RESEARCH PAPER

Maternal Hypoglycemia During Oral Glucose Tolerance Test and Neonatal Outcomes: A Retrospective Observational Study

Dr. Samyuktha B S[1], Dr. Rajalekshmi M[2]

[1]Postgraduate, Department of Obstetrics and Gynecology, Saveetha Medical College and Hospital, SIMATS, Chennai, India. Email: samsaro2322@gmail.com

[2]Professor, Department of Obstetrics and Gynecology, Saveetha Medical College and Hospital, SIMATS, Chennai, India. Email: dr.raji@live.in (Corresponding Author)

***Corresponding author: Dr. Rajalekshmi M, Professor, Department of Obstetrics and Gynecology, Saveetha Medical College and Hospital, SIMATS, Chennai, India**

Email: dr.raji@live.in

Received: 5th May, 2026; **Revised:** 18th May, 2026; **Accepted:** 22nd May, 2026; **Available Online:** 11th June, 2026

ABSTRACT

Background

Considerable attention has been directed toward the maternal and neonatal consequences of gestational diabetes mellitus. However, the clinical significance of maternal hypoglycemia occurring during a routine oral glucose tolerance test (OGTT) in women who do not meet diagnostic criteria for gestational diabetes remains poorly understood. Whether such hypoglycemic values constitute a metabolic signal that adversely influences fetal growth is an open question with direct implications for antenatal surveillance.

Objective

To determine whether maternal hypoglycemia during the 75-g OGTT is associated with adverse neonatal outcomes, with particular attention to small for gestational age (SGA) and low birth weight (LBW) in a non-diabetic obstetric cohort.

Methods

A retrospective, record-based observational study was conducted at a tertiary care teaching hospital. All women who delivered during a six-month period and had undergone a 75-g OGTT at 24–28 weeks without a diagnosis of gestational diabetes were included. Maternal hypoglycemia was defined as any plasma glucose value below 3.9 mmol/L (70 mg/dL) at the fasting, one-hour, or two-hour time point. The primary outcomes were SGA (birth weight <10th percentile), LGA (>90th percentile), and mean birth weight. Secondary outcomes included LBW (<2500 g), macrosomia, preterm birth, NICU admission, and Apgar scores. Group comparisons were made using the independent t-test and chi-square test. Logistic regression was performed to assess independent associations after adjusting for maternal age, BMI, parity, and gestational weight gain.

Results

Of 716 eligible women, 52 (7.3%) were classified as hypoglycemic and 664 (92.7%) as normoglycemic. Women in the hypoglycemia group had a significantly lower booking BMI (22.1 ± 2.8 vs. 24.3 ± 3.4 kg/m²; p<0.001). Mean birth weight was lower in the hypoglycemia group (2685 ± 412 g vs. 2894 ± 436 g; p=0.002). SGA was more common among neonates of hypoglycemic mothers (21.2% vs. 10.1%; p=0.01), as was LBW (26.9% vs. 14.7%; p=0.02). On multivariable analysis, maternal hypoglycemia was independently associated with SGA (adjusted OR 1.92; 95% CI 1.01–3.64; p=0.04).

Conclusion

Maternal hypoglycemia during the 75-g OGTT occurs in approximately one in thirteen non-diabetic pregnancies and is independently associated with SGA. These findings suggest that low glucose values during OGTT, not merely elevated ones, warrant clinical attention. Prospective studies with serial fetal growth surveillance are needed to determine whether targeted antenatal monitoring in this subgroup improves outcomes.

Keywords: maternal hypoglycemia; oral glucose tolerance test; small for gestational age; neonatal outcomes; fetal growth restriction; retrospective observational study.

How to cite this article: Samyuktha BS, Rajalekshmi M. Maternal Hypoglycemia During Oral Glucose Tolerance Test and Neonatal Outcomes: A Retrospective Observational Study. Int J Drug Deliv Technol. 2026;16(58s):1651-1658. DOI: 10.25258/ijddt.16.58s.175

Source of support: Nil.

Conflict of interest: None

1. INTRODUCTION

Gestational diabetes mellitus (GDM) occupies a disproportionately large share of obstetric and perinatal research, and with good reason: its prevalence is rising globally, and its consequences for mother and neonate are well characterised [1,2]. The adverse outcomes associated with GDM — including macrosomia, preterm birth, neonatal hypoglycaemia, and caesarean delivery — have been consistently documented across diverse obstetric populations [3]. Yet the glucose tolerance test that serves as the primary diagnostic instrument for GDM also generates information that is routinely disregarded. Specifically, low glucose values recorded during the 75-g OGTT are noted, documented, and then set aside, because clinical thresholds for concern have been established only at the upper end of the glycaemic spectrum.

This may be an oversight. During the second half of pregnancy, fuel partitioning shifts substantially in favour of the fetoplacental unit. The placenta facilitates glucose transfer to the fetus through facilitated diffusion, a process that is directly proportional to the maternal-fetal glucose concentration gradient and therefore sensitive to reductions in maternal circulating glucose [4,5,6]. When maternal glucose falls below the threshold for normal fuel delivery, fetal glucose availability may be compromised, with potential downstream effects on fetal growth and adipose deposition. The theoretical underpinning for a relationship between maternal hypoglycaemia and impaired fetal growth is, therefore, plausible.

Epidemiological data on this question are sparse but suggestive. A handful of studies have identified hypoglycaemic OGTT values in pregnant women without GDM at a prevalence ranging from 5% to 10%, and some have reported an association with reduced birth weight and increased rates of SGA [7,8,9,10]. Among underweight women in particular, low glucose challenge test results have been independently associated with SGA after adjustment for confounders [9]. More recently, a study examining flat OGTT curves noted reduced mean birth weight among affected neonates, highlighting the relevance of subnormal glucose profiles during pregnancy [10]. However, these studies have varied considerably in their definition of hypoglycaemia, the OGTT protocol employed, the confounders addressed, and the outcomes evaluated. No consensus definition of clinically significant hypoglycaemia during OGTT exists, and the question of whether such values independently predict neonatal outcomes — after accounting for maternal BMI and other determinants of fetal growth — remains unresolved [7,8,9,10].

The present study was designed to address this gap. Using retrospectively collected data from a large tertiary obstetric unit, we examined whether maternal plasma glucose values below 3.9 mmol/L at any time point during the 75-g OGTT were associated with SGA, LBW, and other adverse neonatal outcomes in women who did not meet diagnostic criteria for GDM. A secondary aim was to determine whether any such association persisted after adjustment for relevant confounders.2. METHODS

2.1 Study Design and Setting

This was a retrospective, record-based observational study, reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [11]. The study was conducted in the Department of Obstetrics and Gynaecology of a tertiary care teaching hospital. Data were drawn entirely from existing hospital electronic medical records and laboratory information systems. No intervention was performed, and there was no direct contact with participants. The study period covered all eligible deliveries over six consecutive months.

2.2 Study Population

The study population comprised all pregnant women who delivered at the institution during the defined period and had undergone a standard 75-g OGTT between 24 and 28 weeks of gestation, consistent with internationally recommended screening practice [12]. Women were included if they were aged 18 years or older, carried a singleton pregnancy, had complete OGTT records (fasting, one-hour, and two-hour plasma glucose values), and had complete delivery and neonatal outcome data available.

Women were excluded if they had pre-existing diabetes mellitus or had received a diagnosis of gestational diabetes mellitus following OGTT. Additional exclusion criteria were multifetal gestation, chronic hypertension, known autoimmune disease or malignancy, documented fetal congenital anomaly, and incomplete or missing medical records.

2.3 Exposure Definition

Maternal hypoglycaemia was defined as any plasma glucose value below 3.9 mmol/L (70 mg/dL) at any time point during the 75-g OGTT — fasting, one hour, or two hours post-load — consistent with the American Diabetes Association threshold for clinically significant hypoglycaemia [7]. Participants were classified into two mutually exclusive groups: the hypoglycaemia group (at least one value below the threshold) and the normoglycaemia group (all values at or above the threshold).

2.4 Data Collection

Data were extracted from electronic records using a structured proforma. Maternal variables collected included age, pre-pregnancy or booking BMI, gravidity, parity, gestational weight gain (where available), all three OGTT glucose values, and gestational age at delivery. Neonatal variables

collected included birth weight, sex, gestational age at birth, Apgar scores at one and five minutes, and requirement for NICU admission.

2.5 Outcome Measures

The primary outcomes were SGA (birth weight below the 10th percentile for gestational age and sex), LGA (birth weight above the 90th percentile), and mean birth weight. Gestational age-specific percentile thresholds were applied using standard institutional reference charts.

Secondary outcomes comprised LBW (birth weight below 2500 g), macrosomia (birth weight above 4000 g), preterm birth (delivery before 37 completed weeks of gestation), NICU admission, and Apgar score below 7 at one and five minutes.

2.6 Sample Size Justification

Based on institutional records, approximately 150 deliveries occur per month. Over six months, an estimated 900 deliveries were expected. After excluding cases of GDM (approximately 10–15% of the obstetric population) and other ineligible cases, the anticipated eligible sample size was 750–800. Given a reported OGTT hypoglycaemia prevalence of approximately 7% in non-diabetic pregnant populations [7,8], between 50 and 60 hypoglycaemic cases were expected — a sample judged sufficient to detect clinically meaningful differences in SGA rates between groups. A consecutive sampling strategy was employed to include all eligible cases within the study period.

2.7 Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS version 20.0. Continuous variables are reported as mean ± standard deviation, and categorical variables as frequencies and percentages. Between-group comparisons were performed using the independent samples t-test for continuous variables and the chi-square test for categorical variables.

Univariate logistic regression was performed to estimate odds ratios (OR) with 95% confidence intervals for each neonatal outcome. Multivariable logistic regression was then conducted, adjusting for maternal age, BMI, parity, and gestational weight gain, to evaluate whether any observed associations were independent of these confounders. Statistical significance was set at $p<0.05$.

Subgroup analysis was performed comparing outcomes between women with fasting hypoglycaemia and those with post-load hypoglycaemia. Sensitivity analysis was conducted excluding the approximately 15% of participants with missing gestational weight gain data to assess whether this affected the primary findings.

2.8 Ethical Considerations

The study protocol was submitted to the Institutional Ethics Committee for review and approval prior to data access. As the study involved retrospective analysis of de-identified hospital records with no patient interaction or alteration of clinical care, it was classified as minimal risk. All data were anonymised before analysis. In view of the retrospective, record-based design and use of de-identified data, a waiver of informed consent was requested. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki [13].

3. RESULTS

3.1 Participant Flow

During the study period, 912 deliveries were recorded. Of these, 842 women had undergone a 75-g OGTT between 24 and 28 weeks of gestation. After applying eligibility criteria, 126 women were excluded (Figure 1), yielding a final study population of 716 participants. Of these, 52 women (7.3%) were classified into the hypoglycemia group and 664 (92.7%) into the normoglycemia group.

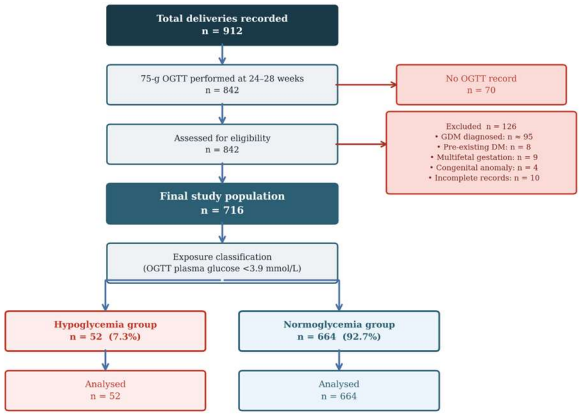

Figure 1. Participant Flow Diagram. OGTT, oral glucose tolerance test; GDM, gestational diabetes mellitus.

3.2 Baseline Maternal Characteristics

Baseline characteristics are summarised in Table 1. Mean maternal age was similar between groups (24.8 ± 3.6 vs. 25.6 ± 4.1 years; p=0.12). Booking BMI was significantly lower in the hypoglycemia group (22.1 ± 2.8 vs. 24.3 ± 3.4 kg/m²; $p<0.001$). A higher proportion of primigravida women was observed in the hypoglycemia group (61.5% vs. 48.2%), though this difference did not reach statistical significance (p=0.07). Mean gestational weight gain was lower in the hypoglycemia group, but this difference was also non-significant (9.1 ± 2.3 vs. 10.4 ± 2.8 kg; p=0.09). Gestational weight gain data were available for 612 of the 716 participants.

Table 1. Baseline Maternal Characteristics

Variable	Hypoglycemia (n=52)	Normoglycemia (n=664)	p-value
Age (years), mean ± SD	24.8 ± 3.6	25.6 ± 4.1	0.12
BMI (kg/m²), mean ± SD	22.1 ± 2.8	24.3 ± 3.4	**<0.001**
Primigravida, n (%)	32 (61.5%)	320 (48.2%)	0.07
Multipara, n (%)	20 (38.5%)	344 (51.8%)	—
Gestational weight gain* (kg), mean ± SD	9.1 ± 2.3	10.4 ± 2.8	0.09

*BMI, body mass index; SD, standard deviation. Bold p-values indicate statistical significance (p<0.05). *Gestational weight gain available for n=612 participants.*

3.3 Neonatal Outcomes

Neonatal outcomes are presented in Table 2. Mean birth weight was significantly lower in neonates of hypoglycemic mothers compared to normoglycemic mothers (2685 ± 412 g vs. 2894 ± 436 g; p=0.002). SGA occurred in 21.2% of neonates in the hypoglycemia group versus 10.1% in the normoglycemia group (p=0.01), and LBW was recorded in 26.9% versus 14.7% respectively (p=0.02). Rates of LGA, macrosomia, preterm birth, NICU admission, and low Apgar scores did not differ significantly between groups. The outcome distribution is illustrated in Figure 2 and Figure 3.

Table 2. Comparison of Neonatal Outcomes Between Groups

Outcome	Hypoglycemia (n=52)	Normoglycemia (n=664)	p-value
Birth weight (g), mean ± SD	2685 ± 412	2894 ± 436	**0.002**
SGA, n (%)	11 (21.2%)	67 (10.1%)	**0.01**
LGA, n (%)	1 (1.9%)	42 (6.3%)	0.18
LBW (<2500 g), n (%)	14 (26.9%)	98 (14.7%)	**0.02**
Macrosomia (>4000 g), n (%)	0 (0%)	14 (2.1%)	0.31
Preterm birth, n (%)	7 (13.5%)	65 (9.8%)	0.34
NICU admission, n (%)	9 (17.3%)	76 (11.4%)	0.19
Apgar <7 at 1 min, n (%)	6 (11.5%)	52 (7.8%)	0.28
Apgar <7 at 5 min, n (%)	2 (3.8%)	18 (2.7%)	0.65

SGA, small for gestational age; LGA, large for gestational age; LBW, low birth weight; NICU, neonatal intensive care unit. Bold p-values indicate statistical significance (p<0.05).

Figure 2. Mean Birth Weight by Maternal OGTT Glycaemic Status

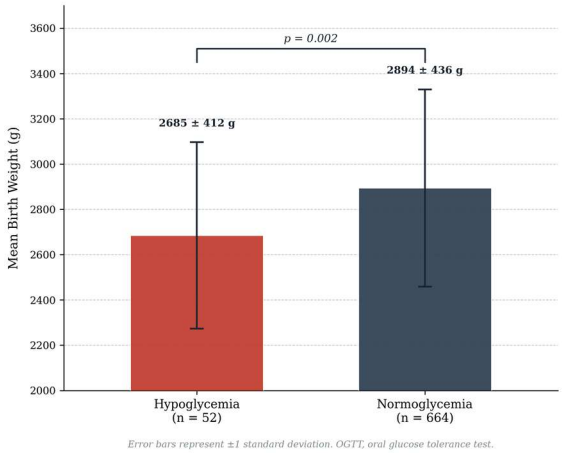

Figure 2. Mean birth weight (g) by maternal OGTT glycaemic status. Error bars represent ±1 SD. OGTT, oral glucose tolerance test.

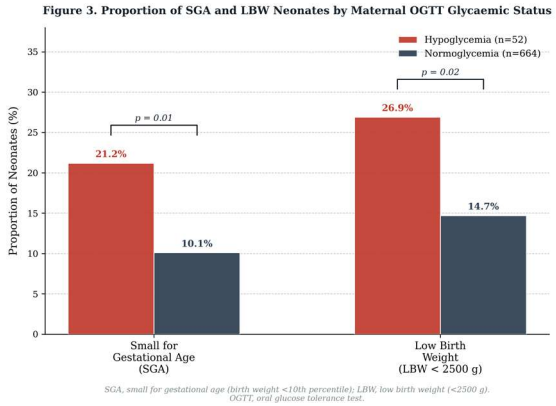

Figure 3. Proportion (%) of small for gestational age (SGA) and low birth weight (LBW) neonates by maternal OGTT glycaemic status.

3.4 Logistic Regression Analyses

On univariate analysis (Table 3), maternal hypoglycemia was associated with a significantly increased odds of SGA (OR 2.38; 95% CI 1.28–4.41; p=0.006) and LBW (OR 2.13; 95% CI 1.18–3.85; p=0.01). No significant associations were observed for preterm birth or NICU admission.

Table 3. Univariate Logistic Regression Analysis

Outcome	Odds Ratio (OR)	95% CI	p-value
SGA	2.38	1.28–4.41	**0.006**
LBW	2.13	1.18–3.85	**0.01**
Preterm birth	1.43	0.62–3.26	0.34
NICU admission	1.62	0.77–3.41	0.19

OR, odds ratio; CI, confidence interval. Bold p-values indicate statistical significance (p<0.05). Reference group: normoglycemia.

After adjusting for maternal age, BMI, parity, and gestational weight gain, the association between maternal hypoglycemia and SGA remained statistically significant (adjusted OR 1.92; 95% CI 1.01–3.64; p=0.04) (Table 4). The adjusted association with LBW was attenuated and no longer significant (adjusted OR 1.54; 95% CI 0.86–2.78; p=0.14), suggesting partial mediation by BMI and gestational weight gain. No significant adjusted associations were observed for preterm birth or NICU admission.

Table 4. Multivariable Logistic Regression Analysis (Adjusted)

Outcome	Adjusted OR	95% CI	p-value
SGA	1.92	1.01–3.64	**0.04**
LBW	1.54	0.86–2.78	0.14
Preterm birth	1.21	0.52–2.81	0.66
NICU admission	1.38	0.63–3.02	0.41

Adjusted for maternal age, BMI, parity, and gestational weight gain. Bold p-values indicate statistical significance (p<0.05).

3.5 Additional Analyses

Subgroup analysis by timing of hypoglycemia (fasting versus post-load) did not reveal statistically significant differences in neonatal outcomes. However, women with fasting hypoglycemia showed a non-significant trend toward lower mean birth weight compared with those whose hypoglycemia occurred only at post-load time points. Sensitivity analysis excluding participants with missing gestational weight gain data did not materially alter the primary findings.

4. DISCUSSION

In this retrospective study of 716 non-diabetic pregnant women from a tertiary obstetric unit, maternal hypoglycaemia during the 75-g OGTT was observed in 7.3% of the cohort. These women delivered neonates with lower mean birth weights and higher rates of SGA and LBW. After adjustment for maternal age, BMI, parity, and gestational weight gain, the association with SGA remained independently significant, though the adjusted association with LBW was attenuated. No statistically significant differences were found for preterm birth, NICU admission, or Apgar scores.

The prevalence of OGTT-associated hypoglycaemia in our cohort (7.3%) is consistent with rates reported in prior observational studies, which have documented this phenomenon in approximately 5%–10% of non-diabetic pregnancies [7,8]. That such a finding occurs with appreciable frequency, yet receives no systematic clinical follow-up under current guidelines, warrants reconsideration. Our data suggest that low OGTT

glucose values are not benign incidental findings but may identify a subset of women at elevated risk of delivering SGA neonates [7,8,14,15].

The mechanism through which maternal hypoglycaemia during OGTT might impair fetal growth is not entirely established, but several pathways are plausible. Placental glucose transfer is largely driven by the maternal–fetal glucose concentration gradient, and transient maternal hypoglycaemia during a critical window of placental development at 24–28 weeks could reduce net glucose flux to the fetus [4,5,6]. Additionally, recurrent or habitual hypoglycaemia — of which the OGTT value may be an index — could signal chronic nutritional insufficiency or an underlying substrate metabolism phenotype, manifesting as both reduced maternal BMI and impaired fetal growth. The significantly lower booking BMI in the hypoglycaemia group is consistent with this hypothesis, and the attenuation of the LBW association after adjustment for BMI supports partial mediation through maternal nutritional status [4,5].

Comparable findings have been reported in other institutional studies from similar populations, where low glucose values during oral loading tests were independently associated with SGA and composite neonatal morbidity [9,10,14]. A large retrospective cohort study found that a low maternal glucose challenge test value was significantly associated with increased neonatal morbidity driven by higher rates of SGA, even after multivariable adjustment [14]. Some investigators have proposed that the combination of low fasting glucose and exaggerated post-load insulin responses may characterise a phenotype of glucose homeostasis associated with intrauterine growth restriction, and that affected women may benefit from enhanced antenatal surveillance [14,15]. Whether this represents a biologically distinct subpopulation or a continuous spectrum of metabolic risk is not resolved by the present data but merits prospective investigation.

Several strengths of this study deserve mention. The sample was drawn from a single high-volume institution with standardised OGTT and delivery documentation practices, reducing the heterogeneity introduced by multi-centre data pooling. The exclusion of all GDM-positive cases eliminates the most obvious confounder and isolates the signal of interest. The use of multivariable logistic regression to adjust for key confounders — including BMI, which was significantly different between groups — strengthens the causal inference possible from these observational data. The sensitivity analysis confirming stability of findings after exclusion of cases with missing gestational weight gain data further supports the robustness of the primary result.

The limitations of this study must be acknowledged. The retrospective design inherently constrains the completeness and standardisation of data collection. Gestational weight gain data were unavailable for approximately 15% of the cohort, introducing potential residual confounding. OGTT hypoglycaemia was identified at a single time point, and no information on habitual dietary intake, fasting duration prior to the test, or physical activity patterns — all of which influence fasting glucose — was available. The definition of hypoglycaemia as a single value below 3.9 mmol/L at any time point during OGTT may lack specificity; a more stringent definition requiring multiple abnormal values might identify a higher-risk subgroup. Furthermore, as this study was conducted at a single tertiary centre serving a largely South Indian population, generalisability to other ethnic and geographic settings requires caution. The sample size, while adequate for detecting differences in SGA, may have been insufficient to demonstrate statistically significant associations for outcomes with lower event rates such as macrosomia or perinatal asphyxia.

Future research should address these limitations through prospective designs with serial fetal growth ultrasonography in women identified with OGTT hypoglycaemia. Characterisation of the full glucose and insulin response profile during OGTT — rather than relying on discrete threshold values — would provide a more granular understanding of the metabolic phenotype. Additionally, studies examining whether targeted nutritional intervention in women with OGTT hypoglycaemia can improve fetal growth outcomes would have direct clinical relevance.

5. CONCLUSION

Maternal hypoglycaemia during the 75-g OGTT, occurring in approximately 7% of non-diabetic pregnancies in this cohort, was independently associated with SGA after adjustment for relevant maternal characteristics. Women with OGTT hypoglycaemia also delivered neonates with lower mean birth weights and higher rates of LBW, though the latter association was partly explained by differences in maternal BMI. These findings indicate that the lower end of the glucose spectrum during OGTT carries prognostic information that is not currently captured in routine clinical practice. Prospective studies are needed to determine whether identifying and monitoring this subgroup translates into improved neonatal outcomes.

6. DECLARATIONS

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflicts of Interest

The authors declare no conflicts of interest.

Ethics Approval and Consent to Participate

The study was approved by the Institutional Ethics Committee. In view of the retrospective, record-based design and use of de-identified data, a waiver of individual informed consent was granted. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional data governance policies.

Author Contributions

Conceptualisation, study design, data collection, analysis, and manuscript preparation were conducted by the authors. All authors reviewed and approved the final manuscript.

Acknowledgements

The authors acknowledge the support of the Department of Obstetrics and Gynaecology and the Medical Records Department of the institution for facilitating data access.

— References to be added in a separate phase —

REFERENCES

1. GBD 2021 Diabetes Collaborators. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. Lancet. 2023;402(10397):203–234. doi:10.1016/S0140-6736(23)01301-6.
2. Cho NH, Shaw JE, Karuranga S, Huang Y, da Rocha Fernandes JD, Ohlrogge AW, et al. IDF Diabetes Atlas: global estimates of diabetes prevalence for 2017 and projections for 2045. Diabetes Res Clin Pract. 2018;138:271–281. doi:10.1016/j.diabres.2018.02.023.
3. Malaza N, Masete M, Adam S, Dias S, Nyawo T, Pheiffer C. A systematic review to compare adverse pregnancy outcomes in women with pregestational diabetes and gestational diabetes. Int J Environ Res Public Health. 2022;19(17):10846. doi:10.3390/ijerph191710846.
4. Joshi NP, Mane AR, Sahay AS, Sundrani DP, Joshi SR, Yajnik CS. Role of placental glucose transporters in determining fetal growth. Reprod Sci. 2021;29(10):2744–2759. doi:10.1007/s43032-021-00699-9.
5. Stanirowski PJ, Lipa M, Bomba-Opon D, Wielgos M. Expression of placental glucose transporter proteins in pregnancies complicated by fetal growth disorders. Adv Protein Chem Struct Biol. 2020;123:95–131. doi:10.1016/bs.apcsb.2019.12.003.
6. Tanner AR, Kennedy VC, Lynch CS, Winger QA, Anthony RV, Rozance PJ. Increasing maternal glucose concentrations is insufficient to restore placental glucose transfer in chorionic somatomammotropin RNA interference pregnancies. Am J Physiol Endocrinol Metab. 2024;326(5):E602–E615. doi:10.1152/ajpendo.00331.2023.
7. Bayraktar B, Balikoglu M, Kanmaz AG. Pregnancy outcomes of women with hypoglycemia in the oral glucose tolerance test. J Gynecol Obstet Hum Reprod. 2020;49(4):101703. doi:10.1016/j.jogoh.2020.101703.
8. Raviv S, Wilkof-Segev R, Maor-Sagie E, Naeh A, Yoeli Y, Hallak M, et al. Hypoglycemia during the oral glucose tolerance test in pregnancy — maternal characteristics and neonatal outcomes. Int J Gynaecol Obstet. 2021;158(3):585–591. doi:10.1002/ijgo.14037.
9. Shinohara S, Uchida Y, Hirai M, Hirata S, Suzuki K. Relationship between maternal hypoglycaemia and small-for-gestational-age infants according to maternal weight status: a retrospective cohort study in two hospitals. BMJ Open. 2016;6(12):e013749. doi:10.1136/bmjopen-2016-013749.
10. Lopian M, Segal E, Neiger R, Many A, Kashani Ligumsky L. The implications of a "flat" oral glucose tolerance test curve in pregnancy. Am J Perinatol. 2024;42(4):478–485. doi:10.1055/a-2405-1909.
11. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. J Clin Epidemiol. 2008;61(4):344–349. doi:10.1016/j.jclinepi.2007.11.008.
12. Petrovic O. How should we screen for gestational diabetes? Curr Opin Obstet Gynecol. 2014;26(2):54–60. doi:10.1097/GCO.0000000000000049.
13. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191–2194. doi:10.1001/jama.2013.281053.
14. Ding JJ, Lundsberg LS, Culhane JF, Patridge C, Milley L, Son M. The association between a low 50-gram, 1-hour glucose challenge test value and neonatal morbidity. J Matern Fetal Neonatal Med. 2023;36(2):2245527. doi:10.1080/14767058.2023.2245527.

15. Blunt C, Mathew S, Mung SM, Krishnamurthy R, Jude EB. Hypoglycaemia following the 2-hour 75g OGTT in pregnancy — investigating maternal and foetal outcomes. Diabetes Metab Syndr. 2024;18(3):102977. doi:10.1016/j.dsx.2024.102977.