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Original Article

Elevated TXNIP and reduced PINK1 in gestational diabetes mellitus: association with dyslipidemia and pregnancy complications

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Abstract: Objectives: To investigate the association between thioredoxin-interacting protein (TXNIP), PTEN-induced putative kinase 1 (PINK1) and lipid metabolism in gestational diabetes mellitus (GDM), and to assess their clinical significance. Methods: This case-control study included 220 pregnant women (110 with GDM and 110 healthy controls) recruited from 2022 to 2024. Clinical assessments included glucose, lipids, and thyroid function profiles. TXNIP and PINK1 mRNA expression were measured using RT-qPCR. Pregnancy outcomes were documented using standardized clinical protocols. Results: Compared to controls, GDM patients had significantly higher fasting blood glucose (FBG), 2-hour postprandial blood glucose (2hPBG), glycated hemoglobin (HbA1c), and homeostasis model assessment of insulin resistance (HOMA-IR), along with reduced fasting insulin (FINS). Thyroid function tests showed elevated triiodothyronine (T3), and thyroxine (T4) levels in the GDM group. Lipid profiles revealed increased triglycerides (TG), total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C), and decreased high-density lipoprotein cholesterol (HDL-C). TXNIP expression was significantly elevated, while PINK1 expression was decreased in GDM patients. Correlation analysis indicated strong associations between TXNIP and PINK1 levels with lipid values. GDM was also associated with adverse pregnancy outcomes, including higher rates of cesarean delivery, preterm birth, macrosomia, neonatal hypoglycemia, and lower Apgar scores. Conclusions: Dyslipidemia in GDM-characterized by elevated TC, TG, and LDL-C and decreased HDL-C-may be associated with upregulation of TXNIP and downregulation of PINK1. These molecular alterations could contribute to metabolic disturbances and poor pregnancy outcomes. Monitoring lipid profiles alongside TXNIP and PINK1 expression may aid in the clinical management of GDM and its complications.

Keywords: TXNIP, PINK1, blood lipid metabolism, gestational diabetes mellitus, correlation

Introduction

Gestational diabetes mellitus (GDM) refers to glucose intolerance first recognized during pregnancy and poses substantial health risks to both the mother and fetus [1, 2]. It is associated with adverse pregnancy outcomes, including macrosomia, neonatal hypoglycemia, and an increased risk of long-term metabolic disorders in offspring [3-5]. GDM is typically characterized by insulin resistance and hyperglycemia, often accompanied by dyslipidemia-manifested as elevated triglycerides and low-density lipoprotein cholesterol (LDL-C), along with decreased high-density lipoprotein cholesterol (HDL-C) [6]. These lipid abnormalities can worsen metabolic dysfunction, heighten cardiovascular risk, and contribute to a proinflammatory state that worsens pregnancy outcomes [7].

Recent evidence highlights the critical roles of oxidative stress and mitochondrial dysfunction in the pathogenesis of GDM [8-10]. Thioredoxin-interacting protein (TXNIP) is a key regulator of intracellular oxidative stress and contributes to insulin resistance by promoting β-cell apoptosis and inhibiting insulin signaling [11]. Its overexpression disrupts glucose homeostasis and plays a pathogenic role in diabetes [12]. In contrast, PTEN-induced kinase 1 (PINK1) is essential for maintaining mitochondrial quality

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Table 1. Primer sequences for target and reference genes used in RT-qPCR

Gene name	Forward (5'-3')	Reverse (5'-3')
TXNIP	TGCCACCACCGACTTATACTGA	CCTGCTGACCACCTCCTACA
PINK1	CACACTGTTCCTCGTTATGAAGA	CTTGAGATCCCGATGGGCAAT
β-Actin	TCCCTGGAGAAGAGCTACGAGC	TGCCACAGGACTCCATGCCCAG

TXNIP: thioredoxin interacting protein; PINK1: PTEN-induced putative kinase 1.

through mitophagy [13]. By eliminating damaged mitochondria, PINK1 helps sustain cellular energy homeostasis and limit oxidative injury [14]. In GDM, reduced PINK1 expression may lead to mitochondrial dysfunction and heightened oxidative stress, thereby aggravating insulin resistance and lipid abnormalities [15].

Although TXNIP and PINK1 have been implicated in diabetic pathophysiology, their specific involvement in lipid metabolism during GDM remains poorly understood. Given that lipid homeostasis is vital for fetal development and maternal adaptation during pregnancy, disturbances in lipid regulation may have profound implications for gestational health. This study aims to clarify the roles of TXNIP and PINK1 in the context of lipid metabolism in GDM. Elucidating these molecular interactions may provide insight into GDM-related metabolic disturbances and identify biomarkers or therapeutic targets for disease management.

Materials and methods

Research strategy

From February 2022 to June 2024, a total of 110 patients diagnosed with GDM at the Affiliated Women and Children's Hospital of Ningbo University according to standardized diagnostic criteria [16] were enrolled as the GDM group. During the same period, 110 healthy pregnant women were selected as the healthy control (HC) group. All participants provided written informed consent. Clinical data were retrieved from electronic medical records.

Inclusion criteria: Patients aged ≥18 years; singleton pregnancy; normal liver and kidney function; complete cognitive and behavioral capacity.

Exclusion criteria: Patients with malignancies; history of miscarriage; prior chemotherapy, immunotherapy, or radiotherapy; or pregestational type 1 or type 2 diabetes mellitus.

This study was approved by the Institutional Review Board and Ethics Committee of the Affiliated Women and Children's Hospital of Ningbo University.

Laboratory indicators

Fasting venous blood samples (5 mL) were collected upon admission, anticoagulated with EDTA, and centrifuged at 2800 rpm for 15 minutes to obtain serum. An automated biochemical analyzer (AU5800, Beckman Coulter, USA) was used to measure fasting blood glucose (FBG), 2-hour postprandial blood glucose (2hPBG), glycated hemoglobin (HbA1c), fasting insulin (FINS), homeostasis model assessment for insulin resistance (HOMA-IR), free triiodothyronine (FT3), free thyroxine (FT4), thyroid-stimulating hormone (TSH), total triiodothyronine (T3), total thyroxine (T4), total cholesterol (TC), triglycerides (TG), LDL-C, and HDL-C.

Remaining blood samples were stored in a certified biobank at -80°C under standardized protocols. All patients had previously consented to the use of de-identified biological specimens for research purposes.

Gene expression analysis

Venous blood collected at admission was centrifuged at 3500 rpm to separate serum, from which total RNA was extracted using TRIzol reagent (Invitrogen, 15596026; Shanghai Yubo Biotechnology Co., Ltd.) according to the manufacturer's instructions. Complementary DNA (cDNA) was synthesized using the PrimeScript™ RT-PCR Kit (RR055A, Takara; Shanghai Shanran Biotechnology Co., Ltd.). Quantitative real-time PCR was conducted using the BIO-RAD CFX96 system (Shanghai Aolu Biotechnology Co., Ltd.) with TB Green Premix Ex Taq II (RR820A, Tli RNaseH Plus, Guangzhou Peiyu Biological Products Co., Ltd.).

β-Actin was used as an internal control. Each sample was tested in triplicate, and relative gene expression was calculated using the $2^{-\Delta\Delta CT}$ method. Primer sequences are listed in **Table 1**.

Table 2. Comparison of general information

Data	HC group (n = 110)	GDM group (n = 110)	t/χ^2	P
Age (years)	28.27 ± 2.75	28.49 ± 2.65	0.612	0.541
Pre-pregnancy BMI (kg/m^2)	24.14 ± 1.26	23.98 ± 1.33	0.913	0.362
Gestational age (weeks)	28.02 ± 2.01	28.14 ± 2.06	0.440	0.660
Hypertension [n (%)]	18 (16.36%)	22 (20.00%)	0.489	0.484
Family history of diabetes [n (%)]	15 (13.64%)	29 (26.36%)	5.568	0.018
Household registration [n (%)]			0.322	0.571
Rural	36 (32.73%)	40 (36.36%)		
Town	74 (67.27%)	70 (63.64%)		
Educational status [n (%)]			0.178	0.673
Bachelor degree or above	72 (65.45%)	69 (62.73%)		
High school and below	38 (34.55%)	41 (37.27%)		
Ethanol intake [n (%)]			0.000	1.000
Yes	2 (1.82%)	1 (0.91%)		
No	108 (98.18%)	109 (99.09%)		
Smoking status [n (%)]			0.000	1.000
Yes	2 (1.82%)	1 (0.91%)		
No	108 (98.18%)	109 (99.09%)		
Pregnancy times (times) [n (%)]			0.520	0.471
< 2	72 (65.45%)	77 (70.00%)		
≥ 2	38 (34.55%)	33 (30.00%)		

HC: healthy control; GDM: gestational diabetes mellitus; BMI: body mass index.

Pregnancy outcomes

Pregnancy outcomes were systematically recorded and analyzed, including incidences of polyhydramnios, premature rupture of membranes, cesarean delivery, preterm birth, fetal distress, macrosomia, neonatal hypoglycemia, and other complications [17].

Neonatal status was assessed using the Apgar scoring system at 1 and 5 minutes post-delivery by a trained midwife or physician [18]. The Apgar score ranges from 0 to 10, with higher scores indicating better neonatal health. If the 5-minute score remained below 7, further evaluations were conducted every 5 minutes until 20 minutes postpartum.

Statistical analysis

All statistical analyses were performed using SPSS 29.0 (SPSS Inc., Chicago, IL, USA). Categorical variables were expressed as counts and percentages [n (%)], and compared using the chi-square test. Continuous variables were tested for normality using the Shapiro-Wilk test. Normally distributed data were expressed as mean ± standard deviation (Mean ± SD), and analyzed using independent-sample t-tests with variance adjustment as needed. A two-tailed *P*-value <0.05 was considered significant.

Pearson correlation analysis was conducted to evaluate relationships between TXNIP, PINK1, and lipid metabolism values. Spearman correlation analysis was used to assess associations between lipid metabolism and GDM.

Results

Comparison of general characteristics

There were no statistically significant differences between the groups in terms of age, gestational age, pre-pregnancy body mass index (BMI), hypertension, smoking or alcohol consumption, gravidity, household registration, or education level (all P > 0.05; **Table 2**). However, a significantly higher proportion of participants in the GDM group had a family history of diabetes compared to controls (P < 0.05), suggesting possible hereditary predisposition to GDM.

Comparison of glucose metabolism values

Glucose metabolism markers differed significantly between groups. The GDM group showed elevated levels of FBG, 2hPBG, HbA1c, and HOMA-IR compared to the control group (all P <

Table 3. Comparison of glucose related values

Data	HC group (n = 110)	GDM group (n = 110)	t	P
FBG (mmol/L)	5.63 ± 1.15	7.81 ± 1.56	11.784	< 0.001
2hPBG (mmol/L)	7.01 ± 1.59	10.58 ± 2.15	14.008	< 0.001
HbA1c (%)	5.13 ± 1.14	6.54 ± 1.29	8.583	< 0.001
FINS (mmol/L)	11.62 ± 2.32	10.82 ± 2.84	2.289	0.023
HOMA-IR	1.02 ± 0.35	1.65 ± 0.49	11.071	< 0.001

HC: healthy control; GDM: gestational diabetes mellitus; FBG: fasting blood glucose; 2hPBG: 2 hours postprandial blood glucose; HbAlc: glycated hemoglobin; FINS: fasting insulin; HOMA-IR: homeostasis model insulin resistance index.

Table 4. Comparison of thyroid function values

Data	HC group (n = 110)	GDM group (n = 110)	t	P
FT3 (pmmol/L)	4.62 ± 0.82	4.72 ± 0.52	1.054	0.293
FT4 (pmmol/L)	12.21 ± 3.28	11.62 ± 2.58	1.479	0.141
TSH (μIU/mL)	1.86 ± 0.78	2.05 ± 0.85	1.684	0.094
T3 (ng/mL)	1.89 ± 0.83	2.21 ± 0.91	2.730	0.007
T4 (ng/mL)	110.27 ± 11.38	115.09 ± 18.06	2.368	0.019

HC: healthy control; GDM: gestational diabetes mellitus; FT3: free tri-iodothyronine; FT4: free thyroxine; TSH: thyroid stimulating hormone; T3: triiodothyronine; T4: thyroxine.

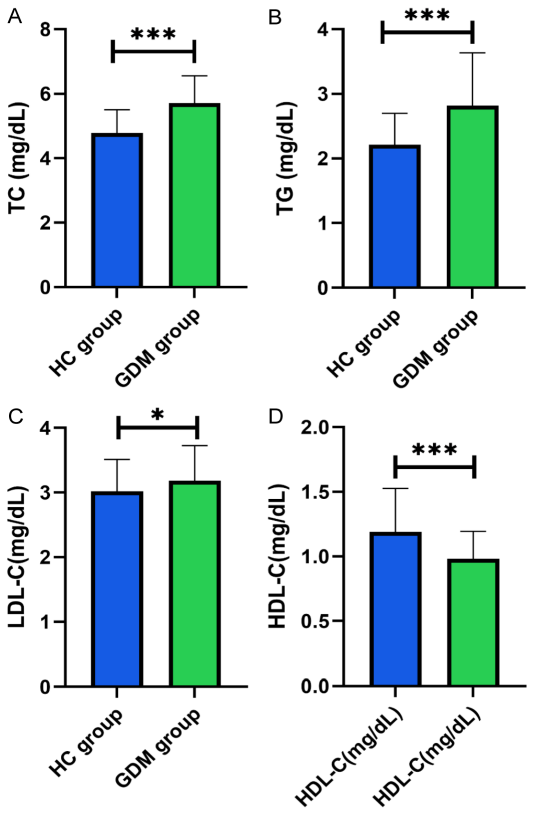

Figure 1. Comparison of lipid metabolism values between the two groups. (A) TC (B) TG (C) LDL-C (D) HDL-C. *P < 0.05, ***P < 0.001. HC: healthy control; GDM: gestational diabetes mellitus; TC: total cholesterol; TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

0.05; **Table 3**). In contrast, FINS was significantly lower in the GDM group (P < 0.05), indicating impaired insulin secretion or sensitivity. These results reinforce the need for strict glycemic monitoring in GDM management.

Comparison of thyroid function values

Significant differences in thyroid function were observed between groups. Total T3 (P = 0.007), and total T4 (P = 0.019) were all significantly elevated in the GDM group (**Table 4**). However, TSH, free T3 (FT3), and free T4 (FT4) levels showed no significant differences (both P > 0.05). These findings suggest that GDM may be associated with altered thyroid hormone metabolism or regulatory mechanisms during pregnancy.

Comparison of lipid metabolism values

Lipid profiles showed marked differences between groups. TC, TG, and LDL-C were significantly higher in the GDM group (both P < 0.001 for TC and TG; P = 0.017 for LDL-C), whereas HDL-C was significantly lower (P < 0.001; **Figure 1**). These results underscore the importance of lipid monitoring in GDM pregnancies to mitigate metabolic and cardiovascular risks.

Comparison of TXNIP and PINK1 expression

Expression levels of TXNIP and PINK1 differed significantly between groups. TXNIP mRNA lev-

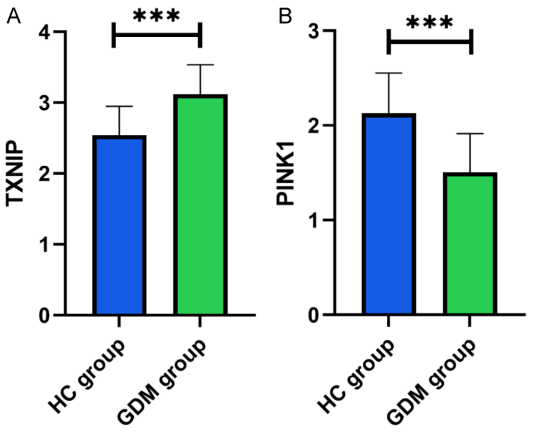

Figure 2. Expression of TXNIP and PINK1 levels between the two groups. (A) TXNIP (B) PINK1. ***P < 0.001. HC: healthy control; GDM: gestational diabetes mellitus; TXNIP: thioredoxin interacting protein; PINK1: PTEN-induced putative kinase 1.

els were significantly elevated in the GDM group, whereas PINK1 expression was significantly reduced (both P < 0.001; **Figure 2A**, **2B**). These findings suggest involvement of TXNIP and PINK1 in the pathophysiology of GDM.

Comparison of pregnancy outcomes

Adverse pregnancy outcomes were more common in the GDM group. These included higher rates of premature rupture of membranes (P = 0.005), cesarean delivery (P < 0.001), preterm birth (P = 0.010), macrosomia (P = 0.038), and neonatal hypoglycemia (P = 0.041) (**Table 5**). There were no significant differences in the incidence of polyhydramnios (P = 0.130) or fetal distress (P = 0.130). These findings highlight the heightened obstetric risks associated with GDM.

Comparison of neonatal outcomes

Neonatal outcomes were significantly poorer in the GDM group. Birth weight (P = 0.014) was significantly higher, while 1-minute Apgar scores (P < 0.001), and 5-minute Apgar scores (P = 0.006) were all significantly lower in the GDM group compared to controls (**Table 6**). This indicates compromised neonatal health and adaptation.

Correlation between TXNIP and lipid metabolism

TXNIP expression positively correlated with TC (ρ = 0.316, P < 0.001), TG (ρ = 0.279, P < 0.001), and LDL-C (ρ = 0.152, P = 0.025), and negatively with HDL-C (ρ = -0.237, P < 0.001) (**Figure 3**). These results suggest TXNIP may contribute to dysregulated lipid metabolism in GDM.

Correlation between PINK1 and lipid metabolism

PINK1 expression negatively correlated with TC (ρ = -0.374, P < 0.001) and TG (ρ = -0.231, P < 0.001), and positively with HDL-C (ρ = 0.241, P < 0.001). No significant correlation was observed with LDL-C (ρ = -0.032, P = 0.635) (**Figure 4A-D**). These findings suggest that PINK1 may exert a regulatory role in lipid homeostasis, particularly affecting cholesterol and triglyceride levels.

Correlation between lipid values and GDM

GDM status was positively correlated with TC (ρ = 0.513, P < 0.001), TG (ρ = 0.400, P < 0.001), and LDL-C (ρ = 0.146, P = 0.031), and negatively correlated with HDL-C (ρ = -0.357, P < 0.001) (**Figure 5**). These associations confirm that adverse lipid profiles are strongly linked to GDM.

Discussion

The findings of this study provide important insight into the associations among TXNIP, PINK1, and lipid metabolism in GDM, along with their clinical implications. Significant differences in glucose and lipid metabolic function were observed between GDM patients and HCs, accompanied by altered expression of TXNIP and PINK1, which may play crucial roles in GDM pathophysiology.

GDM patients exhibited significantly higher levels of FBG, 2hPBG, HOMA-IR, and HbA1c compared to HCs. Total T3 and total T4 were significantly elevated in the GDM group compared to HCs. Moreover, dyslipidemia was prominent in the GDM group, characterized by elevated TG, TC, and LDL-C, and reduced HDL-C levels. These lipid abnormalities are consistent with previous findings linking GDM to increased cardiovascular risk [19, 20]. Dysregulated lipid metabolism may further promote insulin resistance and exacerbate metabolic disturbances during pregnancy.

A key finding of this study was the altered expression of TXNIP and PINK1 in GDM patients. TXNIP was significantly upregulated,

Table 5. Comparison of pregnancy outcomes

Data	HC group (n = 110)	GDM group (n = 110)	X^2	P
Excessive amniotic fluid [n (%)]	0 (0.00%)	4 (3.64%)	2.292	0.130
Premature rupture of membrane [n (%)]	1 (0.91%)	10 (9.09%)	7.751	0.005
Cesarean section [n (%)]	10 (9.09%)	44 (40.00%)	28.371	< 0.001
Premature [n (%)]	1 (0.91%)	9 (8.18%)	6.705	0.010
Fetal distress [n (%)]	0 (0.00%)	4 (3.64%)	2.292	0.130
Macrosomia [n (%)]	0 (0.00%)	6 (5.45%)	4.283	0.038
Neonatal Hypoglycemia [n (%)]	1 (0.91%)	8 (7.27%)	4.171	0.041

HC: healthy control; GDM: gestational diabetes mellitus.

Table 6. Comparison of neonatal outcomes

Data	HC group (n = 110)	GDM group (n = 110)	t	P
Newborn weight (g)	3212.79 ± 420.61	3337.91 ± 319.45	2.485	0.014
1 min Apgar	9.44 ± 0.25	9.17 ± 0.28	7.777	< 0.001
5 min Apgar	9.54 ± 0.21	9.45 ± 0.25	2.788	0.006

HC: healthy control; GDM: gestational diabetes mellitus.

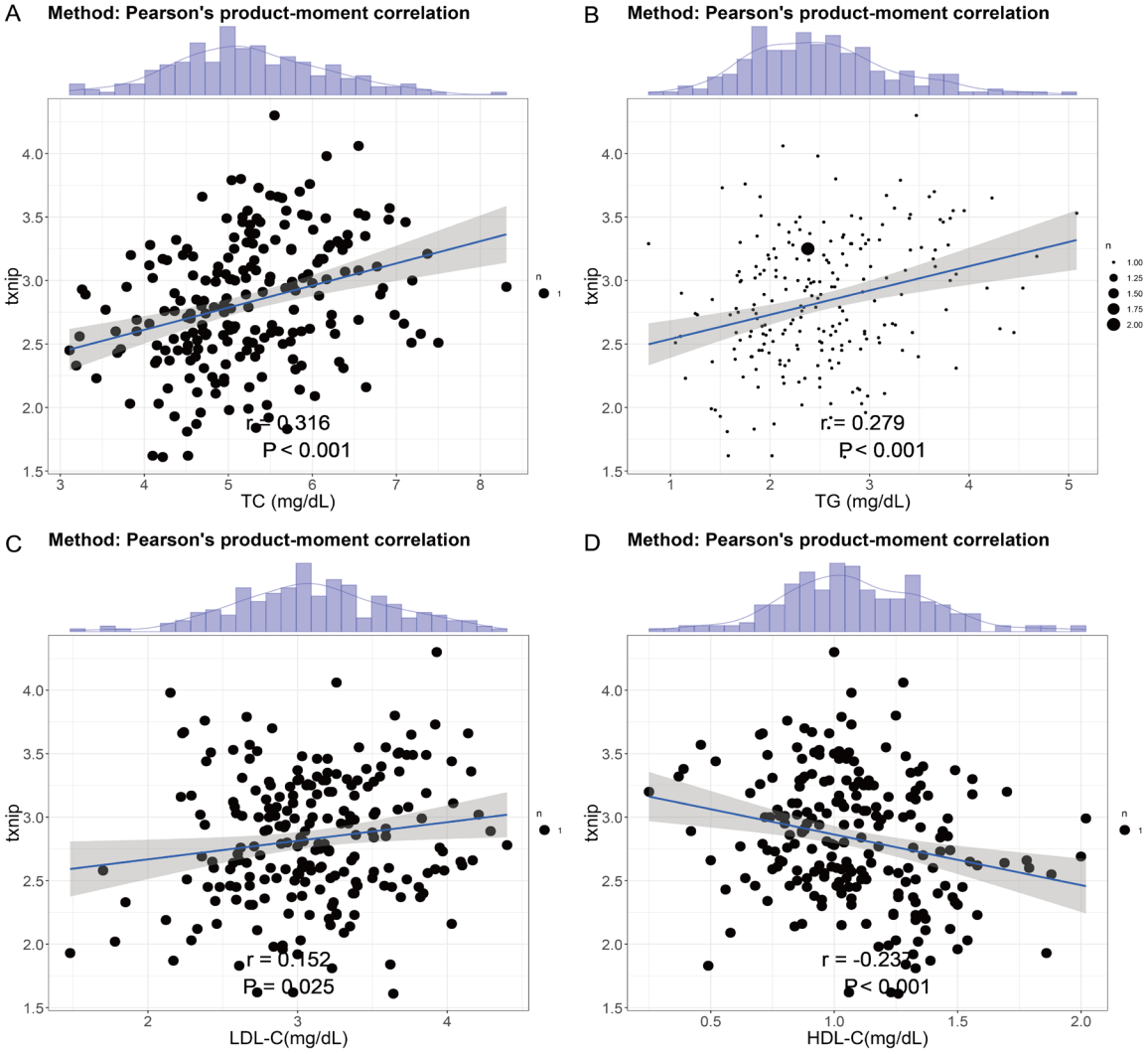

Figure 3. The correlation between TXNIP and lipid metabolism. (A) Correlation between TXNIP and TC (B) Correlation between TXNIP and TG (C) Correlation between TXNIP and LDL-C (D) Correlation between TXNIP and HDL-C. TXNIP: thioredoxin interacting protein; TC: total cholesterol; TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

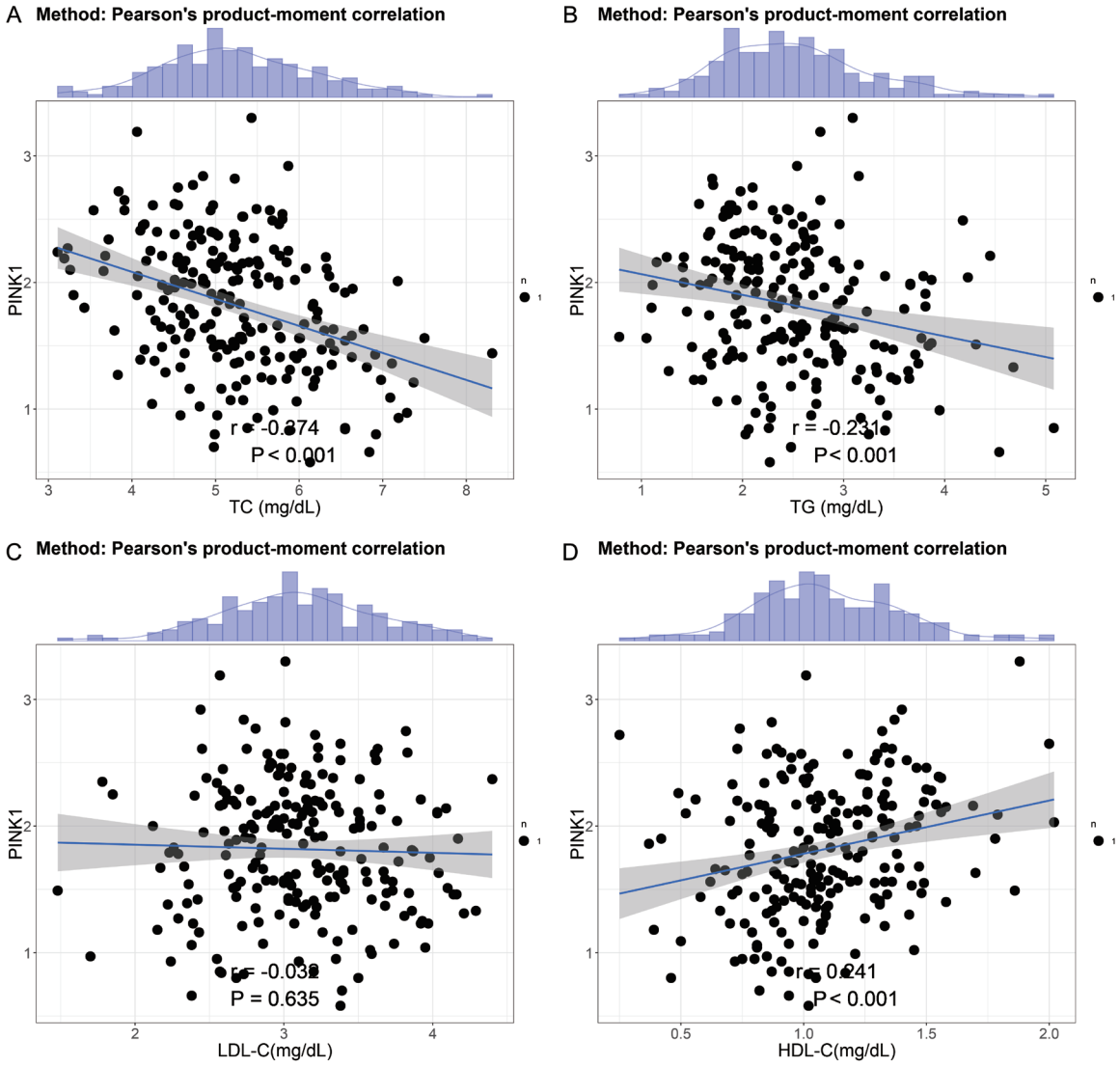

Figure 4. The correlation between PINK1 and lipid metabolism. (A) Correlation between PINK1 and TC (B) Correlation between PINK1 and TG (C) Correlation between PINK1 and LDL-C (D) Correlation between PINK1 and HDL-C. PINK1: PTEN-induced putative kinase 1; TC: total cholesterol; TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

whereas PINK1 was downregulated, suggesting their involvement in the metabolic dysfunctions associated with GDM.

TXNIP is known to regulate oxidative stress and has been implicated in the development of diabetes and its complications [21]. Elevated TXNIP expression may contribute to insulin resistance and dyslipidemia by influencing transcription factors such as sterol regulatory element-binding proteins (SREBPs), which control cholesterol and lipid synthesis [22]. Moreover, TXNIP-induced oxidative stress may promote lipid peroxidation, disrupting lipid homeostasis and worsening the metabolic burden in GDM [23-25]. Consistent with this, TXNIP expression was positively correlated with TC, TG, and LDL-C, and negatively correlated with HDL-C, sup-

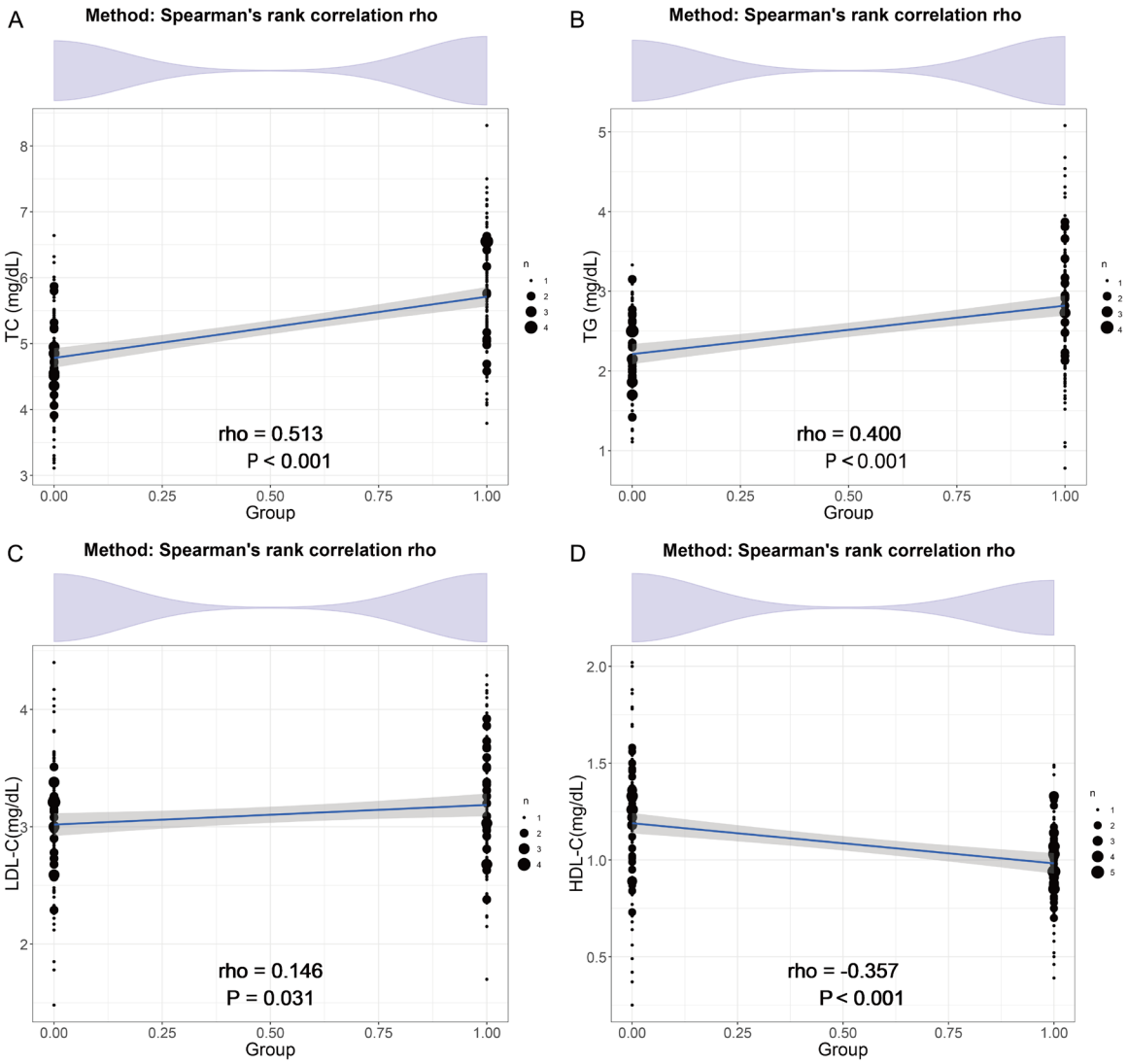

Figure 5. The correlation between lipid metabolism and GDM. (A) Correlation between GDM and TC (B) Correlation between GDM and TG (C) Correlation between GDM and LDL-C (D) Correlation between GDM and HDL-C. GDM: gestational diabetes mellitus; TC: total cholesterol; TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

porting its role in promoting lipid accumulation and impairing HDL function in GDM.

Conversely, PINK1, a mitochondrial kinase essential for mitophagy and mitochondrial quality control [26, 27], was significantly downregulated in GDM patients. Reduced PINK1 activity may impair mitochondrial function, elevate oxidative stress, and exacerbate lipid dysregulation. PINK1 expression was negatively correlated with TC and TG, and positively correlated with HDL-C, suggesting a role in promoting lipid clearance and preserving metabolic homeostasis. The absence of a significant correlation between PINK1 and LDL-C indicates that PINK1 may exert its lipid-modulatory effects through selective pathways rather than directly regulating LDL metabolism.

In addition to metabolic disturbances, GDM was associated with adverse pregnancy outcomes, including increased rates of cesarean delivery, preterm birth, macrosomia, and neonatal hypoglycemia. These findings align with prior reports that link GDM to higher obstetric and neonatal risks [28-30]. Higher birth weight

and lower Apgar scores in newborns of GDM mothers underscore the effect of maternal metabolic status on neonatal well-being.

The clinical implications of these findings are substantial. First, the pronounced abnormalities in glucose and lipid metabolism support the need for comprehensive metabolic monitoring during pregnancy. Early intervention targeting both glycemic control and lipid regulation may help reduce adverse pregnancy outcomes and long-term cardiometabolic risk. Second, the differential expression of TXNIP and PINK1 suggests their value as diagnostic biomarkers or therapeutic targets. Future research should explore whether reducing TXNIP expression or enhancing PINK1 activity could improve metabolic profiles in GDM.

Despite its strengths, this study has limitations. It was conducted at a single center, and the sample size, although adequate, may not fully represent the general population. Moreover, the cross-sectional design limits causal inference between TXNIP, PINK1, and GDM development. Longitudinal and mechanistic studies are warranted to confirm these associations and elucidate the pathways involved.

In conclusion, this study demonstrates that GDM is associated with significant disruptions in glucose, thyroid function, and lipid metabolism, accompanied by altered expression of TXNIP and PINK1. These molecules may play pivotal roles in GDM pathophysiology and may be biomarkers or therapeutic targets. Further research is needed to elucidate their mechanistic roles and make them into targets for improved management of GDM and its complications.

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Disclosure of conflict of interest

None.

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