

Association between Sleep Efficiency and Dyslipidemia in Adults Aged 40-65 Years: Postprint

Authors: Wang Die, Wu Bangyun, Tan Cunyao, Chen Shihui, Li You, Meng Yue, Wang Dashan, Hu Jin, Wang Ziyun, Wang Junhua, Wang Junhua

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Abstract

Background: The decline in sleep efficiency (SE) among middle-aged and elderly individuals may affect their blood lipid metabolism, necessitating further in-depth analysis. **Objective:** To analyze the association between SE and different blood lipid indicators in a population aged 40-65 years, explore the mediating role of central obesity in the relationship between SE and blood lipids, and provide a reference basis for investigating the pathways through which SE leads to dyslipidemia and for the prevention and treatment of dyslipidemia. **Methods:** From March to November 2022, face-to-face surveys were conducted on subjects who met the inclusion and exclusion criteria at the Physical Examination Center of Fuquan First People's Hospital, comprising three components: questionnaire survey, physical examination, and laboratory indicator testing. A directed acyclic graph (DAG) was used to identify the minimum set of control variables required for adjusting the association between SE and dyslipidemia: age, sex, smoking, alcohol consumption, family income, physical activity, and education level. Unconditional binary Logistic regression analysis was employed to explore the association between SE and different dyslipidemia indicators. **Results:** This study ultimately included 1,095 participants, grouped according to the presence or absence of dyslipidemia. Statistically significant differences were observed between the two groups in terms of sex, smoking, alcohol consumption, physical activity, waist circumference, hip circumference, and waist-to-hip ratio (WHR) ($P < 0.05$). After adjusting for control variables, compared with high SE, individuals with low SE had an increased risk of hypertriglyceridemia (OR=1.35, 95%CI=1.03~1.77), while no association was found between SE and other blood lipid indicators. Mediation effect analysis revealed that WHR played a partial mediating role in the association between SE and hypertriglyceridemia ($\beta=0.019$, 95%CI=0.005~0.034), with the mediating effect accounting for 32.76% of the total effect. WHR exhibited a masking effect in the association between SE and low HDL cholesterolemia. **Conclusion:** WHR may be an important pathway in

the association between low SE and hypertriglyceridemia.

Full Text

Association between Sleep Efficiency and Dyslipidemia among Adults Aged 40 to 65

WANG Die¹, WU Bangyun², TAN Cunyao², SHEN Shihui², LI You¹, MENG Yue¹, WANG Dashan¹, HU Jin¹, WANG Ziyun¹, WANG Junhua^{1*}

¹School of Public Health, Guizhou Medical University/the Key Laboratory of Environmental Pollution Monitoring and Disease Control, Gui'an New Area 561113, China

²Fuquan Center for Disease Control and Prevention, Fuquan 550599, China

Corresponding author: WANG Junhua, Associate professor; E-mail: gywangjunhua@qq.com

Abstract

Background: Lipid metabolism in middle-aged and older adults may be influenced by decreased sleep efficiency (SE), but their association requires further in-depth analysis. **Objective:** To analyze the association between sleep efficiency (SE) and different lipid indices in individuals aged 40–65 years, and to explore the mediating role of central obesity in the association between SE and lipid profiles, thereby providing a reference for understanding the pathways through which SE contributes to dyslipidemia and for its prevention and treatment. **Methods:** From March to November 2022, a face-to-face investigation was conducted at the physical examination center of Fuquan First People's Hospital. Unconditional binary logistic regression was used to analyze the association between SE and different lipid indicators, and a mediation model was used to analyze the mediating effect of waist-hip ratio (WHR). **Results:** A total of 1,095 subjects were included in this study and divided into two groups according to the presence or absence of dyslipidemia. There were statistically significant differences between the two groups in gender, smoking, drinking, exercise, waist circumference, hip circumference, and waist-hip ratio ($P < 0.05$). After adjusting for control variables, the low SE group showed an increased risk of hypertriglyceridemia compared with the high SE group (OR=1.35, 95%CI=1.03–1.77). No statistically significant association was found between SE and other lipid indicators. Mediation analysis showed that WHR had a partial mediating effect in the association between SE and hypertriglyceridemia ($\beta=0.019$, 95%CI=0.005–0.034), accounting for 32.76% of the total effect. WHR exhibited a masking effect on the association between SE and low HDL cholesterolemia. **Conclusion:** Central obesity may be an important pathway in the association between low SE and hypertriglyceridemia.

Keywords: Sleep efficiency; Dyslipidemias; Association; Middle aged

Introduction

Dyslipidemia is a major cause of cardiovascular disease [1], and its prevalence among adults in China has remained high in recent years [2-3]. Therefore, prevention of dyslipidemia should be prioritized. Sleep problems represent one influential factor, with previous studies demonstrating associations between sleep efficiency (SE) and high-density lipoprotein cholesterol (HDL) [4] and triglycerides (TG) [5-6] in adolescents, though no association between SE and TG was found in older adults [7].

Low SE is a common sleep problem among middle-aged and older adults, yet research on its association with blood lipids remains limited. The underlying mechanisms are also unclear. WU et al. found that individuals with low SE had a 2.85-fold increased probability of obesity compared with those with normal SE [8], and dyslipidemia rates are higher in obese patients than in non-obese individuals [9]. Central obesity demonstrates a stronger correlation with the overall metabolic effects of insulin resistance than general obesity [10], suggesting that central obesity may mediate the relationship between SE and lipid indicators. Meta-analysis results show that SE decreases with age, particularly after 40 years [11]. While most existing research on sleep and dyslipidemia has focused on sleep duration, studies specifically examining SE remain relatively scarce. SE, calculated as the ratio of total sleep time to time in bed, accurately reflects effective sleep duration. Research indicates that individuals with low SE typically experience sleep fragmentation—intermittent, disrupted sleep patterns—and SE thus indirectly captures sleep interruption [12]. Therefore, this study analyzes the association between SE and different lipid indicators among 40–65-year-olds and explores the mediating role of obesity in the SE-lipid relationship to provide a reference for elucidating the mechanisms through which SE contributes to dyslipidemia.

Methods

Study Population This study utilized baseline survey data from a prospective cohort study on sleep patterns and their association with arteriosclerosis among middle-aged and elderly populations in Guizhou Province. The research protocol was approved by the Guizhou Medical University Human Research Ethics Committee [Approval No. 2021(174)]. Participants included individuals aged 40–65 years undergoing routine physical examinations at the Fuquan First People’s Hospital Physical Examination Center, with lipid testing as part of their examination panel. Detailed inclusion and exclusion criteria have been described previously [13]. From March to November 2022, a total of 1,104 individuals were surveyed. Nine participants were excluded: two had incomplete

examination data (missing lipid profiles), two worked night shifts with atypical sleep schedules, and five had fasting durations shorter than 8 hours. The final study sample comprised 1,095 participants.

Data Collection and Measures The investigation consisted of three components: questionnaire survey, physical examination, and laboratory testing. (1) A self-designed questionnaire administered through face-to-face interviews collected general demographic and socioeconomic information, health-related behaviors (smoking, alcohol consumption), and sleep patterns. (2) Waist circumference (WC, measured at the midpoint between the upper border of the iliac crest and the lower border of the rib arch at the midaxillary line) and hip circumference (HC, measured at the maximum gluteal protuberance) were measured using tape measures with precision to 0.1 cm, and waist-hip ratio (WHR) was calculated. (3) Total cholesterol (TC), triglycerides (TG), HDL cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were measured using a Beckman AU5800 automatic biochemical analyzer. (4) Definitions of relevant indicators are provided in Table 1 .

Quality Control The research team developed a comprehensive survey manual and conducted unified training for all investigators before the formal survey, including a pilot study. To ensure data reliability and completeness, questionnaires were cross-checked on the day of collection, and participants were contacted promptly to address any missing, erroneous, or illogical information. Data were double-entered using EpiData 3.1 software.

Statistical Analysis Statistical analyses were performed using R version 4.3.1. Normally distributed continuous variables were expressed as mean \pm standard deviation and compared between groups using t-tests. Non-normally distributed continuous variables were presented as median (P25, P75) and compared using Wilcoxon rank-sum tests. Categorical data were expressed as frequencies and percentages, with between-group comparisons conducted using chi-square tests.

A directed acyclic graph (DAG) identified the minimum set of control variables required for analyzing the association between SE and dyslipidemia: age, gender, smoking, alcohol consumption, household income, physical activity, and education level. Unconditional binary logistic regression was employed to examine the association between SE and various dyslipidemia indicators. Mediation analysis was conducted using the simple mediation model (Process function, model 4) from the “bruceR” package in R. The coefficient product method was used for testing, with bootstrap estimation of the 95% confidence interval for the indirect effect (ab). A 95%CI that did not include zero indicated a significant mediating effect, and the proportion mediated (ratio of indirect effect to total effect) was calculated [21].

Results

Basic Characteristics of Study Participants A total of 1,095 participants were included, comprising 609 males (55.6%) and 486 females (44.4%). Based on dyslipidemia status, 604 participants (55.2%) were classified into the dyslipidemia group and 491 (44.8%) into the normal lipid group. Statistically significant differences between the two groups were observed for gender, smoking, alcohol consumption, physical activity, waist circumference, hip circumference, and waist-hip ratio ($P < 0.05$). No significant differences were found for age, education level, household income, or SE ($P > 0.05$). Detailed characteristics are presented in Table 2 .

Association Between SE and Dyslipidemia Indicators Unconditional binary logistic regression analysis was performed with dyslipidemia, hypertriglyceridemia, low HDL cholesterolemia, high LDL cholesterolemia, and high TC cholesterolemia as dependent variables and SE as the independent variable. Variable assignments are shown in Table 3 . In the unadjusted model, SE was not associated with overall dyslipidemia ($P > 0.05$) but was associated with hypertriglyceridemia ($P < 0.05$). After adjusting for control variables, SE remained significantly associated only with hypertriglyceridemia ($P < 0.05$). Compared with the high SE group, individuals with low SE had an increased risk of hypertriglyceridemia (OR=1.35, 95%CI=1.03-1.77). No statistically significant associations were found between SE and other lipid indicators ($P > 0.05$). These results are illustrated in the forest plot in Figure 1 [Figure 1: see original paper].

Mediation Analysis Results Further analysis examined the role of central obesity in the association between SE and each dyslipidemia indicator using bootstrap mediation analysis. The simple mediation model “SE \rightarrow WHR \rightarrow hypertriglyceridemia” revealed a total effect of SE on TG of $\beta = 0.058$ (95%CI=0.002-0.113), a direct effect of $\beta = 0.039$ (95%CI=-0.016-0.092), and an indirect effect of $\beta = 0.019$ (95%CI=0.005-0.034). The proportion mediated was 32.76%, indicating that WHR partially mediated the effect of SE on hypertriglyceridemia risk (Figure 2B [Figure 2: see original paper]).

The “SE \rightarrow WHR \rightarrow HDL” model showed a non-significant total effect of SE on HDL [$\beta = -0.018$ (95%CI=-0.069-0.034)], with a direct effect of $\beta = -0.034$ (95%CI=-0.084-0.017) and an indirect effect of $\beta = 0.016$ (95%CI=0.004-0.029). The opposite signs of the direct and indirect effects suggest that WHR may exert a masking (suppressing) effect on the association between SE and low HDL cholesterolemia (Figure 2C [Figure 2: see original paper]). WHR did not demonstrate a statistically significant mediating effect in the associations between SE and overall dyslipidemia, high TC cholesterolemia, or high LDL cholesterolemia (Figure 2A, 2D, 2E [Figure 2: see original paper]).

Discussion

This study found a negative association between SE and hypertriglyceridemia among 40-65-year-olds, consistent with previous research findings [22]. Studies have demonstrated that SE affects insulin sensitivity [23], and in states of insulin resistance, TG levels increase while HDL levels decrease [24-25]. Previous research has also found that high TC and high LDL are not associated with insulin resistance [26-27], which may partially explain why this study did not detect associations between SE and TC or LDL. Another reason for the lack of observed associations between SE and lipid indicators other than TG may be the higher prevalence of hypertriglyceridemia compared with other dyslipidemia types in our study population; future studies with larger sample sizes are needed to investigate these relationships.

This study found that WHR partially mediates the association between SE and hypertriglyceridemia. Low SE may alter hypothalamic-pituitary-adrenal axis function, leading to increased cortisol levels [28]. Cortisol plays an important role in adipocyte formation and, in addition to affecting fat deposition, can redistribute adipose tissue to the abdominal region [29], thereby causing central obesity. Central obesity may influence hypertriglyceridemia through increased lipolytic activity in visceral adipocytes, elevated insulin and pro-inflammatory cytokine levels, and increased sympathetic nervous system activity [30], as well as through increased release of inflammatory cytokines [interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α)] that stimulate lipid release and affect blood lipid levels. Therefore, central obesity may be a key factor in preventing and controlling hypertriglyceridemia among individuals with low SE, and controlling central obesity may help reduce hypertriglyceridemia risk in this population. The mediation proportion of 32.76% suggests that additional mediating pathways likely exist between SE and hypertriglyceridemia.

This study also found that WHR may have a masking effect on the association between SE and HDL abnormalities. Specifically, while no direct association was observed between SE and HDL, when WHR was included as a mediator, SE showed an indirect effect on HDL abnormalities, with direct and indirect effects having opposite signs. This phenomenon may occur because low SE alters hypothalamic-pituitary-adrenal axis function, affecting fat deposition and redistributing adipose tissue to the abdominal region [29], thereby causing central obesity. Central obesity is more closely associated with the overall metabolic effects of insulin resistance [10], which can alter lipoprotein quantity and composition, leading to abnormal lipid levels and affecting HDL metabolism [9]. This study did not directly detect an association between SE and HDL but found that low SE can affect HDL through the central obesity pathway. Previous studies have found that sleep deprivation may increase caloric intake and weight gain by mediating fluctuations in appetite-regulating neuropeptides (e.g., leptin and ghrelin) [31], and that sleep deprivation causes fatigue, leading to reduced physical activity and potential weight gain [32]. These physiological disturbances may result in elevated TC and TG levels, thereby increasing dyslipidemia risk.

Compared with insufficient sleep duration, low SE provides a more precise measure of sleep deficiency arising from internal cognitive or biobehavioral processes, whereas sleep duration may be constrained by daily schedules and does not necessarily reflect endogenous sleep deficiency [33]. Therefore, SE should be emphasized as a sleep metric with potential predictive value for cardiovascular outcomes. The lack of statistically significant association between SE and HDL in this study may be due to insufficient sample size. However, the findings suggest that SE can affect HDL through mediating pathways, and ignoring these pathways might overlook the impact of low SE on HDL indicators and the importance of controlling WHR as a potential strategy for reducing low HDL cholesterolemia. Future research on SE and lipid indicators should expand sample sizes and examine the influence of mediating pathways.

In summary, WHR may represent an important pathway in the association between low SE and hypertriglyceridemia. However, this study has limitations: it is a cross-sectional study that provides exploratory findings on the association between SE and dyslipidemia and its mechanistic pathways, which require verification through prospective cohort studies.

References

- [1] LIU Lu, LIU Yinghua. Nutritional intervention strategies for hyperlipidemia[J]. *China Medical News*, 2021, 36(9):11. DOI:10.3760/cma.j.issn.1000-8039.2021.09.112.
- [2] SONG P K, MAN Q Q, LI H, et al. Trends in lipids level and dyslipidemia among Chinese adults, 2002-2015[J]. *Biomed Environ Sci*, 2019, 32(8):559-570. DOI:10.3967/bes2019.074.
- [3] PAN L, YANG Z H, WU Y, et al. The prevalence, awareness, treatment and control of dyslipidemia among adults in China[J]. *Atherosclerosis*, 2016, 248:2-9. DOI:10.1016/j.atherosclerosis.2016.02.006.
- [4] CESPEDES FELICIANO E M, QUANTE M, RIFAS-SHIMAN S L, et al. Objective sleep characteristics and cardiometabolic health in young adolescents[J]. *Pediatrics*, 2018, 142(1):e20174085. DOI:10.1542/peds.2017-4085.
- [5] WU N N, JAMNIK V K, KOEHLE M S, et al. Associations between sleep characteristics and cardiovascular risk factors in adolescents living with type 1 diabetes[J]. *J Clin Med*, 2022, 11(18):5295. DOI:10.3390/jcm11185295.
- [6] SIMON S, RAHAT H, CARREAU A M, et al. Poor sleep is related to metabolic syndrome severity in adolescents with PCOS and obesity[J]. *J Clin Endocrinol Metab*, 2020, 105(4):e1827-1834. DOI:10.1210/clinem/dgz285.
- [7] LIU X, HUANG L M, WU Q, et al. Sleep characteristic profiles and the correlation with spectrum of metabolic syndrome among older adult: a cross-sectional study[J]. *BMC Geriatr*, 2022, 22(1):414. DOI:10.1186/s12877-022-03074-8.
- [8] KIM M. Association between objectively measured sleep quality and obesity in community-dwelling adults aged 80 years or older: a cross-sectional study[J].

- J Korean Med Sci, 2015, 30(2):199-206. DOI:10.3346/jkms.2015.30.2.199.
- [9] ZHU J J, ZHANG Y, WU Y L, et al. Obesity and dyslipidemia in Chinese adults: a cross-sectional study in Shanghai, China[J]. *Nutrients*, 2022, 14(11):2321. DOI:10.3390/nu14112321.
- [10] WU H Z, BALLANTYNE C M. Metabolic inflammation and insulin resistance in obesity[J]. *Circ Res*, 2020, 126(11):1549-1564. DOI:10.1161/CIRCRESAHA.119.315896.
- [11] OHAYON M M, CARSKADON M A, GUILLEMINAULT C, et al. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan[J]. *Sleep*, 2004, 27(7):1255-1273. DOI:10.1093/sleep/27.7.1255.
- [12] REED D L, SACCO W P. Measuring sleep efficiency: what should the denominator be?[J]. *J Clin Sleep Med*, 2016, 12(2):263-266. DOI:10.5664/jcsm.5498.
- [13] GAI Yun, WU Bangyun, TAN Cunyao, et al. Association between sleep chronotype and cognitive function in routine physical examination population aged 40-65 years[J]. *Modern Preventive Medicine*, 2023, 50(16):3031-3037. DOI:10.20043/j.cnki.MPM.202304356.
- [14] MAZZA M, LAPENTA L, LOSURDO A, et al. Polysomnographic and psychometric correlates of napping in primary insomnia patients[J]. *Nord J Psychiatry*, 2020, 74(4):244-250. DOI:10.1080/08039488.2019.1695285.
- [15] OHAYON M, WICKWIRE E M, HIRSHKOWITZ M, et al. National Sleep Foundation's sleep quality recommendations: first report[J]. *Sleep Health*, 2017, 3(1):6-19. DOI:10.1016/j.sleh.2016.11.006.
- [16] Joint Committee for the Revision of Chinese Guidelines for the Prevention and Treatment of Dyslipidemia in Adults. Chinese guidelines for the prevention and treatment of dyslipidemia in adults (2016 revision)[J]. *Chinese Circulation Journal*, 2016, 31(10):937-950. DOI:10.3969/j.issn.1000-3614.2016.10.001.
- [17] LIU Yin, ZHANG Shengchao. Correlation between hyperlipidemia and waist circumference, waist-to-height ratio, and waist-to-hip ratio in chronic disease population[J]. *Chinese Journal of Gerontology*, 2021, 41(22):5135-5140. DOI:10.3969/j.issn.1005-9202.2021.22.075.
- [18] OU Wensen, WU Wenlin, WANG Jinming, et al. Analysis of smoking and smoking cessation status among adults in the Pearl River Delta region of Guangdong Province[J]. *Chinese General Practice*, 2017, 20(33):4188-4193. DOI:10.3969/j.issn.1007-9572.2017.00.064.
- [19] FANG Yuehui, HE Yuna, BAI Guoyin, et al. Analysis of drinking status among Chinese men aged 20-79 years in 2012[J]. *Chinese Journal of Epidemiology*, 2018, 39(3):280-285. DOI:10.3760/cma.j.issn.0254-6450.2018.03.006.
- [20] Healthy China Action Promotion Committee releases "Healthy China Action (2019-2030)" [J]. *Health World*, 2019(12):6-9.
- [21] DING Xiaoyun, JIN Juzhen, YANG Jin, et al. Mediating effect of waist circumference and fasting blood glucose on the association between obstructive sleep apnea syndrome and arteriosclerosis in population aged 40-65 years[J]. *Chinese General Practice*, 2023, 26(21):2597-2602. DOI:10.12114/j.issn.1007-9572.2023.0119.
- [22] FENG N N, YANG J D, XU H J, et al. The associations between sleep architecture and metabolic parameters in patients with obstructive

- sleep apnea: a hospital-based cohort study[J]. *Front Neurol*, 2021, 12:606031. DOI:10.3389/fneur.2021.606031.
- [23] DORENBOS E, RIJKS J M, ADAM T C, et al. Sleep efficiency as a determinant of insulin sensitivity in overweight and obese adolescents[J]. *Diabetes Obes Metab*, 2015, 17(Suppl 1):90-98. DOI:10.1111/dom.12515.
- [24] RASHID S, WATANABE T, SAKAUE T, et al. Mechanisms of HDL lowering in insulin resistant, hypertriglyceridemic states: the combined effect of HDL triglyceride enrichment and elevated hepatic lipase activity[J]. *Clin Biochem*, 2003, 36(6):421-429. DOI:10.1016/s0009-9120(03)00078-x.
- [25] KINUHATA S, HAYASHI T, SATO K K, et al. Sleep duration and the risk of future lipid profile abnormalities in middle-aged men: the Kansai Healthcare Study[J]. *Sleep Med*, 2014, 15(11):1379-1385. DOI:10.1016/j.sleep.2014.06.011.
- [26] YAMADA N, YOSHINAGA H, SAKURAI N, et al. Increased risk factors for coronary artery disease in Japanese subjects with hyperinsulinemia or glucose intolerance[J]. *Diabetes Care*, 1994, 17(2):107-114. DOI:10.2337/diacare.17.2.107.
- [27] HAFNER S M, VALDEZ R A, HAZUDA H P, et al. Prospective analysis of the insulin-resistance syndrome (syndrome X)[J]. *Diabetes*, 1992, 41(6):715-722. DOI:10.2337/diab.41.6.715.
- [28] MASSAR S A A, LIU J C J, MOHAMMAD N B, et al. Poor habitual sleep efficiency is associated with increased cardiovascular and cortisol stress reactivity in men[J]. *Psychoneuroendocrinology*, 2017, 81:151-156. DOI:10.1016/j.psyneuen.2017.04.013.
- [29] VAN ROSSUM E F C. Obesity and cortisol: New perspectives on an old theme[J]. *Obesity*, 2017, 25(3):500-501. DOI:10.1002/oby.21774.
- [30] PAL S, RADAPELLI-BAGATINI S. Association of arterial stiffness with obesity in Australian women: a pilot study[J]. *J Clin Hypertens*, 2013, 15(2):118-123. DOI:10.1111/jch.12038.
- [31] GRANDNER M A, PATEL N P, GEHRMAN P R, et al. Problems associated with short sleep: bridging the gap between laboratory and epidemiological studies[J]. *Sleep Med Rev*, 2010, 14(4):239-247. DOI:10.1016/j.smrv.2009.08.001.
- [32] SPIEGEL K, LEPROULT R, VAN CAUTER E. Impact of sleep debt on metabolic and endocrine function[J]. *Lancet*, 1999, 354(9188):1435-1439. DOI:10.1016/S0140-6736(99)01376-8.
- [33] EIMAN M N, POMEROY J M L, WEINSTEIN A A. Relationship of actigraphy-assessed sleep efficiency and sleep duration to reactivity to stress[J]. *Sleep Sci*, 2019, 12(4):257-264. DOI:10.5935/1984-0063.20190090.

Author Contributions: WANG Die was responsible for conceptualizing the manuscript framework, data analysis, and writing. WU Bangyun, TAN Cunyaoyao, and SHEN Shihui coordinated the fieldwork and conducted surveys. LI You, MENG Yue, and WANG Dashan were responsible for data verification, organization, and entry. HU Jin and WANG Ziyun assisted with topic selection and study design. WANG Junhua reviewed and revised the manuscript.

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