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Original Article

Sleep duration inversely correlates with glucolipid metabolism in patients with new-onset type 2 diabetes mellitus

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Abstract: Objective: To study the correlation between sleep duration and glucolipid metabolism in patients with new-onset type 2 diabetes mellitus (NOT2DM). Methods: The participants were 168 NOT2DM patients. Glucolipid metabolism indices were compared among the short (n=59), middle (n=56), and long sleep groups (n=53), including fasting blood glucose (FBG), glycosylated hemoglobin (HbA1c), total cholesterol (TC), and triglyceride (TG). The correlation of sleep duration with glucolipid metabolism, glycemic control, and body fat distribution (abdominal total fat area [TFA] and visceral fat area [VFA]) was assessed. Finally, the contributors to poor glycemic control were explored. Results: Glucolipid metabolism indices tended to decrease with increasing sleep duration in NOT2DM patients, suggesting a significant negative correlation between sleep duration and glucolipid metabolism. The sleep duration of the good glycemic control group was significantly longer. A significant positive correlation was identified between sleep duration and good glycemic control, while an inverse association was determined between sleep duration and TFA. Body mass index (BMI), sleep duration, FBG, TG, and TFA were all independent contributors to poor glycemic control. Conclusions: Sleep duration in NOT2DM patients was inversely related to glucolipid metabolism and TFA and positively correlated with good glycemic control. High BMI, FBG, TG, and TFA, as well as short sleep duration, may increase the risk of poor glycemic control.

Keywords: Type 2 diabetes mellitus, sleep duration, glycolipid metabolism, correlation study

Introduction

With improvements in the living environment and lifestyle, as well as the intensification of population aging, diabetes mellitus (DM), as a metabolic disease, is on the rise, along with obesity and tumors [1]. Type 2 DM (T2DM) represents most DM cases, with up to 90% of DM patients having T2DM. Currently, DM affects 10.5% of the global population to varying degrees, and the risk of developing DM among adolescents is gradually increasing [2, 3]. At present, T2DM is mainly managed by lifestyle interventions, dietary modification plus physical activity, medication, medical equipment, and bariatric surgery, but all have less than satisfactory therapeutic outcomes [4, 5]. Therefore, continuous exploration is needed to optimize treatment and understand the pathogenesis of DM to develop more suitable and efficient management plans.

The pathogenesis of T2DM involves pancreatic β cell dysfunction and relative insulin deficiency resulting from insulin resistance. In addition, abnormal glucolipid metabolism and long-term chronic inflammation caused by obesity are also important contributors [6-9]. Sleep and circadian rhythm disturbances adversely affect glucose metabolism and T2DM, leading to abnormalities in glucose metabolism and a 20 to 30 percent decrease in insulin sensitivity, thereby increasing the risk of T2DM (up to 50 percent) [10, 11]. As reported by Wan Mahmood et al. [12], sleep disruption has an independent negative effect on lipid metabolism in T2DM patients, manifested mainly as significantly higher total cholesterol (TC) and triglyceride (TG) levels in patients with short sleep duration. Short sleep duration has also been associated with a higher body mass index (BMI) in T2DM patients, and there is a U-shaped correlation between sleep duration and TG/high-density

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lipoprotein cholesterol (HDL-C) [13]. The above all suggest a certain correlation between sleep duration and lipid metabolism in T2DM patients. There are also reports indicating that short sleep duration in adults is closely related to an increase in visceral obesity, and whether increasing sleep duration can help reduce the risk of visceral obesity remains controversial [14, 15].

Despite existing literature reporting the correlation between sleep, type 2 diabetes, glucolipid metabolism, and body fat, this study remains innovative in the following aspects: First, focusing on new-onset T2DM (NOT2DM) patients, the study excluded the confounding effects of long disease duration and complex medication history on the relationship between sleep and metabolism, thereby providing more robust evidence of the association at the disease onset stage. Second, this study integrated glucolipid metabolism, glycemic control, and body fat distribution to evaluate systematically the correlation between sleep duration and multi-dimensional metabolic phenotypes. Based on the above, we decided to explore the association of sleep duration with glucolipid metabolism and body fat distribution in patients with new-onset T2DM (NOT2DM), aiming to provide more clinical suggestions for its prevention and treatment.

Materials and methods

General information

168 patients with NOT2DM who received treatment at our hospital between May 2022 and December 2025 were selected. According to the sleep duration, they were divided into short (59 cases with a sleep duration <6 hours), middle (56 cases with a sleep duration within 6-8 hours), and long (53 cases with a sleep duration >8 hours) sleep groups. The mean ages (years) of the short, middle, and long sleep groups were (53.39±8.8), (50.27±9.81), and (52.43±9.36), respectively, with the corresponding number of cases of male patients being 32, 29, and 30. The three groups were clinically comparable, as no marked multi-group differences were found in general data (P>0.05). This research was reviewed and approved by the Hunan University of Traditional Chinese Medicine ethics committee.

Criteria for patient enrollment and exclusion

Inclusion criteria: Treatment-naive patients with newly diagnosed T2DM who agreed to participate, had stable weight for at least 3 months before enrollment, and had normal communication and cognitive abilities were included.

Exclusion criteria: Patients who were using hypoglycemic drugs or receiving other traditional Chinese medicine hypoglycemic treatments, pregnant or lactating women, and those who had been treated with antibiotics, hormones, immunosuppressants, and microecological preparations within the last 3 months were excluded; in addition, patients with DM emergencies, other serious primary diseases, severe heart, lung, kidney, and endocrine system diseases, psychiatric disorders, or psychological diseases were also excluded.

Detection indicators

After fasting for 10 hours, blood was drawn on an empty stomach in the early morning to obtain patients' serum samples. Fasting blood glucose (FBG) was measured using glucose oxidase with an automatic biochemical analyzer. Glycosylated hemoglobin (HbA1c) determination used ion-exchange liquid chromatography with a glycosylated hemoglobin analyzer. TC and TG were quantified by an automatic biochemical analyzer. The abdominal total fat area (TFA) and visceral fat area (VFA) were measured by dual-energy X-ray absorptiometry.

Statistical analyses

The data of this study were analyzed by SPSS20.0. The data were tested for normality by the Shapiro-Wilk method and homogeneity of variance by Levene's test. If both conditions were met, the measured data were reported as mean ± standard deviation (SD) and examined for differences among multiple groups of means using one-way analysis of variance, with subsequent pairwise comparisons made using the Bonferroni method. Data deviating from normality or homogeneity of variance were shown as median (interquartile range), with multi-group comparisons conducted using the Kruskal-Wallis H test, and subsequent pairwise comparisons performed using a Bonferroni correction for *P*-value adjustment. For count data, the number of cases/percentage (n/%) was used for statistical description and the χ^2 test for comparison. The correlation of sleep duration

Table 1. Analysis of basic characteristics of three groups of patients

Characteristics	Short sleep group (n=59)	Middle sleep group (n=56)	Long sleep group (n=53)	χ^2/F/H	P
Male	32 (54.24)	29 (51.79)	30 (56.60)	0.255	0.880
Age (years old)	53.39±8.80	50.27±9.81	52.43±9.36	1.674	0.191
Weight (kg)	69.00 (61.00, 74.00)	71.00 (65.00, 74.75)	70.00 (62.00, 74.50)	2.134	0.344
Body mass index (kg/m^2)	28.00 (25.00, 31.00)	27.00 (25.00, 29.00)	26.00 (23.50, 28.50)	4.768	0.092
Married	42 (71.19)	45 (80.36)	44 (83.02)	2.553	0.279
Family medical history	12 (20.34)	8 (14.29)	10 (18.87)	0.772	0.680

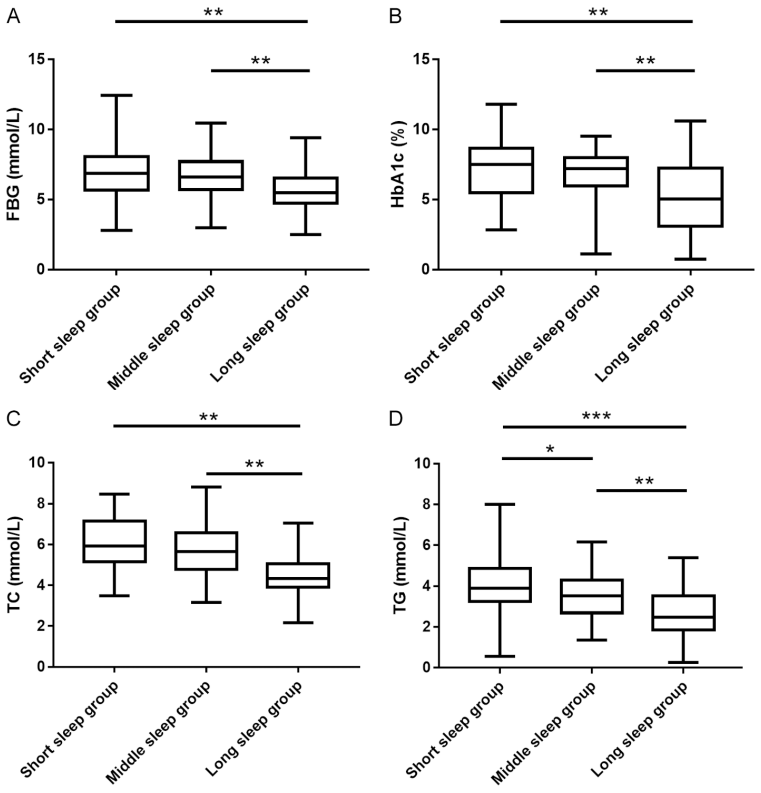

Figure 1. Analysis of glucolipid metabolism indexes in the three groups of patients. A. Comparison of fasting blood glucose (FBG) among the three groups of patients. B. Comparison of glycosylated hemoglobin (HbA1c) among the three groups of patients. C. Comparison of total cholesterol (TC) among the three groups of patients. D. Comparison of triglyceride (TG) among the three groups of patients. Note: *, **, and *** represent P<0.05, P<0.01, and P<0.001, respectively.

with indices related to glucolipid metabolism and body fat distribution was identified by Spearman correlation coefficients. A point-biserial correlation analysis was used to assess the association between sleep duration (an integer continuous variable) and glycemic control (a binary variable). Contributors to poor glycemic control were identified using univariate and multivariate Logistic regression analyses. All analyses relied upon a P<0.05 significance criterion.

Results

Basic characteristics of the three groups

The three groups showed no differences in basic characteristics such as sex (male), age, weight, BMI, marriage (married), and family history (P> 0.05). See **Table 1**.

Levels of glucolipid metabolism indices in the three groups

We detected and compared the levels of glucolipid metabolism indicators such as FBG, HbA1c, TC, and TG among the three groups of patients. The long sleep group was found to have lower FBG, HbA1c, TC, and TG than the other two groups (P<0.05). The TG in the middle sleep group was markedly reduced compared to the short sleep group (P<0.05). In the comparison of FBG, HbA1c, and TC between the short and middle sleep groups, the middle sleep group showed lower FBG, HbA1c, and TC than the short sleep group, but the differences were not significant (P>0.05). See **Figure 1**.

Correlation analysis between sleep duration and glucolipid metabolism

We named the short, middle, and long sleep groups as 1, 2, and 3, respectively, to analyze the correlation between sleep duration and glucolipid metabolism by Spearman correlation

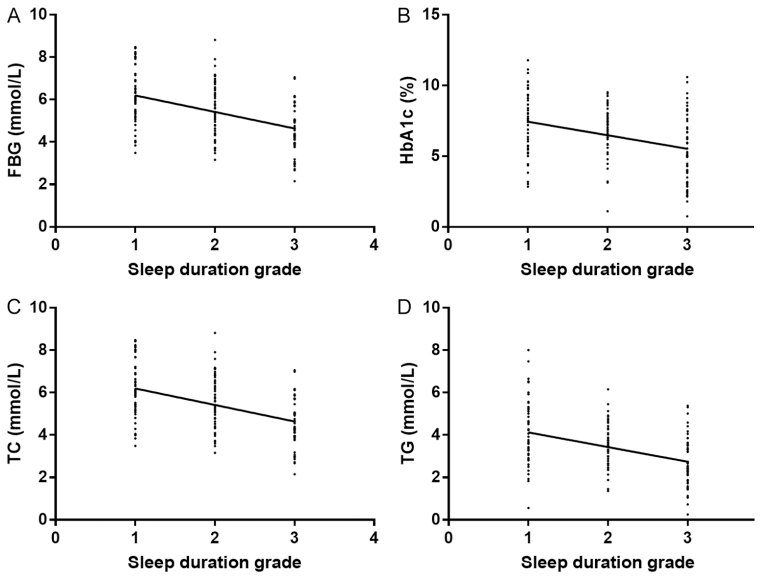

Figure 2. Spearman correlation coefficient analysis of the correlation between sleep duration and glucolipid metabolism. A. There was a significant negative connection between sleep duration and fasting blood glucose (FBG; r=-0.439, P<0.001). B. Sleep duration was negatively correlated with glycosylated hemoglobin (HbA1c; r=-0.322, P<0.001). C. There was a significant inverse connection between sleep duration and total cholesterol (TC; r=-0.421, P<0.001). D. Sleep duration was negatively correlated with triglyceride (TG; r=-0.418, P<0.001).

Table 2. Correlation analysis between sleep duration and glucolipid metabolism

Indicators		Sleep duration
FBG	r	-0.439
	P	<0.001
HbA1c	r	-0.322
	P	<0.001
TC	r	-0.421
	P	<0.001
TG	r	-0.418
	P	<0.001

Note: FBG, fasting blood glucose; HbA1c, glycosylated hemoglobin; TC, total cholesterol; TG, triglyceride.

coefficients. The data showed an inverse association between sleep duration and FBG, HbA1c, TC, and TG (P<0.05). See **Figure 2**; **Table 2**.

Comparison of sleep duration between good and poor glycemic control groups

We further classified patients with HbA1c <7% as the good glycemic control group (n=90) and those with HbA1c ≥7% as the poor glycemic control group (n=78) for comparative analysis, which revealed a significantly longer sleep duration in patients with well-controlled blood glucose (P< 0.05). See **Figure 3**.

Correlation analysis between sleep duration and glycemic control

Additionally, a point-biserial correlation analysis was carried out to further clarify the association of sleep duration with glycemic control, with poor and good glycemic control set as 1 and 2, respectively. A significant positive correlation was determined between sleep duration and glycemic control (r=0.242, P=0.002). See **Figure 4**.

Correlation analysis between sleep duration and body fat distribution

The long sleep group had significantly lower TFA than the short and middle sleep groups (P<0.01), while no statistical significance was found in VFA among the three groups (P>0.05). According to the correlation analysis, sleep duration was negatively correlated with TFA (r=-0.417, P<0.001), but not with VFA (r=-0.119, P=0.126). See **Figure 5** and **Table 3**.

Contributors to poor glycemic control in T2DM patients

According to univariate analysis, BMI, sleep duration, FBG, TG, and TFA were closely related to glycemic control (P<0.05). There was no association with gender, age, weight, marriage (married), family medical history, TC, or VFA (P>0.05). See **Table 4**.

Multivariate analysis further indicated the role of BMI (OR=4.021, 95% CI: 1.916-8.442, P< 0.001), sleep duration (short vs. moderate: OR=2.585, 95% CI: 1.103-6.059, P=0.029; long vs. moderate: OR=0.400, 95% CI: 0.162-0.986, P=0.046), FBG (OR=2.645, 95% CI: 1.282-5.456, P=0.008), TG (OR=2.121, 95% CI: 1.025-4.390, P=0.043), and TFA (OR=2.882, 95% CI: 1.390-5.977, P=0.004) as factors independently associated with poor glycemic control. See **Table 5**.

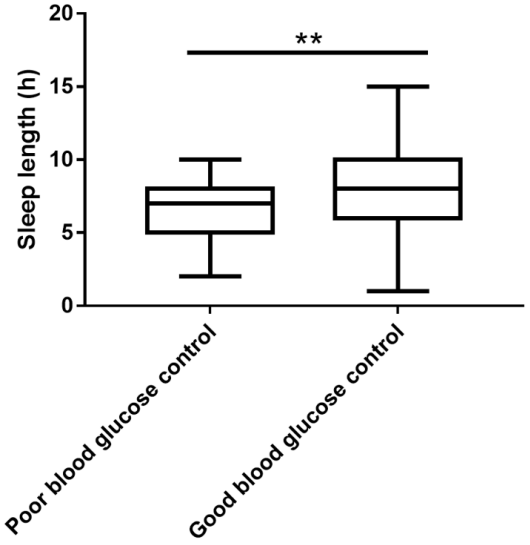

Figure 3. Comparison of sleep duration between good and blood glucose control groups. Note: **P<0.01.

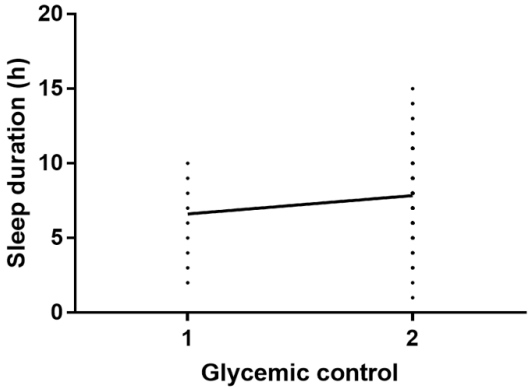

Figure 4. Correlation analysis between sleep duration and glycemic control.

Discussion

Sleep is an essential physiological state for humans, as well as for all other organisms, serving primarily to maintain energy homeostasis [16]. Sleep deprivation is a major manifestation of sleep disorders, which can stimulate the body's hunger and appetite, resulting in an increased risk of obesity [17]. Previous studies have also shown that a shorter sleep duration may contribute to some impairment of glucose tolerance and insulin sensitivity [18]. This study aimed to investigate the correlation of sleep duration with glucolipid metabolism in NOT2DM patients, hoping to provide evidence for the diagnosis and treatment of NOT2DM.

Other studies have pointed out the influence of sleep duration on DM. For example, a study by Zuraikat et al. [19] reported that shortening the sleep duration to an average of 6.2 hours per night for 6 weeks would lead to a significant increase in fasting insulin by a homeostasis model assessment of insulin resistance (HOMA-IR) in women, as well as impaired insulin sensitivity. Epidemiologic cohort evidence suggested that short sleep duration not only increases the risk of obesity, but is also a risk factor for developing T2DM [20]. Another mouse experiment showed that sleep deprivation markedly increases liver glucose production and liver TG levels in mice, while promoting the secretion of liver metabolites, further aggravating liver lipid oxidation and insulin resistance [21]. We first grouped the 168 NOT2DM patients based on their sleep duration: those who slept for less than 6 hours a day were designated as short sleep group, those who slept for 6-8 hours a day as the middle sleep group, and those who slept for at least 8 hours a day as the long sleep group. The levels of glucolipid metabolism indicators such as FBG, HbA1c, TC, and TG were tested in all the three groups of patients. It was found that with increasing sleep time, the long sleep group had significantly lower levels of FBG, HbA1c, TC, and TG; in addition, the TG levels in the middle sleep group were lower compared to the short sleep group. The above results suggest a connection between long sleep duration (>8 h) and lower FBG, HbA1c, TC, and TG levels, and between middle sleep duration (6-8 h) and lower TG concentrations. Subsequently, the Spearman correlation analysis in this study showed a statistical inverse association of sleep duration with FBG ($r=-0.439$, $P<0.001$), HbA1c ($r=-0.322$, $P<0.001$), TC ($r=-0.421$, $P<0.001$), and TG ($r=-0.418$, $P<0.001$), suggesting that increasing sleep duration may be beneficial for controlling glucolipid metabolism in NOT2DM patients. Next, we further analyzed the relationship between sleep duration and glycemic control. The good glycemic control group had a longer sleep duration than the poor glycemic control group. Moreover, a significant positive correlation was found between sleep duration and glycemic control ($r=0.242$, $P=0.002$). In the study of Barikani et al. [22], T2DM patients with long or medium sleep duration showed lower FBG, TC, and BMI, and those

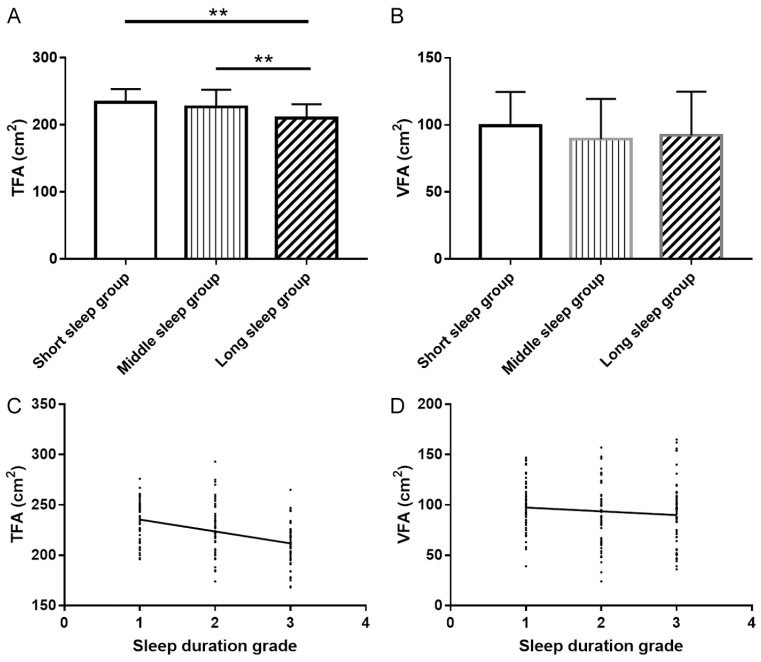

Figure 5. Correlation analysis between sleep duration and body fat distribution. A. Comparison of total fat area (TFA) among the three groups. B. Comparison of visceral fat area (VFA) among the three groups. C. Sleep duration was significantly negatively correlated with TFA (r=-0.417, P<0.001). D. Sleep duration was not significantly associated with VFA (r=-0.119, P=0.126). Note: **P<0.01.

Table 3. Data on the correlation between sleep duration and body fat distribution

Indicator		Sleep duration
TFA	r	-0.417
	P	<0.001
VFA	r	-0.119
	P	0.126

Note: TFA, total fat area; VFA, visceral fat area.

with good glycemic control had a statistically longer sleep duration, similar to our research results. We also found that TFA was significantly lower in the long sleep group than in the short and middle sleep groups, and that sleep duration had a significant negative correlation with TFA, but not with VFA, suggesting that longer sleep duration may improve body fat distribution to some extent, which was reflected mainly by a reduction in TFA levels. The mechanisms underlying the negative effects of sleep deprivation on T2DM may be as follows: First, sleep deprivation is a major trigger for insulin resistance in adipose tissue, which can also downregulate leptin secretion and promote lipolysis, resulting in varying degrees of negative effects on overall energy intake and insulin metabolism [23, 24]. Second, sleep insufficiency affects sympathetic nervous system output, which significantly increases the body's nocturnal secretion of adrenaline and norepinephrine and increase the sympathetic drive, thus potentially leading to a decrease in insulin sensitivity [25]. Third, the increase in cortisol levels caused by lack of sleep can lead to the body's stimulation of glucocorticoid signaling, which in turn may be associated with insulin resistance in adipose tissue [26]. Finally, factors influencing glycemic control in T2DM patients were evaluated. High BMI (≥27 kg/m^2), FBG (≥6.45 mmol/L), TG (≥3.34 mmol/L), and TFA (≥224 cm^2), as well as short sleep duration (<6 h), all increased the possibility of poor glycemic control, while a long sleep duration (>8 h) attenuated the risk.

Several limitations should be acknowledged. First of all, this study was a cross-sectional study, which can only reveal correlations but cannot establish causality. Subsequent longitudinal analysis should be supplemented to further determine the causal relationship. Second, this study was limited by its cross-sectional design and did not account for strong confounders such as exercise habits, diet, smoking/drinking, mental stress, and circadian rhythm. Subsequently, prospective data collection and further verification should be conducted to adjust for the influence of confounders as much as possible. Finally, due to the limited total cases included, the sample sizes of subgroups stratified by gender or age were too small, resulting in insufficient statistical power. Therefore, the universality of study conclusions across gender and age groups needs to be further validated in a prospective study with a larger sample size.

Our research indicated that NOT2DM patients who sleep for at least 8 hours had relatively lower levels of FBG, HbA1c, TC, TG, and TFA, with their sleep duration being negatively related to glucolipid metabolism and TFA and posi-

Table 4. Univariate analysis of poor glycemic control in NOT2DM patients

Characteristic	Poor glycemic control group (n=78)	Good glycemic control group (n=90)	χ^2	P
Sex			0.011	0.918
Male (n=61)	28 (35.90)	33 (36.67)		
Female (n=107)	50 (64.10)	57 (63.33)		
Age (years)			0.441	0.507
<51 (n=80)	35 (44.87)	45 (50.00)		
≥51 (n=88)	43 (55.13)	45 (50.00)		
Weight (kg)			0.729	0.393
<70 (n=77)	33 (42.31)	44 (48.89)		
≥70 (n=91)	45 (57.69)	46 (51.11)		
BMI (kg/m^2)			12.525	<0.001
<27 (n=74)	23 (29.49)	51 (56.67)		
≥27 (n=94)	55 (70.51)	39 (43.33)		
Married			1.247	0.264
No (n=81)	34 (43.59)	47 (52.22)		
Yes (n=87)	44 (56.41)	43 (47.78)		
Family medical history			1.681	0.195
No (n=148)	66 (84.62)	82 (91.11)		
Yes (n=20)	12 (15.38)	8 (8.89)		
Sleep duration			14.740	<0.001
Short (n=59)	38 (48.72)	21 (23.33)		
Moderate (n=56)	25 (32.05)	31 (34.44)		
Long (n=53)	15 (19.23)	38 (42.22)		
FBG (mmol/L)			9.573	0.002
<6.45 (n=84)	29 (37.18)	55 (61.11)		
≥6.45 (n=84)	49 (62.82)	35 (38.89)		
TC (mmol/L)			0.096	0.757
<5.39 (n=84)	38 (48.72)	46 (51.11)		
≥5.39 (n=84)	40 (51.28)	44 (48.89)		
TG (mmol/L)			4.691	0.030
<3.34 (n=84)	32 (41.03)	52 (57.78)		
≥3.34 (n=84)	46 (58.97)	38 (42.22)		
TFA (cm^2)			7.882	0.005
<224 (n=82)	29 (37.18)	53 (58.89)		
≥224 (n=86)	49 (62.82)	37 (41.11)		
VFA (cm^2)			0.616	0.433
<95 (n=83)	36 (46.15)	47 (52.22)		
≥95 (n=85)	42 (53.85)	43 (47.78)		

Note: NOT2DM, new-onset type 2 diabetes mellitus; BMI, body mass index; FBG, fasting blood glucose; HbA1c, glycosylated hemoglobin; TC, total cholesterol; TG, triglyceride; TFA, total fat area; VFA, visceral fat area.

tively correlated with glycemic control. Additionally, high BMI (≥27 kg/m^2), short sleep duration (<6 h), as well as high FBG (≥6.45 mmol/L), TG (≥3.34 mmol/L), and TFA (≥224 cm^2), independently increased the risk of poor glycemic control, while long sleep duration (>8 h) was an independent protective factor. Based on the above, long sleep duration is related to lower levels of glucolipid metabolism indices and TFA, as well as better glycemic control, in NOT2DM patients. Weight management should also be emphasized. However, given that this

Table 5. Multivariate analysis of poor glycemic control in NOT2DM patients

Characteristic	B	SE	WALD	P	OR	95% CI
BMI (kg/m^2)	1.392	0.378	13.526	<0.001	4.021	1.916-8.442
Sleep duration						
Short vs. moderate	0.950	0.435	4.779	0.029	2.585	1.103-6.059
Long vs. moderate	-0.916	0.460	3.965	0.046	0.400	0.162-0.986
FBG (mmol/L)	0.973	0.369	6.935	0.008	2.645	1.282-5.456
TG (mmol/L)	0.752	0.371	4.102	0.043	2.121	1.025-4.390
TFA (cm^2)	1.059	0.372	8.092	0.004	2.882	1.390-5.977

Note: NOT2DM, new-onset type 2 diabetes mellitus; BMI, body mass index; FBG, fasting blood glucose; TG, triglyceride; TFA, total fat area.

study adopted a cross-sectional design, the causal association needs to be further verified by prospective studies.

Disclosure of conflict of interest

None.

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