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Changes in the TyG-BMI Index and Its Correlation with Cognitive Impairment in Patients with Chronic Insomnia Disorder

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Abstract: *This study investigated changes in the triglyceride glucose - body mass index (TyG-BMI) in patients with chronic insomnia disorder (CID) and its relationship with cognitive function, while evaluating its predictive value and pathway. A total of 91 CID patients and 60 healthy controls (HCs) were enrolled, with assessments including sleep quality (PSQI, STOP-Bang), cognitive function (MoCA), fasting plasma glucose (FPG), and triglycerides (TG). Compared with HCs, the CID group exhibited significantly higher BMI, PSQI scores, TG, FPG, and TyG-BMI, alongside lower MoCA scores (all $P < 0.05$). Partial correlation analysis revealed that TyG-BMI was positively correlated with PSQI in the CID group ($r = 0.360$, $P < 0.005$), while in the total sample, TyG-BMI was negatively correlated with MoCA ($r = -0.430$, $P < 0.005$). Receiver operating characteristic curve analysis demonstrated that the area under the curve of TyG-BMI for predicting CID (0.870) and cognitive dysfunction (0.753) were superior to the TyG index (0.801 and 0.685, respectively). Mediation analysis did not support a mediating role of TyG-BMI between insomnia and cognition (95% CI included 0), whereas moderation analysis indicated that the product term of PSQI and TyG-BMI significantly influenced MoCA scores ($B = 0.003$, $P < 0.05$), with the interaction plot showing the flattest slope in the high TyG-BMI group and the steepest in the low TyG-BMI group. Collectively, these findings suggest that TyG-BMI is significantly associated with both chronic insomnia and cognitive function, demonstrating favorable predictive utility, and its moderating role between insomnia and cognition highlights its potential as a novel biomarker for personalized management of insomnia-related cognitive impairment.*

Keywords: *Chronic insomnia disorder; Triglyceride glucose - body mass index; Insulin resistance; Cognition*

1. Introduction

Chronic insomnia disorder (CID) has long posed a significant public health threat, with its prevalence rising steadily year after year. Chronic insomnia refers to difficulty falling asleep, maintaining sleep and/or early-morning awakening for at least three months, accompanied by obvious daytime symptoms such as fatigue, distraction and mood disorders[1]. Among them, patients with CID often complain of impaired daily cognitive function when they come for medical treatment, such as memory decline, distraction, and executive function disorders. Chronic insomnia may also induce a spectrum of systemic pathophysiological alterations, involving the nervous, endocrine, and immune systems. Recently, metabolomics research on chronic insomnia has increasingly attracted scholarly attention. Accumulating evidence indicates that sleep and circadian rhythms exert considerable influence on hormones involved in appetite regulation and energy metabolism. Sleep insufficiency contributes to disruptions in energy balance and glucose homeostasis, thereby increasing the risk of obesity, insulin resistance (IR), and type 2 diabetes. Long-standing IR may further impair cognitive function through mechanisms including enhanced extracellular β-amyloid deposition, intracellular hyperphosphorylation of tau protein, and mitochondrial dysfunction[2].

The triglycerideglucose-body mass index (TyG-BMI) has emerged as a comprehensive and important indicator for metabolic assessment in recent years and is now recognized as a robust

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predictor of IR. Growing evidence has underscored its close association with multisystem diseases spanning cardiovascular, metabolic, and oncological pathways. However, current findings regarding the relationship between TyG-BMI and cognitive performance remain contentious, presenting a "two-sided" picture. One study observed that higher TyG-BMI levels in patients with Alzheimer's disease were correlated with lower tau and phosphorylated tau (pTau) levels and higher Aβ42 levels, suggesting a potentially protective metabolic role against AD pathology[3]. Conversely, a 2025 study involving 876 participants from a health screening population revealed that a higher TyG-BMI index was associated with poorer cognitive performance[4]. These discrepant results indicate that conclusions in this field are not yet fully consistent. This raises the question of whether such discrepancies may be attributed to differences in the study populations. In the context of chronic insomnia, therefore, how does the TyG-BMI index vary, and does it mediate the relationship between insomnia and cognitive impairment? The present study aims to investigate the alterations in TyG-BMI index among patients with CID and its association with cognitive dysfunction.

2. Materials and Methods

2.1. Subjects

Patients with CID were consecutively recruited from the Department of General Medicine and the Sleep Clinic at the First Affiliated Hospital of Bengbu Medical University between January 1, 2026, and December 31, 2026.

Inclusion criteria were as follows: (1) meeting the diagnostic criteria for chronic insomnia according to the third edition of the International Classification of Sleep Disorders (ICSD-3)[5]; (2) aged 18 to 60 years; (3) having received at least 6 years of formal education with normal comprehension ability; (4) a total score >7 on the Pittsburgh Sleep Quality Index (PSQI)[6]; and (5) a total score <7 on the 17-item Hamilton Depression Rating Scale (HAMD-17)[7].

Exclusion criteria included: (1) use of any antidepressant or sedative-hypnotic medications within the preceding 2 weeks; (2) other sleep-related disorders, such as obstructive sleep apnea, restless legs syndrome, or narcolepsy; (3) other conditions known to cause cognitive impairment, including psychiatric disorders, internal medical diseases (e.g., hepatic, renal, cardiovascular, or thyroid disorders), surgical conditions (e.g., craniocerebral or spinal cord injuries), and neurological disorders (e.g., dementia or Parkinson's disease); (4) pregnancy or lactation; (5) visual, auditory, comprehension, or motor impairments precluding completion of the study assessments; and (6) unwillingness to participate.

A total of ninety-one patients with CID were ultimately enrolled in the study. Sixty healthy individuals who underwent physical examinations during the same period in the same hospital (without insomnia complaints and related medical histories, and both PSQI scores and HAMD-17 scores were <7 points) were included as healthy controls(HCs). The control group was matched to the chronic insomnia group with respect to general demographic characteristics (Table 1).

This study was approved by the Ethics Committee of Bengbu Medical University (approval No. [2026]444) and was performed in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

2.2. Baseline data collection

Collection of general data: General information was collected from all participants, including demographic characteristics (i.e., age, sex, height, weight, and years of education), medical history, and family history.

2.3. Assessment of sleep quality and mood states

Assessment of sleep quality and emotional status: (1) The PSQI was administered to evaluate subjective sleep quality over the preceding month in all participants. (2) The STOP-Bang questionnaire was used to assess the risk of sleep apnea. (3) The HAMD-17 was employed to evaluate depressive symptoms and to exclude participants with clinically significant depression.

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2.4. Evaluations of cognitive function

Global cognitive function was assessed using the Chinese-Beijing Version of Montreal Cognitive Assessment(MoCA)[8], which evaluates the following domains: visuospatial and executive function, naming, attention, language, abstraction, delayed recall, and orientation. The total score ranges from 0 to 30, with higher scores indicating better overall cognitive performance.

2.5. Laboratory parameters

Fasting venous blood samples were obtained from all participants to measure fasting plasma glucose (FPG) and triglycerides (TG).

2.6. Calculation of derived indices

Body mass index (BMI) was calculated as body weight divided by height squared (kg/m²). The triglyceride-glucose-body mass index (TyG-BMI) was computed using the following formula: TyG-BMI = ln[TG × FPG / 2] × BMI[9] (with FPG and TG expressed in mg/dL; conversion factors: 1 mg/dL FPG = 18.02 mmol/L FPG; 1 mg/dL TG = 88.6 mmol/L TG).

2.7. Statistical analysis

Statistical analyses were performed using SPSS software (version 27.0; IBM Corp., Armonk, NY, USA). Continuous variables with a normal distribution were expressed as mean ± standard deviation (SD), and between-group comparisons were conducted using the independent-samples *T*-test. Continuous variables with a non-normal distribution were presented as median (interquartile range) [$M(P_{25}, P_{75})$] and compared between groups using the Mann–Whitney *U* test. Categorical variables were reported as counts (n) and analyzed using the chi-square (χ^2) test. Spearman's rank correlation analysis was employed to assess the correlations between TyG-BMI and various clinical parameters within the CID group and across all participants. Partial correlation analyses were performed after adjusting for potential confounders, including age, sex, years of education, and depression severity (HAMD-17 scores). Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the predictive value of TyG-BMI for insomnia and cognitive impairment, with sensitivity and specificity calculated accordingly. Mediation analysis was performed using the PROCESS macro (version 4.0; developed by Andrew F. Hayes, 2022). Specifically, Model 4 was applied to test the mediating role of TyG-BMI in the relationship between insomnia and cognitive function, with bias-corrected percentile bootstrap methods based on 5,000 resamples to estimate 95% confidence intervals (CIs). Model 1 was used to examine the moderating effect of TyG-BMI on the insomnia–cognition relationship, also employing bootstrap resampling (5,000 iterations) for 95% CI estimation. Two-sided *P* values <0.05 were considered statistically significant.

3. Results

3.1. Baseline characteristics and clinical parameters

As shown in Table 1, there were no statistically significant differences between the CID and the HCs in terms of sex, age, years of education, HAMD-17 scores, or STOP-Bang questionnaire scores ($P>0.05$,Table 1). Compared with the HCs, the CID group exhibited higher BMI, PSQI scores, TG, FPG, and TyG-BMI, along with significantly lower MoCA scores, with all differences reaching statistical significance ($P< 0.05$; Table 1).

Table 1: Comparison of general characteristics and clinical parameters between the CID and the HCs

Terms	CID	HCs	Statistics	*P*
Number of cases	91	60	/	/
Male/Female	49/42	24/36	χ^2=2.776	0.096
Age(years)	53.00(50.00, 56.00)	52.00(48.25, 55.75)	*Z*=-1.276	0.202
Weight(Kg)	65.75±10.12	62.13±9.29	*t*=2.217	<0.05
Height(cm)	1.65(1.60, 1.70)	1.68±0.08	*Z*=-2.589	<0.05
BMI(kg/m^2)	24.22(22.77, 25.61)	21.74±1.24	*Z*=-6.476	<0.005
Education(years)	9.00(6.00, 12.00)	9.00(6.00, 12.00)	*Z*=-0.623	0.533

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HAMD-17(score)	3.00(1.00, 5.00)	2.00(1.00, 5.00)	*Z*=-1.344	0.179
PSQI(score)	15.00(13.00, 17.00)	0(0, 1.00)	*Z*=-10.492	<0.005
MoCA(score)	23.00(21.00, 25.00)	27.00(26.00, 27.75)	*Z*=-7.360	<0.005
STOP-Bang(score)	1.00(1.00, 2.00)	1.00(1.00, 2.00)	*Z*=-0.215	0.830
TG(mmol/L)	0.89(0.72, 1.21)	0.68±0.14	*Z*=-5.864	<0.005
FPG(mmol/L)	4.82(4.64, 5.34)	4.71(4.52, 4.83)	*Z*=-3.058	<0.005
Tyg-BMI	194.79(186.31, 213.82)	169.71±10.25	*Z*=-7.674	<0.005

CID:chronic insomnia disorder; HCs, healthy controls; BMI, body mass index; HAMD-17, Hamilton Depression Rating Scale-17; PSQI, Pittsburgh Sleep Quality Index; MoCA, Montreal Cognitive Assessment; STOP-Bang, STOP-Bang questionnaire; TG, triglyceride; FPG, fasting plasma glucose; TyG-BMI, triglycerideglucose-body mass index.

3.2. Correlation between TyG-BMI and clinical parameters

Spearman's rank correlation analysis revealed that TyG-BMI was positively correlated with PSQI scores in the CID group. In the total sample, TyG-BMI was positively correlated with PSQI scores and negatively correlated with MoCA scores ($P< 0.05$; Table 2).

Given that age, sex, years of education, and depressive status may influence both sleep and cognitive function, partial correlation analyses were performed after controlling for these factors. The results remained consistent: TyG-BMI was positively correlated with PSQI scores in the CID group, and in the total sample, TyG-BMI was positively correlated with PSQI scores and negatively correlated with the MoCA scores($P< 0.05$; Table 2).

Table 2: Correlation analysis between TyG-BMI and clinical parameters

	CID (*n*=91)		Total sample(*n*=151)	
	Spearmananalysis	Partialcorrelation analysis	Spearmananalysis	Partialcorrelation analysis
PSQI (scors)	0.283*	0.359**	0.602**	0.588**
MoCA (scors)	-0.145	-0.170	-0.483**	-0.423**

Note:***P*<0.005;**P*<0.05; PSQI, Pittsburgh Sleep Quality Index; MoCA,Montreal Cognitive Assessment.

3.3. The diagnostic value of TyG-BMI and TyG for the risk of chronic insomnia and cognitive impairment

ROC curve analysis demonstrated that the area under the curve (AUC) of TyG-BMI for diagnosing chronic insomnia was 0.870 ($P< 0.001$), with an optimal cutoff value of 187.45. The AUC of TyG-BMI for predicting cognitive impairment was 0.753($P< 0.001$), with an optimal cutoff value of 183.81. Notably, the overall predictive performance of TyG alone was inferior to that of TyG-BMI (Table 3, Figure 1-2).

Table 3: Diagnostic value of TyG-BMI and TyG for insomnia and cognitive impairment

	Chronic insomnia		cognitive impairment	
	TyG-BMI	TyG	TyG-BMI	TyG
AUC	0.870	0.801	0.753	0.685
Cutoffvalue	187.45	7.925	183.81	7.925
Sensitivity	0.736	0.758	0.674	0.698
specificity	0.983	0.767	0.723	0.646
P	0.000	0.000	0.000	0.000

TyG-BMI, triglycerideglucose-body mass index; TyG, triglycerideglucose; AUC, area under the curve.

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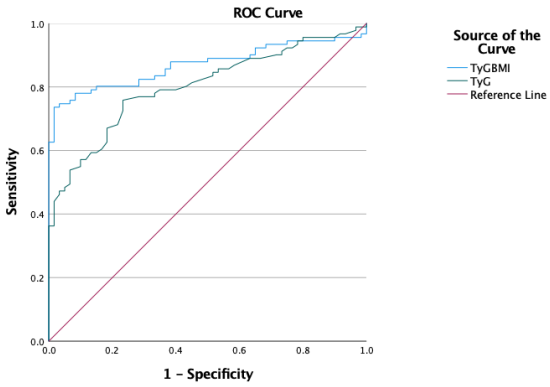

Figure 1: ROC curves of TyG-BMI and TyG for predicting chronic insomnia

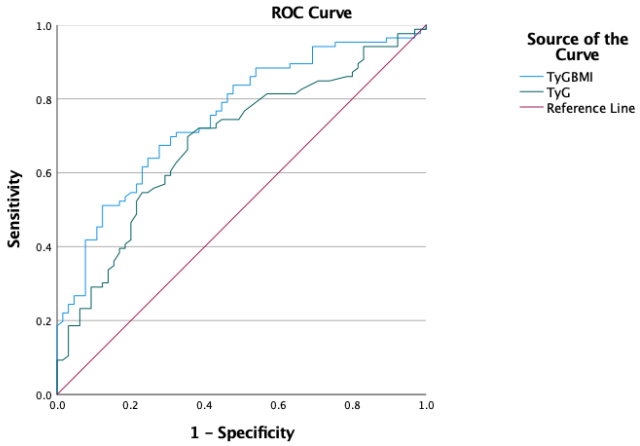

Figure 2: ROC curves of TyG-BMI and TyG for predicting cognitive impairment

3.4. Mediation analysis of TyG-BMI in the relationship between insomnia and cognitive function

Mediation analysis revealed that the direct effect of insomnia on cognitive function was -0.204 [95% CI: (-0.267, -0.141)]. The indirect effect of TyG-BMI on the relationship between insomnia and cognitive function was -0.028 [95% CI: (-0.063, 0.010)], with the 95% confidence interval containing zero, indicating that the mediating effect of TyG-BMI between chronic insomnia and cognitive function was not statistically significant (Table 4).

Table 4: Mediation analysis of TyG-BMI in the relationship between insomnia and cognitive function

Effect type	Pathway	Effect	Boot SE	95%CI	*P*
Total effect	insomnia→Cognitive function	-0.231	0.026	(-0.283, -0.180)	0.000
Direct effect	insomnia→Cognitive function	-0.204	0.032	(-0.267, -0.141)	0.000
Indirect effect	insomnia→TyG-BMI →Cognitive function	-0.028	0.019	(-0.063, 0.010)	-

TyG-BMI, triglyceride glucose-body mass index; Boot SE, bootstrap standard error (bias-corrected); 95% CI, bias-corrected bootstrap confidence interval based on 5,000 resamples.

3.5. Moderating effect of TyG-BMI on the relationship between insomnia and cognitive function

To examine the moderating role of TyG-BMI in the relationship between insomnia and cognitive function, the moderation analysis was conducted with PSQI score as the independent variable, MoCA score as the dependent variable, and TyG-BMI as the moderator variable. The main effects of PSQI score, TyG-BMI, and their product term (PSQI × TyG-BMI) on MoCA score were all statistically significant ($P < 0.05$; Table 5), indicating that TyG-BMI exerted a moderating effect on the relationship between insomnia and cognitive function.

The interaction plot (Figure 3) visually depicts the pattern of this moderating effect: the regression

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lines for the high-, medium-, and low-TyG-BMI groups all declined with increasing PSQI scores; however, the slope of the low-TyG-BMI group was steeper than that of the high-TyG-BMI group. With the aggravation of insomnia severity, cognitive function in the low-TyG-BMI group was poorer than that in the high-TyG-BMI group. These findings suggest that a higher TyG-BMI level may buffer the negative impact of insomnia on cognitive function, whereas a lower TyG-BMI level may exacerbate this impairment.

Table 5: Moderation analysis of TyG-BMI on the relationship between insomnia and cognitive function

Variable	B	SE	t	*P*	95%CI
Constant	24.218	0.240	100.739	0.000	(23.743, 24.693)
PSQI	-0.167	0.036	-4.661	0.000	(-0.238, -0.096)
TyG-BMI	-0.030	0.012	-2.578	0.011	(-0.052, -0.007)
PSQI×TyG-BMI	0.003	0.001	2.160	0.032	(0, 0.005)

PSQI, Pittsburgh Sleep Quality Index; TyG-BMI, triglycerideglucose-body mass index; B, unstandardized coefficient; SE, standard error; 95% CI, 95% confidence interval.

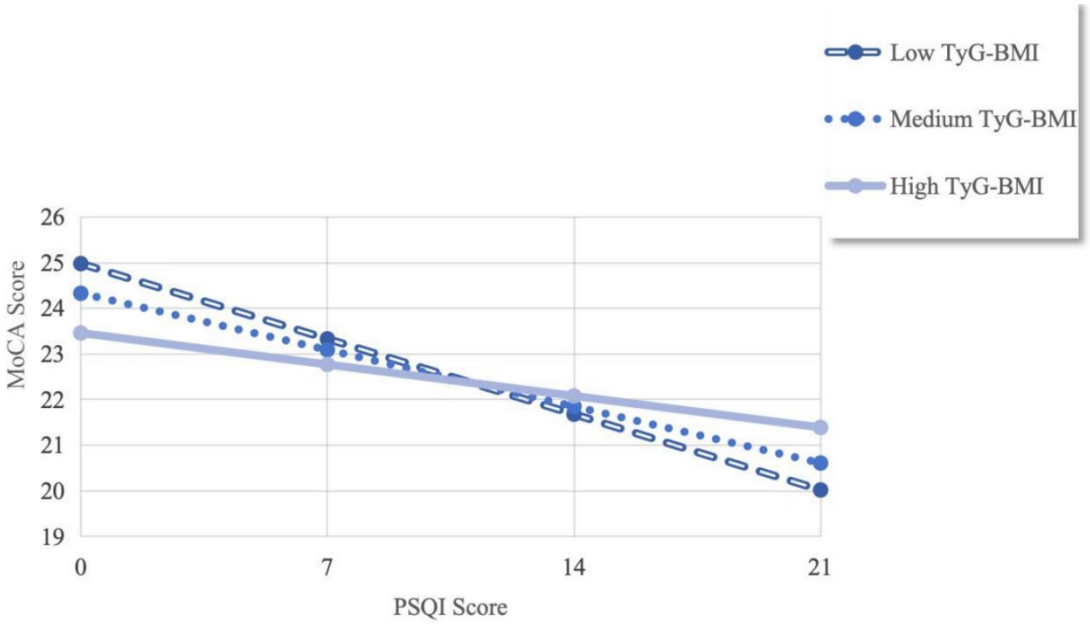

Figure 3: The moderating effect of TyG-BMI on the insomnia–cognitive function relationship.

4. Discussion

Patients with chronic insomnia frequently present with a variety of metabolic disturbances, including abnormalities in glucose metabolism, lipid metabolism, and cortisol secretion. However, previous studies have predominantly focused on single metabolic parameters. The TyG-BMI index, as a novel comprehensive indicator of IR, has been demonstrated to possess strong predictive capacity for identifying IR[10]. Moreover, by incorporating body mass index, this index places greater emphasis on the synergistic effect of systemic adipose accumulation and IR. In the present study, the TyG-BMI level in the CID group was significantly higher than that in the control group (194.79 vs. 169.71, $P< 0.001$). This finding suggests that chronic insomnia may promote the development and progression of IR through mechanisms involving activation of the hypothalamic-pituitary-adrenal (HPA) axis, sympathetic nervous system excitation, and inflammatory responses. Of note, a recent study demonstrated that insomnia significantly alters the lipidomic profile in older adults, including elevations in multiple acylcarnitine species, further supporting the close association between insomnia and metabolic dysregulation.

Prolonged insomnia is known to promote the development of IR, and accumulating evidence has established a close association between IR and increased blood brain barrier (BBB) permeability, which may in turn accelerate neurodegenerative processes and thereby contribute to cognitive dysfunction[11,12]. However, investigations examining the relationship between TyG-BMI—a comprehensive metabolic indicator—and both chronic insomnia and cognitive function in this specific population remain relatively scarce. To further elucidate the interplay among these three factors, we

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conducted correlation analyses in both the chronic insomnia group and the total sample. The results demonstrated that TyG-BMI was positively correlated with PSQI scores in the CID group, suggesting that the degree of metabolic disturbance is closely associated with insomnia severity. In the total sample, TyG-BMI was negatively correlated with MoCA scores, further supporting the notion that IR may impair cognitive function through pathways involving neuroinflammation, cerebral energy metabolism disturbances, and vascular endothelial dysfunction. Subsequently, ROC curve analyses were performed to evaluate the predictive value of TyG-BMI for chronic insomnia and cognitive impairment. The results showed that TyG-BMI yielded a superior AUC for predicting chronic insomnia compared with TyG alone (0.870 vs. 0.801). Similarly, for predicting cognitive impairment, TyG-BMI also outperformed TyG (0.753 vs. 0.685). These findings are consistent with previous reports demonstrating that TyG-BMI possesses better predictive performance for cognitive impairment than TyG alone[10]. Collectively, these results suggest that TyG-BMI may serve as a simple and feasible metabolic screening indicator for the early clinical identification of individuals at high risk for insomnia-related cognitive decline.

The above correlation analyses indicated that TyG-BMI was significantly associated with both insomnia severity and cognitive dysfunction, yet whether TyG-BMI influences the direction or magnitude of the relationship between insomnia and cognition remained unclear. To address this, we further constructed a regression model with PSQI as the independent variable, MoCA as the dependent variable, and TyG-BMI as the moderator to examine their interaction effect. The results showed that the product term of PSQI and TyG-BMI had a significant effect on MoCA scores (B=0.003, P<0.05), indicating that TyG-BMI exerted a significant moderating role in the relationship between insomnia severity and cognitive dysfunction. As depicted in the interaction plot (Figure 3), the regression lines for the high-, medium-, and low-TyG-BMI groups all declined with increasing PSQI scores; however, the slope for the high-TyG-BMI group was the flattest, whereas that for the low-TyG-BMI group was the steepest. These findings suggest that a higher TyG-BMI level may buffer the negative impact of insomnia on cognitive impairment, whereas a lower TyG-BMI level may exacerbate this impairment. This finding appears inconsistent with our initial hypothesis; nevertheless, several recent studies offer plausible explanations. First, a U-shaped curvilinear relationship may exist between TyG-related indicators and cognitive function, whereby both excessively high and low levels are associated with an increased risk of cognitive impairment[13]. In our study, the CID group already exhibited a state of metabolic abnormality, and the distribution of TyG-BMI levels in this group may have fallen precisely within the "protective" segment of the U-shaped curve, thereby manifesting a buffering effect on cognitive function. Second, BMI may play a critical modifying role in the association between TyG and cognition—among underweight individuals, a high TyG index is significantly correlated with cognitive decline, whereas this association dissipates in overweight/obese populations[14]. Furthermore, the protective effect of TyG-BMI on cognitive function appears more pronounced in non-diabetic and low-BMI subgroups[15]. Collectively, these lines of evidence suggest that the buffering effect of elevated TyG-BMI observed in our study may reflect a protective role of greater metabolic reserve against insomnia-related cognitive impairment, rather than a purely detrimental effect of IR.

The mediation analysis did not reveal a statistically significant mediating effect of TyG-BMI on the relationship between insomnia and cognitive dysfunction, as the 95% confidence interval for the indirect effect contained zero. This suggests that TyG-BMI does not serve as a major mediating pathway through which insomnia leads to cognitive decline. Taken together with the moderating effect, these findings indicate that TyG-BMI is more likely to function as a contextual factor that moderates the impact of insomnia on cognition, rather than as an intermediary variable that transmits the deleterious effects of insomnia. From the perspective of clinical intervention, these findings imply that reducing TyG-BMI levels may not be effective in halting the progression from insomnia to cognitive impairment. Conversely, for insomnia patients with low TyG-BMI levels, appropriately enhancing metabolic reserve may confer greater protective benefits.

5. Conclusion

In summary, the present study demonstrates that patients with chronic insomnia present with a propensity toward IR, as reflected by significantly elevated TyG-BMI levels. This composite metabolic index is not only closely associated with the severity of insomnia and the level of cognitive function but also exhibits robust predictive utility for both chronic insomnia and cognitive impairment. More importantly, TyG-BMI was found to significantly moderate the deleterious effect of insomnia on cognitive performance. These findings underscore the importance of routine metabolic evaluation in patients with chronic insomnia in clinical settings. Early detection and timely intervention targeting IR

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may represent a promising strategy to delay or attenuate the progression of insomnia-related cognitive decline.

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