*Iran J Public Health, Vol. 54, No.8, Aug 2025, pp.1712-1720*

**Original Article**

# Association between Thyrotoxicosis and Gestational Diabetes Mellitus: A Mendelian Randomization Study

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**(Received 11 Oct 2024; accepted 26 Jan 2025)**

**Abstract**

**Background:** Insulin resistance and abnormal glucose metabolism are the main characteristics of thyrotoxicosis and gestational diabetes mellitus (GDM). However, it remains unclear whether thyrotoxicosis increases the risk of GDM. Therefore, this research aimed to explore the causality between thyrotoxicosis and GDM by using a Mendelian randomization (MR) analysis.

**Methods:** A MR analysis was conducted to explore the causal effects of thyrotoxicosis on GDM. Summary statistics data of thyrotoxicosis (3115 thyrotoxicosis cases and 187684 controls) and GDM (13039 cases and 197831 controls) were derived from genome-wide association study. We selected MR Egger, Weighted median, Inverse-variance weighted, Simple mode and Weighted mode to evaluate the causal effect between thyrotoxicosis and GDM.

**Results:** By using a two-sample MR analysis, we found a strong causal relationship between thyrotoxicosis and GDM as indicated by Inverse-variance weighted (OR=1.069; beta=0.067; 95%CI=1.023-1.118; *P*=0.003), Weighted median (OR=1.087; beta=0.084; 95%CI=1.040-1.137; *P*=0.0002), Simple mode (OR=1.102; beta=0.097; 95%CI= 1.038-1.170; *P*=0.013) and Weighted mode (OR=1.089; beta=0.085; 95%CI=1.033-1.147; *P*=0.013). No significant pleiotropy, heterogeneity, genetic correlations or bi-directional causal relationship was existed in this study. Bayesian colocalization suggested that thyrotoxicosis colocalized with GDM on rs10830963 (PP.H4 = 1.000), where rs10830963 was located on *MTNR1B* gene locus.

**Conclusion:** Thyrotoxicosis had a causal effect on the risk of developing GDM, and the exposure of thyrotoxicosis increased the risk of GDM.

**Keywords:** Mendelian randomization; Gestational diabetes mellitus; Thyrotoxicosis; Insulin resistance

## Introduction

Gestational diabetes mellitus (GDM), characterized as glucose intolerance, is the most prevalent metabolic disorder during pregnancy (1). The prevalence of GDM varies substantially worldwide depending on population characteristics, ranging from 2% to 30% and steadily increasing globally (2-4). Metabolic abnormalities underlying GDM lead to adverse pregnancy outcomes, including stillbirth, fetal macrosomia and preterm delivery (5-7). Meanwhile, GDM increases the risk of developing type 2 diabetes mellitus (T2DM) and obesity after pregnancy (8).

The risk factors of GDM are still not fully recognized since the mechanism of GDM is yet unclear. While several epidemiological characteristics have been marked as the risk factors of GDM, including previous GDM (9), ages (10), obesity (11), and family history of diabetes (12), most of them are lack of accuracy in predicting the risk of GDM. Therefore, identifying more reliable risk factors is essential for early detection and prediction of GDM.

Insulin resistance and abnormalities in pancreatic beta cells are two major factors in women with GDM. Therefore, these factors affected glucose metabolism might be potential predictors for GDM. Thyroid dysfunction regulated glucose homeostasis, while thyrotoxicosis, characterized as the presence of excess thyroid hormone action at the tissue level, occurs more commonly in women compared with men (2% vs 0.2%) (13, 14), identified to increase the risk of atrial fibrillation (15), osteoporosis (16) and T2DM (17). Meanwhile, thyrotoxicosis is associated with reduced insulin sensitivity (18) and altered metabolism of glucose and free fatty acid (19, 20). Although several studies reported that thyrotropin level increased the risk of developing GDM in women (21), the other researchers found 14.3% of women with hypothyroidism developed GDM compared to 5.8% of hyperthyroid women (22). However, whether thyrotoxicosis causally associated with GDM is still unknown.

In this study, we conducted a two-sample Mendelian randomization analysis to evaluate the causal association of thyrotoxicosis on GDM based on summary data derived from the genome-wide association study (GWAS). Our study illustrated the link between thyrotoxicosis and GDM, highlighting potential mechanistic in genetics of thyrotoxicosis -GDM relationship.

## Methods

### *Samples and study designs*

This study adhered to Strengthening the Reporting of Observational Studies in Epidemiology using Mendelian Randomization (STROBE-MR) guidelines (23). In this study, we used genome-wide association studies (GWAS) summary statistics data to investigate whether the exposure of thyrotoxicosis had a causal association on GDM by conducting a two sample MR analysis. The genetic associations with thyrotoxicosis were acquired from the Finn biobank. The Finn biobank collects the deep traits and genetic data of 190799 individuals with thyrotoxicosis information, including 3115 thyrotoxicosis cases and 187684 controls (https://r5.finngen.fi/). The GWAS summary data of GDM were obtained from the Finn biobank, including 13039 cases and 197831 controls (24). Single-nucleotide polymorphisms (SNPs) were selected as instrumental variables (IVs) to explore the causal relationship between thyrotoxicosis and GDM. Meanwhile, the GWAS summary statistics of thyrotoxicosis and GDM were obtained from European ancestry. All MR analysis should fulfill the three important hypotheses: i) the IVs are strongly correlated to exposure ($P < 5e–8$); ii) the exposure and outcome are independent of any known confounders; iii) the IVs affected outcome only via exposure. All data for this study were sourced from previously published research and public databases, thus obviating the need for additional ethical approval.

### *IVs selection*

To ensure each SNPs have independent effect, we conducted a linkage disequilibrium (LD) analysis. After conducting a clumping procedure (clumping distance=10000 kb, $R^2<0.001$), we further evaluated the strength of IVs by calculating F-statistics, and excluded the IVs when F-statistics<10. The detailed mathematical formula of calculation of F-statistics were well-described in previous study (25). Additionally, the minor allele frequency (MAF) of IVs should more than the threshold of 0.01. Finally, we removed SNPs with incompatible alleles via a harmonized analysis.

### *MR analysis, sensitivity analysis and colocalization analysis*

All MR analysis and statistical analysis were conducted in R 4.2.0 (R Development Core Team, Australia). The MR analyses were conducted by using the TwoSampleMR v0.5.7 (https://mrcieu.github.io/TwoSampleMR/news/index.html). In this study, we selected inverse-variance weighted (IVW) as the main approach to evaluate causal relationships between thyrotoxicosis and GDM. Weighted mode, Weighted median, Mendelian randomization-egger (MR-Egger) were also used to investigate the potential causal relationship comprehensively. The Mendelian randomization pleiotropy residual sum and outlier (MR-PRESSO) method was used to determine outlier variants and horizontal pleiotropy. Meanwhile, we performed MR-Egger regression analysis to explore potential pleiotropy. Furthermore, the heterogeneity was quantified by using Cochran's Q-test via the MR-Egger and IVW methods. Additionally, we performed reverse MR analysis to estimate bidirectional causality between thyrotoxicosis and GDM traits. Finally, we estimated the colocalization of thyrotoxicosis and GDM traits via performing a Bayesian test using coloc R package (https://chr1swallace.github.io/coloc/, version 5.1.0). All variants within 200 kb of the leading SNPs in the thyrotoxicosis traits were selected for colocalization analysis. The threshold of posterior probability of H4 (PP.H4) was set as 0.95.

## Results

### *Linkage disequilibrium score regression analysis*

By using linkage disequilibrium score regression (LDSC) regression, we estimated genetic correlations and heritability between thyrotoxicosis and GDM. We found that heritabilities ($h^2$) were 1.3% and 2.2% for thyrotoxicosis and GDM, respectively (Table 1). Meanwhile, no significant genetic correlation was found between thyrotoxicosis and GDM (rg= −0.052, *P*=0.639).

**Table 1:** LDSC Regression Analyses

| Traits | H2 (se) | P_H2 | rg(se) | P_rg |
|---|---|---|---|---|
| Thyrotoxicosis | 0.013(0.003) | 2.01E-04 | -0.052(0.110) | 0.639 |
| GDM | 0.022(0.003) | 3.94E-11 | | |
| LDSC, linkage disequilibrium score regression; GDM, gestational diabetes mellitus | | | | |

### *Characteristics of the IVs*

11 SNPs were strongly associated with thyrotoxicosis ($P < 5e{-}8$) in European population. After harmonizing analysis, 2 variants were excluded due to duplicated SNPs and incompatible alleles. Meanwhile, the F-statistic of all selected IVs were more than 10 (Table S1). Finally, 9 SNPs were selected as IVs for further MR analysis (Table S1).

### *Forward MR analysis and Sensitivity analysis*

MR analysis (Table 2, Fig. 1) indicated that the exposure of thyrotoxicosis was associated with increased risk of GDM as indicated by IVW (OR=1.069; beta=0.067; 95%CI= 1.023-1.118; *P*=0.003), Weighted median (OR=1.087; beta = 0.084; 95%CI=1.040-1.137; *P*=0.0002), Simple mode (OR=1.102; beta = 0.097; 95%CI=1.038-1.170; *P*=0.013) and Weighted mode (OR=1.089; beta=0.085; 95%CI=1.033-1.147; *P*=0.013). By using MR pleiotropy residual sum and outlier (MR-PRESSO) analysis, we found no significant pleiotropy and outlier SNP in this study (*P* = 0.120). Meanwhile, the intercept of pleiotropy test was -0.0114 (*P* = 0.513), suggesting that no significant pleiotropic effect in the study. Nevertheless, no significant heterogeneity was found by using heterogeneity analysis based on MR-Egger (Q = 12.464, *P* = 0.086) and IVW (Q = 13.310, *P* = 0.102) tests (Table S2). Furthermore, no obvious and potential outliers IVs was observed in our study as indicated by leave-one-out (Fig. 2)

and funnel plots (Fig. 3). Collectively, these results indicated that the results of this study were of heightened reliability.

**Table 2:** Thyrotoxicosis was associated with the risk of GDM by MR analysis

| Method | IVs(n) | Beta | SE | OR (95%CI) | *P* |
|---|---|---|---|---|---|
| MR Egger | 9 | 0.098 | 0.051 | 1.103(0.998-1.218) | 0.095 |
| Weighted median | 9 | 0.084 | 0.023 | 1.087(1.040-1.137) | 0.0002 |
| Inverse variance weighted | 9 | 0.067 | 0.023 | 1.069(1.023-1.118) | 0.003 |
| Simple mode | 9 | 0.097 | 0.031 | 1.102(1.038-1.170) | 0.013 |
| Weighted mode | 9 | 0.085 | 0.027 | 1.089(1.033-1.147) | 0.013 |
| MR, Mendelian randomization; GDM, gestational diabetes mellitus; IV, instrumental variables; SE, standard error; OR, odds ratio | | | | | |

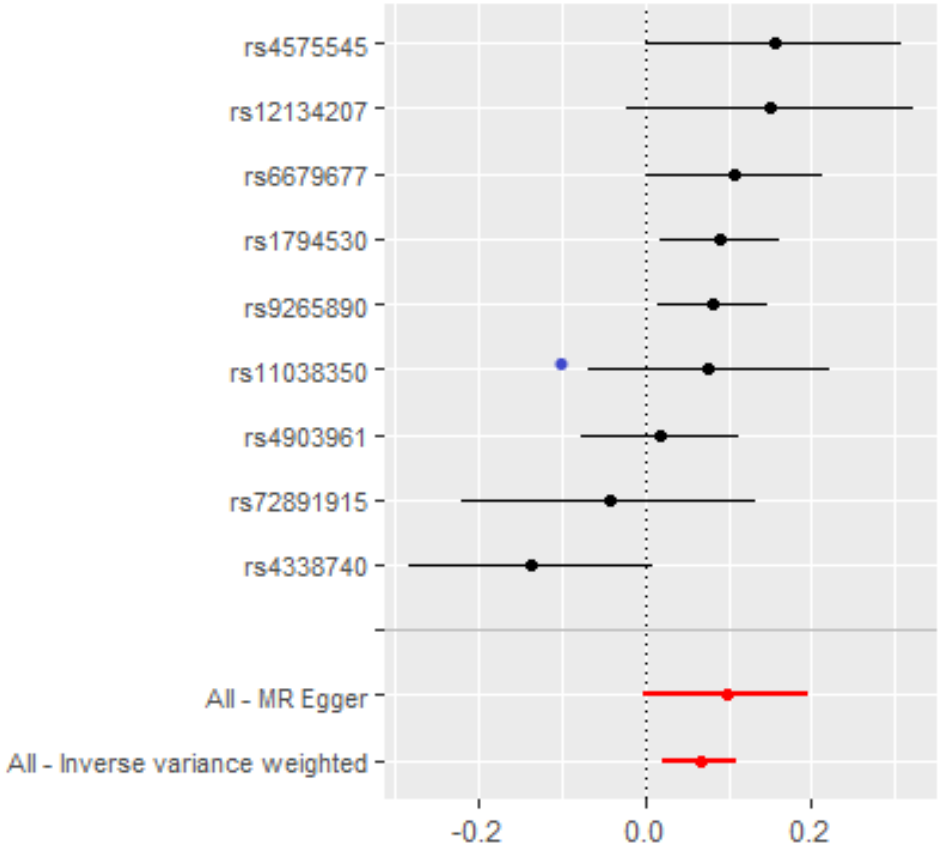


**Fig. 1:** MR analysis indicated that the exposure of thyrotoxicosis was associated with increased risk of GDM. MR, Mendelian randomization; GDM, gestational diabetes mellitus. MR-Egger, Mendelian randomization-egger

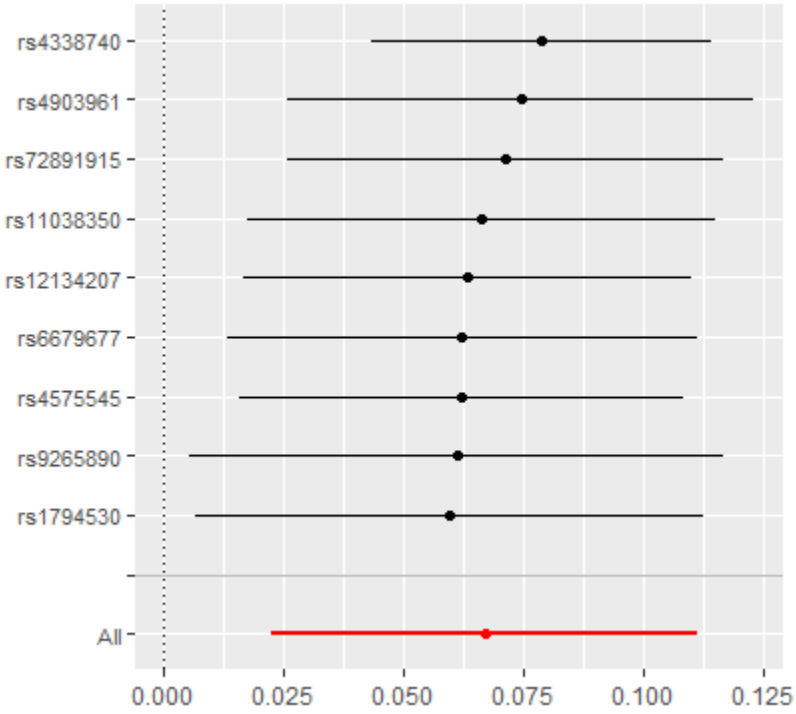


**Fig. 2:** Leave-one-out analysis. Each dot in the forest plot represents the MR estimate (using IVW) excluding that particular IVs. MR, Mendelian randomization; IVW, inverse-variance weighted; IV, instrumental variables

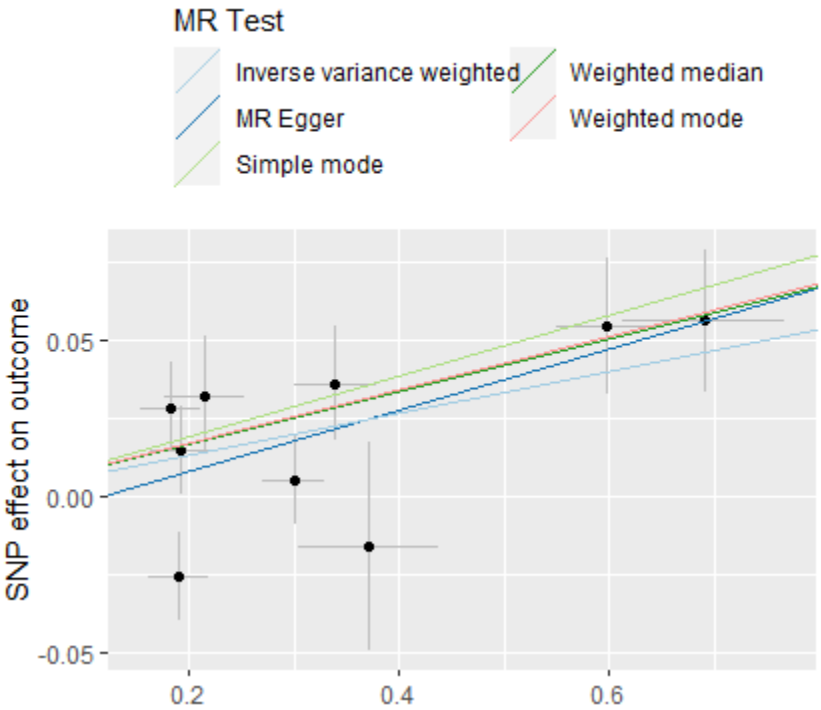


**Fig. 3:** The scatterplot depicted the causal relationship between thyrotoxicosis and GDM. MR-Egger, Mendelian randomization-egger; GDM, gestational diabetes mellitus, SNP, single-nucleotide polymorphisms

### *Reverse MR analysis*

In reverse MR analysis, 11 SNPs were selected as IVs (Table S3). By performing a reverse MR analysis, no significant bidirectional causality was found between GDM and thyrotoxicosis (Table 3), as indicated by *P* value of IVW method (*P*=0.959), MR Egger (*P*=0.424), Weighted median (*P*=0.618), Simple mode (*P*=0.728) and Weighted mode (*P*=0.499).

**Table 3:** GDM was unrelated to the risk of thyrotoxicosis by MR analysis

| Method | IVs(n) | Beta | SE | OR | *P* |
|---|---|---|---|---|---|
| Inverse variance weighted | 11 | 0.004 | 0.075 | 1.004 (0.867-1.162) | 0.959 |
| MR Egger | 11 | -0.132 | 0.158 | 0.876(0.644-1.193) | 0.424 |
| Weighted median | 11 | -0.034 | 0.069 | 0.966(0.845-1.106) | 0.618 |
| Simple mode | 11 | -0.060 | 0.169 | 0.941(0.676-1.311) | 0.728 |
| Weighted mode | 11 | -0.048 | 0.068 | 0.953(0.834-1.090) | 0.499 |
| MR, Mendelian randomization; GDM, gestational diabetes mellitus; IV, instrumental variables; SE, standard error; OR, odds ratio | | | | | |

### *Bayesian colocalization between thyrotoxicosis and GDM*

In the MR analysis, we found a causal association between thyrotoxicosis and GDM. To further validate the results of the MR analysis, we performed a Bayesian colocalization analysis. We found thyrotoxicosis colocalized with GDM on rs10830963 (PP.H4=1.000, Fig. 4), where rs10830963 was located on *MTNR1B* gene locus. In addition, the MAF of rs10830963 is 0.280 in European population according to dbSNP database.

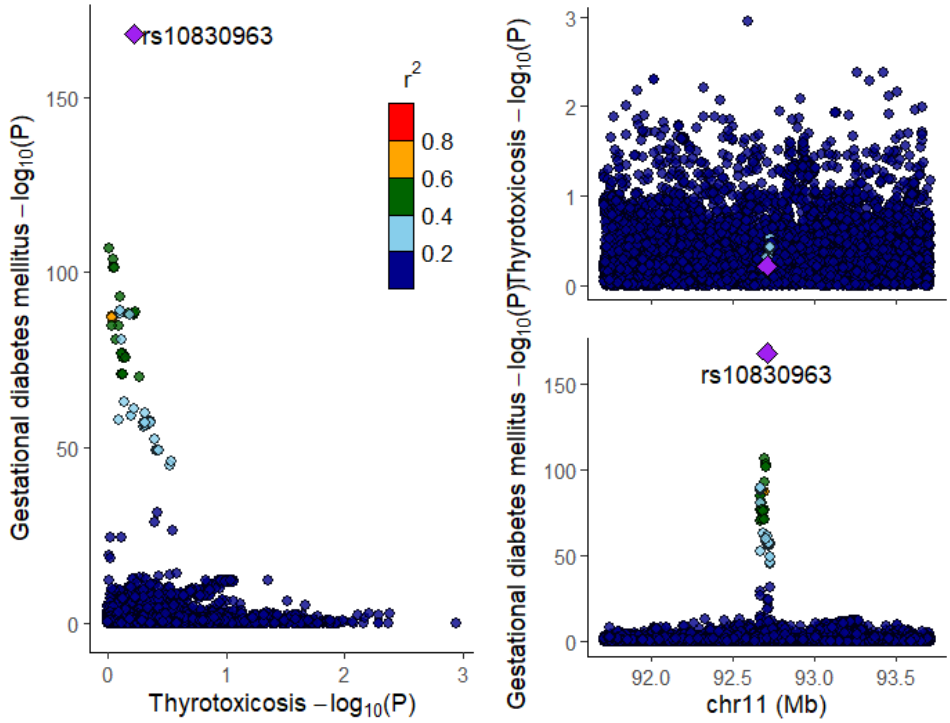


**Fig. 4:** Bayesian colocalization analysis found that thyrotoxicosis was colocalized with GDM on MTNR1B rs10830963 locus. GDM, gestational diabetes mellitus.

## Discussion

This two-sample MR study aimed to investigate the genetic causality between exposures to thyrotoxicosis and GDM by reanalyzing the summary statistics from GWAS studies. We found that thyrotoxicosis increased the risk of developing GDM, which was reliable and robust in sensitivity analysis. In addition, no bi-directional causal relationship existed in this study. Furthermore, thyrotoxicosis was colocalized with GDM on rs10830963 (PP.H4 = 1.000) via a Bayesian colocalization analysis, where rs10830963 was located on MTNR1B gene locus.

Indeed, several studies explored the relationship between thyrotoxicosis and GDM. A recent study reported that high thyroid stimulating hormone (TSH) and thyroid autoimmunity were associated with a 4-fold increased risk for GDM in 1170 women with singleton pregnancies (26). Moreover, Pinar Kumru and colleagues found that thyroid dysfunction had an impact on pregnancy outcomes (27), indicated that thyroid function plays vital roles in GDM and further affected outcomes of pregnancy. These findings were consistent with our results. Taken together, our study confirmed that thyrotoxicosis increased the risk of GDM for the first time by using a two-sample MR analysis, suggesting thyrotoxicosis acted as a genetic risk factor of GDM.

The next important question is why thyrotoxicosis affects the risk of GDM. Thyrotoxicosis was a key factor in regulating resistance, secretion, sensitivity and clearance of insulin (19, 28-30), which was also the main characteristics of GDM. Notably, previous studies have reported that thyroid supplementation in pregnancy was correlated to GDM risks (28). Therefore, thyrotoxicosis could reduce the response of insulin, resulting in insulin resistance, indicating that a potential causal relationship between thyrotoxicosis and GDM, which was consistent with our results. Collectively, thyrotoxicosis increased the risk of GDM by reducing sensitivity of insulin and promoting resistance of insulin.

By performing a COLOC analysis, we found the colocalization of thyrotoxicosis and GDM on the *MTNR1B* gene locus. This observation has not been previously reported by researchers. The *MTNR1B* gene encodes melatonin receptor 1B, a signaling receptor of melatonin, regulating the effects of circadian disruption on glucose metabolism (29). Importantly, the polymorphisms in *MTNR1B* were found associated with type 2 diabetes (30), and maternal gestational weight gain and childhood obesity (31). Additionally, the *MTNR1B* variant rs10830963 has been associated with an increased risk of GDM in both Asian and European populations (32, 33). Although no direct association between MTNR1B and thyrotoxicosis has been established, melatonin may promote thyrotoxicosis in preclinical models by regulating oxidative stress (34), as well as innate and adaptive immunity (35). Taken together, the increased risk of GDM associated with thyrotoxicosis may potentially involve disruptions in glucose metabolism mediated through melatonin-related signaling pathways. Further research is warranted to explore this relationship in detail.

This study has several limitations. First, we selected 9 SNPs as IVs with limited association with thyrotoxicosis (R2 [%]:1.1%-6.5%). Thus, other factors besides thyrotoxicosis that affected the risk of developing GDM should be considered in future studies. Second, we only used summary statistics of GDM and thyrotoxicosis in European populations, whether the causal association between thyrotoxicosis and GDM exists in other populations needs to be explored in further investigations. Third, inconsistencies in diagnostic criteria for GDM may have contributed to variations in the results. Lastly, MR analysis did not fully capture the lifetime impact of genetic variants whose correlation to the exposure changes over time, such as weight in pregnant women.

## Conclusion

Thyrotoxicosis had a causal relationship with an increased risk of developing GDM. Our findings

offered genetic evidence supporting the notion that individuals with thyrotoxicosis may have an inherent predisposition to a higher risk of GDM due to reduced insulin sensitivity and increased insulin resistance. Further studies are needed to determine their relationship in more detail.

## Journalism Ethics considerations

Ethical issues (Including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc.) have been completely observed by the authors.

## Acknowledgements

This work was supported by the National Natural Science Foundation of China [grant number 82304629], the Natural Science Foundation of Xiamen, China [grant number 3502Z202371048].

## Conflict of interest

The authors declare that there is no conflict of interests.

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