($p = 0.004$) (Supplementary material: Appendix A2). The sleep disturbances domain ($p = 0.027$), sleep medication use domain ($p = 0.019$), and daytime dysfunction domain ($p = 0.024$) were significantly associated with higher VLDL blood levels (Supplementary material: Appendix A3).

Table 2
Univariate analysis of all complete sleep questionnaires (full sample) and those who also underwent portable sleep study (overnight polygraphy; analytical sample).

Sleep characteristics	Full sample ($N = 1801$)	Analytical Sample ($N = 573$)	p
AHI (events/h)	–	10 ± 10	–
ODI (events/h)	–	10 ± 11	–
Nadir SpO_2	–	82 ± 10	–
Basal SpO_2	–	95 ± 5	–
AHI ≥5 (%)	–	58	–
AHI ≥15 (%)	–	18	–
AHI ≥30 (%)	–	5	–
ESS continuous score	7 ± 5	7 ± 5	0.094
ESS >10 (%)	30	34	**0.009**
PSQI total score	5 ± 3	5 ± 3	0.095
PSQI ≥5 (%)	46	50	**0.018**
Sleep duration (self-reported) ≥6 h (%)	94	93	0.180
Sleep duration − self-reported (min)	472 ± 106	461 ± 87	**0.002**
Sleep efficiency (%)	92 ± 34	93 ± 16	0.200
Bed time (hours: minutes)[a]	22:44 ± 01:60	22:40 ± 01:40	<**0.001**

Data are shown as mean ± standard deviation (SD) for continuous and percentage for categorical variables. p-Value is by independent t-test for continuous and normally distributed variables (the test is independent for each covariate) (p-value indicates comparison of those included in analytic sample to those excluded) and by Chi-squared for categorical variables.
AHI ≥5, mild OSA; AHI ≥15, moderate OSA; AHI ≥30, severe OSA; AHI, apnea–hypopnea index; BMI, body mass index; ESS >10, excessive daytime sleepiness; ESS, Epworth sleepiness scale; ODI, oxygen desaturation index (with 4% drop in oxygen saturation); OSA, obstructive sleep apnea; PSQI ≥5, poor sleep quality; PSQI, Pittsburgh sleep quality index; SpO_2, oxygen saturation.
Those left blank were not measured by polygraphy.
Bold values represent statistical significance (p-value < 0.05).
[a] Bedtime = median and IQR 25th, 75th (22:50 h and 21:50 h, 23:50 h) for the full sample. Bedtime = median and IQR 25th, 75th (22:50 h and 21:50 h, 23:50 h) for the analytical sample. Bedtime was skewed, so p-value by Mann–Whitney, a non-parametric test.

4. Discussion

Prior evidence supports that sleep duration and quality may be a potentially modifiable risk factor for cardiovascular and metabolic disease. We tested for associations between cardiometabolic variables and poor sleep quality, assessed by the PSQI, controlling for sleep duration and severity of OSA. Our findings showed that: (1) 50% of this rural-based population reported poor sleep quality, even within a normal sleep duration range; (2) higher VLDL cholesterol and triglycerides levels were associated with higher PSQI score (ie, worse sleep quality), even after adjusting for potential confounders; and (3) the PSQI component most significantly associated with both high VLDL and triglyceride blood levels was daytime dysfunction. Notably, our results appear to be independent of OSA as we have not only adjusted for AHI (continuous score), but also tested for these associations excluding those with moderate-to-severe OSA (AHI ≥15). Furthermore, the results remained statistically significant. Thus, our results are in line with the hypothesis that lipid alterations may be early markers of metabolic dysfunction induced by poor sleep quality.

There are complex mechanisms behind the association between sleep duration and cardiovascular risk factors because both short [3,10,26,27] and long sleep duration [12,13] have been associated with cardiometabolic alterations. Studies have shown that obesity is associated with poor sleep quality, sleep fragmentation, and sleep duration [3,4,7]. However, only a few of these studies tested this association in the absence of OSA [8,9,11]. A higher PSQI score has been associated with larger waist circumferences, BMI, and percentage of body fat, as well as with higher serum levels of insulin and glucose [28]. Nonetheless, associations between PSQI and lipids (HDL cholesterol and triglycerides) have been inconsistent, being reported to be both negative [28], and positive [12].

Table 3
Multivariable linear regression analysis testing association of cardiovascular risk factors (outcome) and Pittsburgh sleep quality index (main predictor).

Outcome variable	PSQI total score			PSQI <5 vs ≥ 5		
	Beta	SE	p	Beta	SE	p
VLDL-cholesterol (mg/dL)	0.392	0.156	**0.012**	1.763	0.980	0.072
ln Triglycerides (mg/dL)	0.017	0.006	**0.006**	0.071	0.040	0.071
HDL-cholesterol (mg/dL)	−0.194	0.155	0.208	−0.897	0.973	0.356
LDL-cholesterol (mg/dL)	0.082	0.500	0.870	1.886	3.120	0.546
Total cholesterol (mg/dL)	0.495	0.561	0.377	2.932	3.532	0.407
Cholesterol ratio	0.033	0.017	0.060	0.195	0.110	0.077
Glucose (mg/dL)	0.180	0.247	0.468	−0.601	1.560	0.700
HbA1c (%)	−0.015	0.009	0.102	−0.066	0.056	0.238
Systolic BP (mmHg)[a]	0.073	0.152	0.634	0.516	0.961	0.613
Diastolic BP (mmHg)[a]	0.060	0.116	0.602	0.290	0.731	0.693

AHI, apnea–hypopnea index (number of events per hour of sleep); BMI, body mass index; BP, blood pressure; cholesterol ratio, total cholesterol/HDL; ESS, Epworth sleepiness scale; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ln, natural logarithm transformation; OSA, obstructive sleep apnea; PSQI ≥5, poor sleep quality; PSQI, Pittsburgh sleep quality index; SE, standard error; VLDL, very-low density lipoprotein.
Each line represents a unique regression model. Adjustments for age, sex (female = ref), race (White vs others), Wage categories (up to 5 MW vs > 5 MW and >5 MW as ref), Education categories (illiterate vs high school vs technical/graduation and illiterate as ref), smoking status (never as ref), systolic BP, BMI, sleep duration (min); ESS continuous score, bedtime (h), and OSA by AHI continuous score. Missing data were excluded (all complete for regression analysis = 554).
Bold values represent statistical significance (p-value < 0.05).
[a] Not included systolic BP in adjustments.

Table 4
Restricted analysis for those without obstructive sleep apnea (apnea–hypopnea index <15), testing the association between lipid components (outcome) and sleep quality ($N = 472$).

	VLDL-cholesterol (mg/dL)			ln Triglycerides (mg/dL)		
	Beta	SE	p-value	Beta	SE	p-value
PSQI total score	0.446	0.170	**0.009**	0.018	0.007	**0.010**
PSQI cut-off 5	2.188	1.037	**0.035**	0.084	0.426	**0.048**

AHI, apnea–hypopnea index; BMI, body mass index; BP, blood pressure. ESS, Epworth sleepiness scale; ln, natural logarithm transformation; OSA, obstructive sleep apnea; PSQI ≥5, poor sleep quality; PSQI, Pittsburgh sleep quality index; SE, standard error; VLDL, very-low density lipoprotein cholesterol.
Adjustments: age, sex (female = ref), race (White vs others), Wage categories (up to 5 MW vs > 5 MW and >5 MW as ref), Education categories (illiterate vs high school vs technical/graduation and illiterate as ref), smoking status (never as reference), BMI, ESS continuous score, sleep duration (min), bedtime (h), and daytime systolic BP. Missing data were excluded. All complete for regression analysis ($N = 423$).
Bold values represent statistical significance (p-value < 0.05).

Table 5
Restricted analysis for those without lipid-lowering medication ($N = 510$) testing the association between lipid components (outcome) and sleep quality.

	VLDL-cholesterol (mg/dL)			ln Triglycerides (mg/dL)		
	Beta	SE	p	Beta	SE	p
PSQI total score	0.426	0.161	**0.008**	0.019	0.006	**0.004**
PSQI cut-off 5	1.681	1.013	0.097	0.080	0.040	**0.047**

AHI, apnea–hypopnea index; BMI, body mass index; BP, blood pressure; ESS, Epworth sleepiness scale; ln, natural logarithm transformation; PSQI ≥5, poor sleep quality; PSQI, pittsburgh sleep quality index; SE, standard error; VLDL, very-low density lipoprotein cholesterol.
Adjustments: age, sex (female = ref), race (White vs others), Wage categories (up to 5 MW vs > 5 MW and >5 MW as ref), Education categories (illiterate vs high school vs technical/graduation and illiterate as ref), smoking status (never as reference), BMI, ESS continuous score, sleep duration (min), bedtime (h), AHI, and daytime systolic BP. Missing data were excluded. All complete for regression analysis ($N = 462$).
Bold values represent statistical significance (p-value < 0.05).

In the scenario of the controversy about the association between lipid profile and sleep quality [12,28], the CARDIA study showed that a higher PSQI score was not significantly associated with a poorer lipid profile [13]. The poorer lipid profile was statistically associated only with sleep duration [13]. However, VLDL cholesterol levels were not reported in the CARDIA study, as we explored in this current study. In addition, there are several other mechanisms, besides sleep duration, that have been enrolled in the association between sleep disturbances and poor lipid profile [29,30]. For instance, sleep fragmentation, classified as the number of microarousals during sleep, has been associated with increased levels of lipids, cortisol, and blood pressure [29]. A reasonable hypothesis to be considered is that poor sleep quality may be an epiphenomenon of other sleep-related metabolic changes, such as sleep fragmentation [29]. Our results are consistent with these findings. Our findings demonstrated that other sleep disturbances besides OSA [5–7,13,31–35] and sleep duration [12,13,26,27], specifically sleep quality, may be potential risk factors for poor lipid profiles. A clear understanding of the biological mechanisms linking these sleep disturbances and higher VLDL cholesterol levels remains to be identified.

PSQI is a commonly used subjective sleep quality instrument [18,19,36]. Despite the limitations due to its subjective nature, it has also been considered a tool that adds significant contributions to the general quality of life (QoL) evaluation [36]. Our findings showed that the daytime dysfunction domain significantly associated with high levels of both VLDL cholesterol and triglycerides, so it seems to be in line with a previous report regarding the idea that sleep may have significant impact on QoL, even in the absence of sleep-disordered breathing or other health problems, such as lipid alterations [37].

Our study has some limitations. First, the cross-sectional nature does not allow causal inference. Second, although we adjust for major confounders, there may still be residual confounding due to incomplete adjustments and unknown confounders. Sleep medication use was analyzed separately, as it formed a component of the PSQI questionnaire, and is shown in the Supplementary material. We did not ascertain type of sleep medication or duration of use. Similarly, hypnotic medication was not taken into account separately in the current analysis. In addition, the subjective nature of the questionnaires should be carefully evaluated. Finally, overnight polygraphy was performed through a type III portable sleep study, which does not allow sleep duration to be objectively measured. Thus, sleep duration in the current study was self-reported. Moreover, portable overnight polygraphy may underdiagnose OSA [38,39], because there are no brain channels to assess respiratory events related to arousals. Therefore, our results should be interpreted with caution.

5. Conclusion

Our findings revealed that poor sleep quality was independently associated with poorer lipid profile, specifically higher VLDL and triglycerides. Poor sleep quality may reflect an unhealthy lifestyle, but also may be an epiphenomenon of other sleep disturbances, beyond OSA and sleep duration. Our findings were robust even after adjusting for socio-economic status, but possible residual confounding caused by unknown factors should be considered in an observational study. An objectively measured sleep evaluation, by a full polysomnography, may shed light on the mechanistic pathways by which poor sleep quality may lead to elevated lipid blood levels.

Author contributions

Concept and design: G.R.G., G.L.F., and A.C.P. Analysis and interpretation of data: G.R.G., A.C.P., K.L.K., and M.v.S. Drafting the article or revising it critically for important intellectual content: G.R.G., G.L.F., L.K. de P., C.M.O., R.O.A., F.B., A.B.N., M.v.S., K.L.K., J.E.K., and A.C.P. Final approval: all authors.

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Conflict of interest

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2018.12.028.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2018.12.028.

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