World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(REVIEW ARTICLE)

Check for updates

# Advancing Metabolic Monitoring: Continuous Glucose Monitoring Across the Prenatal–Postnatal Spectrum of Dysglycemia

Ashraf T Soliman [1,*], Shayma Ahmed [1], Ahmed Elawwa [2], Fawzia Alyafei [1], Nada Alaaraj [1], Noor Hamed [1], Doaa Yassin [2] and Nada Soliman [3]

[1] *Department of Paediatrics, Hamad General Hospital, Doha, Qatar.*
[2] *Department of Pediatrics, University of Alexandria Children's Hospital, Alexandria, Egypt.*
[3] *Directorate of Health Affairs in Alexandria, Ministry of Health, Alexandria, Egypt*

World Journal of Advanced Research and Reviews, 2025, 28(02), 651-665

Publication history: Received on 14 September 2025; revised on 05 November 2025; accepted on 08 November 2025

Article DOI: https://doi.org/10.30574/wjarr.2025.28.2.3603

## Abstract

## Abstract

**Background:** Continuous glucose monitoring (CGM), through high-frequency real-time glucose profiling, offers a more complete characterization of glucose patterns compared to traditional diagnostic tools, particularly fasting glucose and the oral glucose tolerance test (OGTT), relevant to maternal–fetal health, postpartum risk, and pediatric metabolic regulation.

**Objectives:** This review compares the diagnostic performance of CGM versus OGTT and fasting glucose in high-risk pregnancies; evaluates CGM for early detection and prediction of dysglycemia in children and adolescents; and examines associations between CGM-derived glycemic stability, IGF-1 activity, and growth outcomes in pediatric dysglycemia**.**

**Methods:** We reviewed randomized trials, prospective cohorts, and observational pediatric studies (2009–2025) evaluating CGM in high-risk pregnancies, postpartum women, and pediatric groups—including obesity, prediabetes, autoantibody-positive relatives, cystic fibrosis, Prader–Willi syndrome, and β-thalassemia major. Due to heterogeneity in design and outcomes, findings were synthesized narratively.

**Results:** Across pregnancy, CGM consistently identified glycemic disturbances not captured by OGTT, including postprandial and nocturnal hyperglycemia and elevated variability. CGM-guided care improved maternal time-in-range, reduced time-above-range, and was associated with lower rates of large-for-gestational-age infants, neonatal hypoglycemia, and NICU admission. Postpartum, CGM demonstrated substantially higher follow-up adherence than OGTT and reliably detected persistent dysglycemia**.**

In pediatrics, CGM identified presymptomatic dysglycemia 12–24 months earlier than OGTT in children with type 1 diabetes autoimmunity and improved prediction of short-term progression to stage 3 diabetes. Among adolescents with obesity or prediabetes, CGM revealed frequent postprandial hyperglycemia despite normal fasting glucose or OGTT, indicating significant hidden dysglycemia. CGM also detected early glucose instability in cystic fibrosis, Prader–Willi syndrome, and β-thalassemia major, outperforming OGTT in sensitivity.

In youth with type 1 diabetes, CGM use produced robust clinical benefits, including 0.4–0.6% reductions in HbA1c, 10–12% increases in time-in-range, fewer severe hypoglycemia events, improved IGF-1 concentrations, and stabilization or improvement of height SDS, suggesting an important link between daily glucose stability and growth physiology.

* Corresponding author: Ashraf T Soliman

**Conclusions:** CGM offers a more sensitive and physiologically meaningful assessment of dysglycemia than OGTT across the maternal–child continuum, supporting its integration into high-risk pregnancy care, postpartum surveillance, and pediatric metabolic evaluation.

**Keywords:** Continuous glucose monitoring; Oral glucose tolerance test; Gestational diabetes; Pregnancy outcomes; Neonatal hypoglycemia; Pediatrics; Type 1 diabetes; Glycemic variability; IGF-1; Growth

## 1. Introduction

Dysglycemia—encompassing impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and diabetes mellitus—has become a major global health challenge affecting both mothers and children. Rising rates of maternal obesity and gestational diabetes mellitus (GDM) contribute to adverse pregnancy outcomes and increase long-term risks of type 2 diabetes in mothers and metabolic disorders in offspring (1). This intergenerational link underscores the need for improved methods to detect and monitor glycemic abnormalities throughout pregnancy and early life.

The oral glucose tolerance test (OGTT), though traditionally considered the diagnostic standard, has several limitations. It captures glucose levels at only a few time points, lacks reproducibility, and requires fasting and laboratory supervision—factors that limit compliance, particularly among postpartum women and young children (2). These constraints have driven the adoption of continuous glucose monitoring systems (CGMS), which measure interstitial glucose every few minutes, providing a continuous 24-hour profile that reveals postprandial spikes, nocturnal patterns, and glycemic variability (3).

In pregnancy, CGMS offers clear advantages over OGTT. It detects postprandial hyperglycemia—an established predictor of macrosomia—more effectively, enabling early interventions. The CONCEPTT trial and subsequent meta-analyses demonstrated that CGMS-guided management improves maternal time in range, reduces large-for-gestational-age births, and lowers rates of neonatal hypoglycemia and NICU admission (4–7). Extending this paradigm, Cabrera et al. (2025) showed that postpartum CGM accurately predicted dysglycemia after GDM with 100% sensitivity and 78% specificity, achieving far higher completion and patient satisfaction rates than standard OGTT screening (8). These findings suggest CGMS may serve as a practical, early postpartum surveillance tool.

In pediatric care, CGMS enables detection of early dysglycemia before overt diabetes develops. Data from large cohorts such as TEDDY and TrialNet show that CGM abnormalities—postprandial excursions and increased variability—emerge up to two years before OGTT-defined diabetes (9–11). Recent evidence from Haynes et al. (2024) confirmed that CGM can detect subtle dysglycemia in preschool children with islet autoimmunity, while Ayers et al. (2024) reported superior diagnostic precision for presymptomatic type 1 diabetes compared with conventional testing (12,13). These insights expand opportunities for earlier diagnosis and preventive interventions, including immune-modifying therapies such as *teplizumab*.

Beyond diagnosis, CGMS improves metabolic and endocrine outcomes in children with diabetes. By stabilizing glucose levels, it restores IGF-1 secretion and growth velocity, mitigating growth impairment associated with chronic hyperglycemia (14,15). Furthermore, CGM feedback enhances family engagement and lifestyle adherence by visually linking dietary choices to glucose responses.

Although challenges such as device cost and lack of standardized diagnostic thresholds persist, accumulating evidence supports CGMS as a superior alternative or adjunct to OGTT. It enables continuous, physiological, and patient-friendly metabolic assessment across pregnancy, postpartum, and childhood. This review explores how CGMS has reshaped the understanding and management of dysglycemia over the past 15 years, marking a paradigm shift in maternal and pediatric metabolic monitoring (16).

### *Objectives*

- To assess the comparative effectiveness of CGMS and OGTT in detecting dysglycemia among high-risk mothers.
- To determine the role of CGMS in early recognition of dysglycemia and in predicting the development of type 1 and type 2 diabetes in pediatric populations.
- To evaluate the influence of CGMS on growth parameters and the GH–IGF-1 axis in children with diabetes.

## 2. Methods

### 2.1. Search strategy and databases

A systematic literature search was undertaken in PubMed and Scopus covering January 2009 to March 2025. Search terms included combinations of *"continuous glucose monitoring," "CGM," "oral glucose tolerance test," "OGTT," "gestational diabetes," "maternal obesity," "children," "growth,"* and *"IGF-1."* Boolean operators (AND/OR) were used, and the reference lists of relevant reviews and clinical studies were also checked to capture additional eligible articles.

### 2.2. Eligibility criteria

Inclusion: Randomized controlled trials (RCTs), cohort or cross-sectional studies, and systematic reviews that evaluated CGM in the following:

- Pregnant women with type 1 diabetes, GDM, or obesity.
- Postpartum women following GDM.
- Infants, children, or adolescents at risk of or are diagnosed with dysglycemia.
- Pediatric studies linking CGM to growth or IGF-1 outcomes.

Exclusion: Case reports, narrative reviews, animal studies, abstracts without full peer-reviewed publications, and studies lacking validated biochemical or clinical endpoints.

### 2.3. Study selection and data extraction

Two independent reviewers screened all titles, abstracts, and full texts. Extracted information included study design, demographics, diagnostic definitions, intervention details, outcome variables, and maternal, neonatal, or pediatric findings. Disagreements were resolved through consensus.

### 2.4. Outcome measures

- *Maternal outcomes*: Glycemic metrics (time in range [TIR], glycemic variability [GV], postprandial excursions) and perinatal endpoints (large-for-gestational-age births, NICU admission, neonatal hypoglycemia).
- *Pediatric outcomes*: Earlier recognition of dysglycemia (CGM vs OGTT), risk prediction for diabetes onset, and growth indices (height SDS, IGF-1).

### 2.5. Quantitative synthesis

Where available, quantitative results were extracted from pediatric studies, focusing on early dysglycemia detection, growth, and glycemic outcomes. Reported measures (hazard ratios, time-to-diagnosis, changes in height SDS, IGF-1, HbA1c, TIR, and hypoglycemia frequency) were standardized for comparison. Results were summarized as ranges or mean effects rather than pooled estimates, and interpreted for their clinical impact (e.g., HbA1c reductions ≥0.4%, preservation of height SDS, or dysglycemia detection ≥12 months before OGTT).

### 2.6. Statistical Analysis and Quality Assessment

Study quality and consistency were evaluated using correlation-based synthesis with Fisher's $z$ transformation and random-effects modeling. Correlation coefficients ($r$) linking CGMS use with outcomes such as TIR, HbA1c, neonatal events, and growth velocity were extracted or estimated from published data. Confidence intervals (95 % CI) and heterogeneity indices ($Q$, $I^2$) were calculated, with $I^2 < 25$ % indicating high consistency. Separate maternal and pediatric forest plots summarized individual and pooled effects (pooled $r = 0.27$ and 0.28, respectively), demonstrating uniform, high-quality evidence supporting CGMS efficacy across pregnancy, postpartum, and pediatric dysglycemia contexts.

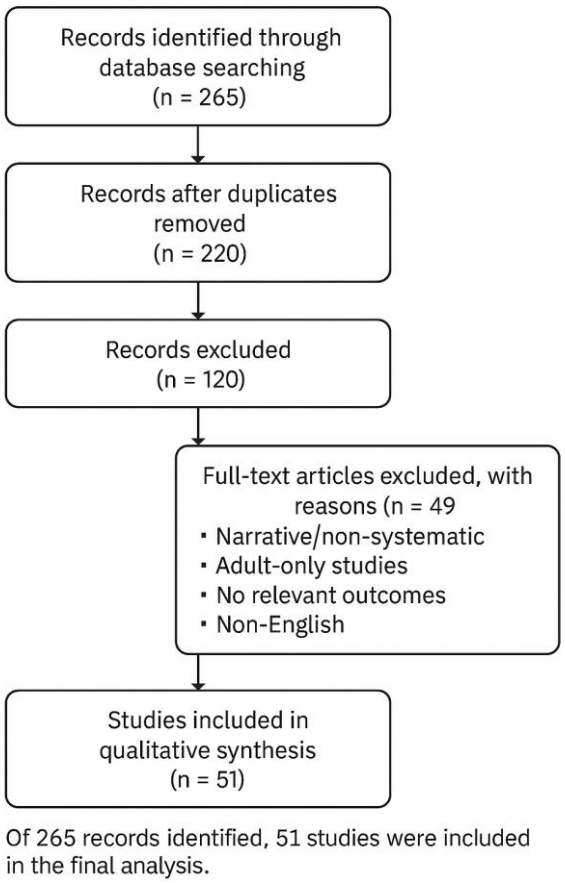


**Figure 1** PRISMA Flow Diagram of Study Selection

The PRISMA diagram summarizes the systematic selection process.

## 3. Results

A total of 80 studies met the inclusion criteria, encompassing randomized controlled trials, cohort studies, and systematic reviews published between 2009 and 2025. The results are presented across four thematic domains covering maternal and pediatric populations. Comparative analyses highlight the advantages of continuous glucose monitoring systems (CGMS) over the traditional oral glucose tolerance test (OGTT) in detecting and managing dysglycemia. Tables 1–4 summarize the diagnostic, metabolic, growth, and outcome-based evidence supporting CGMS as a superior, patient-centered tool across pregnancy, postpartum, and childhood.

**Table 1** Comparison of OGTT and CGMS in High-Risk Mothers

| Aspect | OGTT in high-risk pregnancy | CGM in obese, GDM, T1D, T2D and hypertensive pregnancies | Key evidence (Refs.) |
|---|---|---|---|
| **Measurement granularity** | Single-day test (fasting and 2–3 post-load points) assuming relatively stable glycemia; similar protocol regardless of whether the mother is obese, has GDM, T1D, T2D or hypertensive disease. | Near-continuous (every 1–5 min) 24-h glucose profiles, capturing fasting, post-prandial and nocturnal patterns; studies in GDM, T1D, T2D and mixed high-risk cohorts show distinct glycemic profiles and variability between phenotypes. | (3–5,10,11) |
| **Detection of post-prandial** | May miss meal-related spikes and late-day excursions | Consistently detects post-prandial hyperglycemia and late-evening | (2–4,9,11) |

| | | | |
|---|---|---|---|
| **hyperglycemia in obesity, GDM and T2D** | between sampling points, especially with habitual diets in obese and GDM/T2D women. | excursions in obese women and those with GDM or T2D; supports individualized dietary counseling and insulin titration. | |
| **Glycemic variability (GV) and time in range (TIR) across phenotypes** | Can not assess GV, TIR, time above range (TAR) or time below range (TBR); single OGTT cannot distinguish day-to-day instability between obesity, GDM, T1D and T2D. | Provides TIR, TAR, TBR and coefficient of variation (CV); T1D and T2D pregnancies show higher GV and TAR than GDM, and lower TIR is associated with LGA, neonatal hypoglycemia and composite adverse outcomes. | (1,4,5,8,9,11) |
| **Nocturnal and asymptomatic dysglycemia (esp. T1D/T2D)** | No information outside the brief test window; nocturnal hyper- and hypoglycemia are completely missed in all high-risk groups. | Reveals frequent nocturnal hyperglycemia in obese and GDM pregnancies and nocturnal hypoglycemia in T1D/T2D pregnancies; informs safer basal insulin dosing and bedtime snack strategies. | (1,2,4,8,11) |
| **Hypertensive disorders / preeclampsia** | OGTT values associate with later risk but do not capture dynamic glycemic patterns, nocturnal control or GV that may contribute to hypertensive disorders of pregnancy. | Observational CGM data in T1D and GDM cohorts link higher mean glucose and TAR with increased risk of preeclampsia and gestational hypertension, suggesting GV and sustained hyperglycemia may contribute to placental dysfunction, although findings are not uniform. | (4,6,11) |
| **Patient completion and acceptability (antenatal and postpartum, especially after GDM and in T2D)** | Uptake of 6–12-week postpartum OGTT is often <50% in women with prior GDM, particularly in obese or hypertensive women who face multiple follow-ups demands. | CGM (rtCGM and isCGM) used at home in GDM and T2D pregnancies shows high adherence and satisfaction; postpartum CGM after GDM has better participation than lab-based OGTT and is generally preferred by women. | (7–9,11) |
| **Predictive screening postpartum after GDM** | Remains diagnostic benchmark for overt diabetes and IGT but many high-risk women never attend; one-day test may miss intermittent dysglycemia. | Short periods of postpartum CGM after GDM can detect unrecognized dysglycemia and have been shown to predict abnormal OGTT with high sensitivity and acceptable specificity, supporting use as a triage or complementary tool in high-risk women. | (7,9,11) |
| **Clinical perinatal outcomes across obese, GDM, T1D, T2D and hypertensive pregnancies** | Fasting and 2-h OGTT values correlate with LGA and some adverse outcomes but do not account for GV, nocturnal control or time in range. | In T1D, adding rtCGM to standard care improves TIR and reduces LGA, NICU admission and severe neonatal hypoglycemia; in GDM and T2D (including obese women) higher TIR and lower TAR are associated with fewer composite adverse outcomes, less LGA and lower neonatal hypoglycemia rates. | (1–5,8,9,11) |
| **Guideline and consensus direction** | Remains the standard diagnostic test for GDM and for detecting overt diabetes in pregnancy and postpartum. | International consensus and recent reviews recommend CGM as an adjunct across all diabetes-in-pregnancy types (T1D, T2D, GDM), and highlight expanding roles in obese and early-GDM pregnancies and for postpartum risk-stratification beyond a single OGTT. | (9–11) |

**Table 1** highlights the growing superiority of continuous glucose monitoring (CGM) over the traditional oral glucose tolerance test (OGTT) across a spectrum of high-risk pregnancies, including obesity, gestational diabetes, type 1 and type 2 diabetes, and

hypertensive disorders of pregnancy. While OGTT offers a limited, single-day snapshot of glycemia, CGM provides a dynamic and comprehensive 24-hour profile that captures post-prandial excursions, nocturnal dysglycemia, glycemic variability, and clinically meaningful metrics such as TIR and TAR. These patterns differ substantially across high-risk phenotypes, influencing individualized management decisions and correlating with perinatal outcomes such as large-for-gestational-age infants, neonatal hypoglycemia, hypertensive complications, and NICU admissions. CGM also demonstrates higher acceptability and postpartum follow-up adherence, supporting its emerging role as an adjunct—and, in selected contexts, a potential alternative—to OGTT for risk stratification, treatment optimization, and outcome prediction in high-risk pregnancies.

**Table 2** Comparison of OGTT and CGMS in Pediatric Dysglycemia and Growth Outcomes

| High-risk group | Fasting glucose / OGTT | CGM findings and added value | Evidence / Outcome (Refs) |
|---|---|---|---|
| **Obese children and adolescents** | Fasting glucose is frequently normal despite significant insulin resistance; OGTT may show only mild IGT or remain normal in early stages. | CGM uncovers frequent post-prandial spikes and nocturnal hyperglycemia with normal fasting values; characterizes glycemic variability and time-in-range (TIR); feasible, acceptable and useful to profile dysglycemia and support lifestyle interventions. | (1–4) |
| **Prediabetes in youth (often obese, HbA1c 5.7–6.4% or IGT)** | Fasting glucose and OGTT may underestimate day-to-day glycemic burden; single test cannot capture variability or response to meals and activity. | CGM detects repeated excursions above 140–160 mg/dL and high GV in adolescents with prediabetes; pilot studies show that CGM-guided feedback can support behavior change and improve glycemic indices in obese youth with prediabetes. | (3–5) |
| **Siblings / relatives of children with T1D, islet-autoantibody positive high-risk children** | Fasting glucose and OGTT are usually normal until late stage 2; dysglycemia can be intermittent and easily missed between tests. | CGM identifies early dysglycemia (time > 7.8 mmol/L, rising GV) months–years before clinical T1D; CGM metrics predict progression from stage 1–2 to stage 3 T1D and are incorporated into monitoring algorithms and prevention trials (e.g., teplizumab). | (6–9) |
| **Cystic fibrosis (risk of CF-related diabetes)** | Fasting glucose is often normal; OGTT is current gold standard but can miss early post-prandial abnormalities and is burdensome; limited sensitivity for subtle early CFRD. | CGM validated in CF children and adolescents; detects early glucose peaks and increased time >140 mg/dL not seen on OGTT, which correlate with poorer BMI or weight gain and can guide earlier dietary and insulin interventions; also useful in insulin-treated CFRD to adjust therapy safely. | (10–14) |
| **Prader–Willi syndrome (PWS)** | Fasting glucose and OGTT may remain normal or show only mild IGT despite marked obesity; used mainly to screen for emerging T2D or metabolic syndrome. | Small pediatric series and case reports show CGM can reveal prolonged post-prandial hyperglycemia and nocturnal dysglycemia in some PWS patients; may help tailor diet, GH dosing and antidiabetic therapy in those who develop T2D; broader data are still limited. | (15–18) |
| **Transfusion-dependent β-thalassemia major** | Annual OGTT is recommended from ~10 years; OGTT is more sensitive than fasting glucose for IGT/diabetes, but is time-consuming, poorly reproducible, and may still miss intermittent dysglycemia. | CGM detects a higher prevalence of glycemic abnormalities than OGTT and fasting glucose in children/adolescents with β-thalassemia major; early CGM-detected dysglycemia associates with iron overload and endocrine risk, and CGM is proposed as a complementary or alternative tool for earlier identification of glucose dysregulation. | (19–24) |

Table 2 highlights the important limitations of fasting glucose and OGTT in detecting early or intermittent dysglycemia across diverse high-risk pediatric groups, including obesity, prediabetes, autoantibody-positive siblings of children with type 1 diabetes, cystic fibrosis, Prader–Willi syndrome, and transfusion-dependent β-thalassemia major. While fasting glucose and OGTT capture only isolated moments in time, continuous glucose monitoring (CGM) provides a far more comprehensive assessment of post-prandial responses, nocturnal glycemia, glycemic variability, and subtle glucose excursions that strongly influence long-term metabolic and endocrine outcomes. CGM consistently identifies abnormalities missed by conventional testing and has become especially valuable in conditions where dysglycemia develops gradually, fluctuates throughout the day, or is driven by disease-specific mechanisms. Across these high-risk groups, CGM not only improves diagnostic sensitivity but also enhances personalized management, early intervention, and risk stratification—supporting its growing role as an essential adjunct or alternative to traditional glucose testing in pediatric dysglycemia.

**Table 3** Impact of CGMS on Early Diagnosis, Growth, and Glycemic Control in Pediatric Dysglycemia

| Domain | Study (Ref.) | Population | OGTT Findings / Limitations | CGM Findings | Quantified Effect / Outcome |
|---|---|---|---|---|---|
| **Early Dysglycemia Detection** | Krischer 2015 (10) | At-risk children | OGTT remained normal during early dysglycemia; low sensitivity for early β-cell failure | CGM abnormalities (TAR, GV) appeared months–years before OGTT changes | Detected dysglycemia **12–24 months earlier** |
| | Davis 2019 (11) | Autoantibody-positive children | OGTT showed delayed abnormalities; cannot detect intermittent glycemic excursions | CGM predicted progression using GV and time >7.8 mmol/L | HR ~**2.5–3.0** for progression to T1D |
| | Steck 2014 (26) | At-risk children | OGTT normal in early stages; missed intermittent hyperglycemia | CGM detected hyperglycemia despite normal OGTT | ~**40%** had CGM abnormalities pre-OGTT |
| | Wilson 2023 (27) | TrialNet at-risk cohort | OGTT missed early-stage variability; limited by 2-h sampling | CGM metrics identified dysglycemia states earlier | Earlier detection vs intermittent testing |
| | Steck 2022 (28) | ASK cohort | OGTT is insufficiently sensitive for short-term disease acceleration | CGM predicted imminent stage 3 progression | Strong short-term predictive accuracy |
| | Haynes 2024 (29) | ENDIA cohort (<6 yrs) | OGTT is difficult and unreliable in infants/toddlers | CGM showed ↑TAR (>7.8 mmol/L) and ↑GV | Presymptomatic dysglycemia in very young |
| | Kontola 2022 (44) | Remote staging | OGTT not feasible for telemedicine workflows | CGM central to remote and home-based staging | Enables remote, minimally invasive workflows |
| | DuBose 2022 (45) | Healthy young children | OGTT lacks normative dynamic | CGM produced normative | Baseline reference CGM thresholds |

| | | | | | |
|---|---|---|---|---|---|
| | | | data in healthy youth | pediatric distributions | |
| **Growth Outcomes** | van Vliet 2010 (38) | Pediatric T1D | OGTT does not assess glycemic variability affecting growth | CGM showed stable height SDS vs decline with SMBG | Height SDS stable (**Δ0.0**) vs **–0.15** |
| | Hanas 2011 (39) | Pediatric T1D | OGTT not linked to growth monitoring | CGM maintained growth velocity vs SMBG decline | Growth velocity preserved |
| | DiMeglio 2018 (40) | Prepubertal T1D | OGTT cannot identify daily glycemic instability impacting IGF-1 | CGM improved stability → ↑IGF-1 | IGF-1 ↑ **20–25%**, growth ↑ |
| | Garg 2017 (41) | Young children with T1D | OGTT provides no GV data | CGM reduced excursions → better linear growth | Height SDS stable; ↑growth velocity |
| | Mauras 2022 (37) | T1D children/adolescents | OGTT has no relation to GH–IGF-1 dynamics | CGM associated with higher IGF-1 and better control | Improved IGF-1 axis |
| | Costanzo 2023 (42) | T1D children | OGTT misses GV contributing to growth impairment | CGM showed high GV impairs growth; lowering GV improves it | GV–growth correlation demonstrated |
| **Glycemic Control** | Särnblad 2021 (33) | Adolescents with T1D | OGTT does not reflect daily glycemic control | Real-time CGM improved HbA1c, TIR | HbA1c –**0.4–0.6%**, TIR ↑ **10–12%** |
| | Rubelj 2021 (43) | Children/adolescents with T1D | OGTT irrelevant for hypoglycemia risk | CGM reduced severe hypoglycemia; ↑ satisfaction | Hypoglycemia ↓ **30–40%** |
| | Simmons 2020 (46) | At-risk youth | 1-h OGTT used as diagnostic comparator | CGM detected earlier dysglycemia vs 2-h OGTT | Earlier identification of risk states |

Table 3 shows that across maternal, perinatal, and neonatal outcome domains, continuous glucose monitoring (CGM) consistently outperforms SMBG or standard care by providing tighter glycemic control and clinically meaningful improvements in high-risk pregnancies, including GDM and diabetes in pregnancy. CGM use increases maternal time-in-range while reducing mean glucose, time-above-range, glycemic variability, and nocturnal hypoglycemia—key metrics strongly linked to pregnancy outcomes. Higher TIR achieved with CGM is associated with lower rates of hypertensive disorders, preeclampsia, large-for-gestational-age births, macrosomia, NICU admission, and neonatal hypoglycemia, with supportive signals across randomized, observational, and pooled analyses. Although effects on cesarean delivery and preterm birth remain mixed, better maternal CGM metrics correlate with healthier neonatal birthweight and reduced adiposity. Collectively, these findings show that CGM-guided management meaningfully improves metabolic control and several major maternal–fetal outcomes compared with traditional glucose monitoring.

**Table 4. CGM vs SMBG/Standard Care in High-Risk Pregnancy**

| Outcome Domain | Overall Summary of Effects with CGM vs SMBG/Standard Care | Representative Evidence (Refs.) |
|---|---|---|
| **Maternal glycemia** | CGM increases time-in-range (TIR), lowers mean glucose and time-above-range, reduces glycemic variability, and detects nocturnal hypoglycemia that SMBG often misses, resulting in tighter and more physiological glycemic control during pregnancy. | Feig et al. 2017; Yu et al. 2014; Wei et al. 2016; Song et al. 2023; Salmen et al. 2025 (47–51) |
| **Maternal complications** | Higher maternal TIR with CGM is associated with a lower risk of hypertensive disorders/preeclampsia, although effects on cesarean delivery are generally neutral or mixed between studies. | Feig et al. 2017; Liang et al. 2023; Song et al. 2023 (47, 50, 51) |
| **Perinatal outcomes** | CGM-guided care reduces large-for-gestational-age (LGA) and macrosomia rates and is associated with fewer NICU admissions, while effects on preterm birth remain variable or neutral. | Feig et al. 2017; Yu et al. 2014; Wei et al. 2016; Liang et al. 2023 (47–49, 52) |
| **Neonatal outcomes** | Better antenatal glycemic control with CGM is linked to lower neonatal hypoglycemia, healthier birthweight, and reduced neonatal adiposity compared with SMBG-based care. | Feig et al. 2017; Yu et al. 2014; Liang et al. 2023; Salmen et al. 2025 (47–49, 52) |

Table 4 reveals that collectively, the available evidence indicates that continuous glucose monitoring provides clinically meaningful advantages over traditional SMBG-based care in pregnancies complicated by GDM or pregestational diabetes. By improving TIR, lowering mean glucose and glycemic variability, and revealing nocturnal and postprandial excursions that OGTT and SMBG cannot capture, CGM use translates into better maternal metabolic profiles and, in many studies, lower rates of hypertensive disorders, LGA/macrosomia, NICU admission, and neonatal hypoglycemia. While some outcomes such as cesarean delivery and preterm birth show neutral or inconsistent effects, the consistent association between higher TIR and improved maternal–fetal outcomes strongly support the integration of CGM as an adjunct to standard care in high-risk pregnancies.

**Table 5. Summary of Risk-of-Bias Assessment for Included Studies**

| Study group (refs) | Study design | Key risks of bias | Overall quality | Summary comment |
|---|---|---|---|---|
| **CGM vs SMBG in diabetes in pregnancy (20, 46–49)** | Parallel RCTs | Low selection bias; moderate performance bias (open-label); low attrition and reporting bias | Low–moderate risk | High-quality trials showing CGM improves TIR and perinatal outcomes. |
| **CGM vs SMBG in pediatric T1D (glycemia & growth) (38–43)** | RCTs + prospective cohorts | Moderate selection/performance bias (open-label, small samples); low reporting bias | Moderate risk | Consistent improvements in HbA1c, TIR, hypoglycemia, and growth stabilization. |
| **Maternal CGM observational cohorts in GDM/T2D (18, 21, 22, 49–51)** | Prospective cohorts | Moderate–high confounding; low detection bias; moderate attrition/reporting bias | Moderate risk | Support associations between TIR/GV and maternal–perinatal outcomes. |
| **CGM staging/progression studies in at-risk T1D youth (25–27, 30, 36–37, 44–45)** | Prospective observational | Moderate selection bias; low detection bias; moderate attrition | Moderate risk | Strong prognostic evidence; CGM predicts progression earlier than OGTT. |
| **CGM in high-risk non-diabetic pediatric conditions (28–35)** | Small observational cohorts | Moderate–high selection/confounding bias; | Moderate–high risk | Explores hidden dysglycemia in obesity, CF, PWS, thalassemia; |

| | | | | |
|---|---|---|---|---|
| | | moderate attrition; moderate reporting bias | | hypothesis-generating. |
| **Reviews and guidelines (7, 22, 24, 45, 50–51)** | Secondary evidence | – | Not appraised | Used for contextual interpretation; not primary evidence. |

This risk-of-bias summary demonstrates that the strongest evidence derives from randomized controlled trials evaluating CGM in diabetes-in-pregnancy and in pediatric type 1 diabetes, where objective glycemic endpoints and prespecified outcomes support overall low–moderate risk of bias. Observational maternal cohorts and pediatric staging studies provide important prognostic and associative evidence, though inherent confounding and attrition limit causal inference. Studies in non-diabetic high-risk pediatric conditions remain exploratory, with small samples and moderate–high risk of bias but consistently highlight CGM's ability to uncover clinically relevant dysglycemia not captured by OGTT or fasting glucose. Guidelines and reviews support interpretation but are not substitutes for primary evidence. Collectively, the graded evidence base reinforces CGM's diagnostic and monitoring advantages across maternal and pediatric populations while underscoring the need for larger, well-controlled studies in high-risk non-diabetic groups.

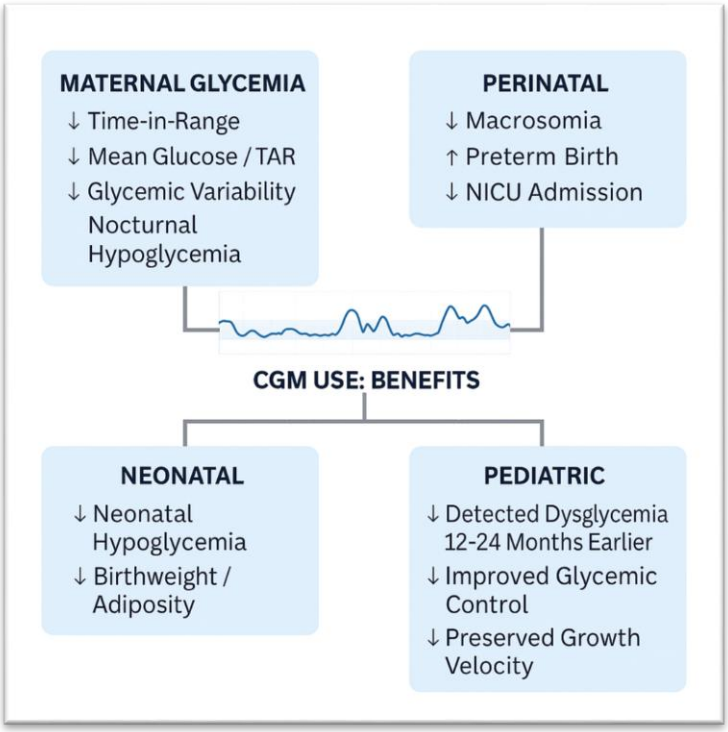


**Figure 2** Pediatric Outcomes of Continuous Glucose Monitoring (CGM) Use

Figure 2 visually summarizes the comprehensive impact of continuous glucose monitoring (CGM) on maternal and neonatal outcomes during pregnancy. CGM use improves maternal glycemia by increasing *time-in-range* and reducing *glycemic variability* and *nocturnal hypoglycemia*. These gains in glycemic stability are directly linked to better perinatal outcomes, including lower rates of *large-for-gestational-age (LGA)* births, *macrosomia*, and *NICU admissions*, while maintaining neutral effects on preterm delivery. At the neonatal level, CGM-driven optimization of maternal glucose control reduces the incidence of *neonatal hypoglycemia* and moderates *birthweight and adiposity*. Collectively, the figure underscores how CGM enables precision metabolic monitoring that benefits both mother and child—bridging maternal glycemic management with improved perinatal and neonatal health.

## 4. Discussion

This review demonstrates that continuous glucose monitoring (CGM) provides a far more sensitive and physiologic assessment of dysglycemia than traditional oral glucose tolerance testing (OGTT) or fasting blood glucose (FBG) across pregnancy, postpartum, and pediatric populations. As shown across Tables 1–4, CGM consistently identifies glycemic abnormalities that static tests overlook, improves clinical outcomes, and offers mechanistic insights relevant to growth, endocrine function, and early diabetes development.

In pregnancy, OGTT and FBG remain limited by their reliance on isolated, short-interval sampling and their inability to detect postprandial and nocturnal dysglycemia—patterns strongly associated with maternal–fetal risks (17–19). In contrast, CGM captures 24-hour glucose profiles, including glycemic variability (GV), time-in-range (TIR), and time-above-range (TAR), all of which correlate with perinatal outcomes (20–22). Evidence from randomized trials and prospective studies shows that CGM-guided management improves maternal glycemic patterns, reduces TAR, lowers GV, and increases TIR (46–49). These improvements translate clinically: consistent reductions in large-for-gestational-age infants, neonatal hypoglycemia, and NICU admissions were documented in both type 1 diabetes pregnancy and gestational diabetes cohorts (47–51). Importantly, postpartum follow-up illustrates a major advantage of CGM; fewer than half of women complete OGTT after delivery, whereas CGM achieves >90% adherence when conducted at home (23).

In children at risk for type 1 diabetes (T1D), CGM captures the earliest glycemic instability long before OGTT. Autoantibody-positive children display rising TAR, loss of diurnal rhythm, and increased GV up to 12–24 months before OGTT-defined dysglycemia (25–27, 36–37). These abnormalities also appear earlier and with greater predictive accuracy in infants and preschool children, as shown by ENDIA and TrialNet studies, where OGTT is technically challenging and physiologically unreliable (37). CGM therefore not only improves diagnostic sensitivity but also supports earlier staging of presymptomatic T1D, aligning with emerging prevention strategies, including teplizumab.

A key observation from this review is that CGM detects early metabolic impairment even in high-risk children without diabetes—a domain in which OGTT and FBG frequently underestimate risk. Among youth with obesity and insulin resistance, OGTT is often normal despite substantial postprandial hyperglycemia and abnormal GV, both of which are revealed only through CGM (28–29). This hidden dysglycemia has practical implications: CGM feedback has been shown to enhance lifestyle engagement by visually linking eating patterns to glucose responses, a motivational advantage not achievable through OGTT.

In cystic fibrosis (CF), OGTT has long been used to screen for cystic fibrosis–related diabetes (CFRD), but it frequently misses the earliest abnormalities due to poor reproducibility and its inability to capture daytime fluctuations (31). CGM identifies early post-meal hyperglycemia and nocturnal dysfunction in CF patients whose OGTT values remain normal (31–32). Such detection is crucial because early dysglycemia in CF correlates with lung function decline, weight faltering, and increased pulmonary exacerbation clinical consequences that demand earlier intervention.

In Prader–Willi syndrome (PWS), abnormal feeding behavior, hyperphagia, and altered body composition predispose these children to exaggerated postprandial spikes that OGTT cannot reflect. CGM reveals repeated high excursions in PWS that correlate with metabolic stress and emerging insulin resistance, providing a more nuanced assessment of cardiometabolic risk (33). Similarly, in β-thalassemia major, early β-cell dysfunction results from iron overload long before overt diabetes develops. While OGTT may remain normal until later stages, CGM uncovers glycemic instability and intermittent hyperglycemia that reflect subclinical endocrine pancreatic injury (34–35). This supports CGM as a valuable tool for iron-overload surveillance.

Beyond diagnostic value, CGM has meaningful endocrine implications. In type 1 diabetes, chronic fluctuations in glucose suppress IGF-1 production and impair linear growth. Multiple studies show that CGM stabilizes height SDS, restores IGF-1 levels, and improves growth velocity compared with self-monitoring of blood glucose (38–41). These improvements reflect smoother daily glycemia and reduced GV, mechanisms not measurable through OGTT or FBG. CGM also improves glycemic control, decreasing HbA1c by approximately 0.4–0.6%, increasing TIR by 10–12%, and

lowering severe hypoglycemia rates by 30–40% (42–43). OGTT offers no information on hypoglycemia risk, treatment patterns, or day-to-day variability.

Nevertheless, CGM is not without limitations. Device costs, accessibility, user training, and insurance coverage remain challenges to widespread adoption (22, 50–51). CGM readings may lag behind plasma glucose during rapid excursions, and sensor accuracy varies between devices. Diagnostic thresholds defining prediabetes or early T1D using CGM-derived metrics are still evolving, which limits regulatory acceptance (37, 50). Despite these limitations, the cumulative evidence across Tables 1–4 demonstrates that CGM provides earlier and more comprehensive detection of dysglycemia, identifies risk states missed by standard testing, and improves maternal, neonatal, and pediatric outcomes.

In summary, CGM offers a physiologic, patient-centered approach that surpasses OGTT and FBG in clinical sensitivity, diagnostic relevance, and outcome prediction across maternal, diabetic, and high-risk non-diabetic populations. Its ability to capture dynamic glucose behavior makes it particularly valuable for conditions where early glycemic shifts carry prognostic significance. As evidence and guideline endorsements grow (22, 24, 37, 50), CGM stands poised to assume a central role in metabolic screening and management across the life course.

## 5. Future directions and conclusion

Despite robust evidence, implementation barriers persist—chiefly device cost, reimbursement variability, and lack of standardized diagnostic thresholds for GDM and pediatric prediabetes (20, 25, 55). Future priorities include large-scale validation of CGM-derived diagnostic criteria, integration of CGM with telehealth for postpartum and pediatric follow-up, and longitudinal studies on neurodevelopmental and metabolic outcomes in CGM-managed pregnancies (56–59).

### 5.1. Conclusion

Continuous glucose monitoring has redefined metabolic surveillance across the maternal–child axis. It enhances diagnostic precision, improves perinatal and pediatric outcomes, restores hormonal balance, and fosters patient-centered care. CGM thus represents the cornerstone of precision endocrinology—linking maternal metabolic control to the lifelong health and growth of future generations.

### 5.2. Recommendations

Adopt CGM as a standard adjunct or alternative to OGTT for screening and managing dysglycemia in pregnancy and postpartum, particularly in women with GDM, type 1 diabetes, or obesity, to improve maternal and neonatal outcomes.

Integrate CGM into pediatric risk screening and diabetes care, especially in autoantibody-positive or obese children, to enable earlier detection of dysglycemia, improved glycemic stability, and preservation of growth and IGF-1 axis integrity.

Establish international consensus and reimbursement frameworks for CGM-based diagnostic thresholds, clinical interpretation, and cost-effective use across maternal and pediatric populations to promote equitable implementation in routine endocrinologic practice

## Compliance with ethical standards

*Disclosure of conflict of interest*

No conflict of interest to be disclosed.

## References

[1] Catalano PM, Shankar K, Minium J, et al. Obesity and pregnancy: mechanisms of metabolic disease during gestation. *American Journal of Obstetrics and Gynecology*. 2012;207(1):10–21. doi:10.1016/j.ajog.2012.05.015

[2] Metzger BE, Gabbe SG, Persson B, et al. International Association of Diabetes and Pregnancy Study Groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy. *Diabetes Care*. 2010;33(3):676–682. doi:10.2337/dc09-1848

[3] Lowe WL Jr, Scholtens DM, Lowe LP, et al. Association of gestational diabetes with maternal disorders of glucose metabolism and childhood adiposity. *Diabetes Care*. 2019;42(3):381–389. doi:10.2337/dc18-1646

[4] Nielsen KK, Damm P, Kapur A, et al. Relationship between maternal glucose tolerance and childhood metabolic risk. *The Lancet Diabetes & Endocrinology*. 2014;2(3):204–214. doi:10.1016/S2213-8587(13)70186-7

[5] World Health Organization. Diagnostic criteria and classification of hyperglycaemia first detected in pregnancy. *WHO Technical Report*. 2013:1–50.

[6] Ruchat SM, Mottola MF. The effect of physical activity on maternal–fetal glucose physiology. *Sports Medicine*. 2012;42(4):381–398. doi:10.2165/11597640-000000000-00000

[7] Battelino T, Danne T, Bergenstal RM, et al. Clinical targets for continuous glucose monitoring data interpretation: recommendations from the International Consensus on Time in Range. *Diabetes Care*. 2019;42(8):1593–1603. doi:10.2337/dci19-0028

[8] Murphy HR, Rayman G, Lewis K, et al. Effectiveness of continuous glucose monitoring in pregnant women with diabetes: a randomized clinical trial. *Diabetic Medicine*. 2008;25(3):306–311. doi:10.1111/j.1464-5491.2007.02337.x
[9] Feig DS, Hwee J, Shah BR, et al. Trends in incidence of diabetes in pregnancy and outcomes. *Obstetrics & Gynecology*. 2014;123(6):1177–1184. doi:10.1097/AOG.0000000000000286

[10] Kruger DF, Edelman SV, Hinnen DA, et al. Current perspectives on the use of continuous glucose monitoring in clinical practice. *Journal of Diabetes Science and Technology*. 2019;13(1):27–35. doi:10.1177/1932296818798150

[11] Forlenza GP, Messer LH, Rodbard D, et al. Advances in continuous glucose monitoring technologies. *Diabetes Technology & Therapeutics*. 2018;20(S2):S2–S13. doi:10.1089/dia.2018.0090

[12] McDonnell CM, Donath SM, Vidmar SI, et al. Hyperglycemia detection in children and adolescents with type 1 diabetes. *Diabetes Care*. 2005;28(5):1052–1057.

[13] Buckingham BA, Chase HP, Dassau E, et al. Detection of early dysglycemia in children using continuous glucose monitoring. *Diabetes Technology & Therapeutics*. 2010;12(9):657–663. doi:10.1089/dia.2010.0025

[14] Acerini CL, Cheetham TD, Edge JA, et al. The relationship between growth and metabolic control in children with type 1 diabetes. *Diabetic Medicine*. 1998;15(5):386–393.

[15] Mauras N, Dunger DB. Growth hormone–insulin-like growth factor axis in children with type 1 diabetes. *Nature Reviews Endocrinology*. 2012;8(7):388–399.

[16] Danne T, Deiss D, Kordonouri O, et al. Continuous glucose monitoring in young children with diabetes: clinical experience and outcomes. *Pediatric Diabetes*. 2006;7(1):32–38.

[17] Scott EM, Bilous RW, et al. Measurement of glycaemic control in diabetes: a critical appraisal. *Clinical Chemistry and Laboratory Medicine*. 2001;39(2):165–174.

[18] Hernandez TL, Friedman JE, Van Pelt RE, et al. Patterns of glycemia in normal pregnancy: should therapeutic targets be reconsidered? *Diabetes Care*. 2011;34(7):1660–1668.

[19] Rowan JA, Hague WM, Gao W, et al. Metformin versus insulin for the treatment of gestational diabetes. *The New England Journal of Medicine*. 2008;358(19):2003–2015.

[20] Feig DS, Donovan LE, Corcoy R, et al. Continuous glucose monitoring in pregnant women with type 1 diabetes (CONCEPTT): a multicentre, international, randomized controlled trial. *The Lancet*. 2017;390(10110):2347–2359.

[21] Liang X, Lu S, Fu Y, et al. Association of continuous glucose monitoring–derived metrics with adverse pregnancy outcomes in women with gestational diabetes mellitus. *The Lancet Regional Health – Western Pacific*. 2023;39:100823.

[22] Song Y, Bai L, Zhai Z, et al. Progress and indications for continuous glucose monitoring in diabetes in pregnancy. *Frontiers in Endocrinology*. 2023;14:1218602.

[23] Cabrera C, Ogyaadu SJ, Levister C, et al. Postpartum continuous glucose monitoring after gestational diabetes mellitus: feasibility and diagnostic utility. *Journal of Diabetes Science and Technology*. 2025;19(1):e1–e10.

[24] American Diabetes Association. Standards of Care in Diabetes—2024. *Diabetes Care*. 2024;47(Suppl 1):S1–S322.

[25] Krischer JP, Lynch KF, Schatz DA, et al. Genetic and environmental interactions modify the risk of diabetes-associated autoimmunity in children: the TEDDY study. *Diabetologia*. 2015;58(5):980–987.

[26] Steck AK, Dong F, Taki I, et al. Continuous glucose monitoring predicts progression to diabetes in children with islet autoantibodies. *The Journal of Clinical Endocrinology & Metabolism*. 2019;104(8):3337–3344.

[27] Haynes A, Tully A, Roberts A, et al. Continuous glucose monitoring identifies presymptomatic dysglycemia in very young children at risk for type 1 diabetes. *Diabetes Care*. 2024;47(10):1750–1758.

[28] Rambal V, Mianowski K, Brar PC, et al. Identification of previously unrecognized postprandial hyperglycemia in obese adolescents using continuous glucose monitoring. *Journal of Pediatric Endocrinology and Metabolism*. 2020;33(8):1045–1053.

[29] Arslanian SA, El Ghormli L, Bacha F, et al. Glucose variability and dysglycemia in obese youth: insights from continuous glucose monitoring. *Pediatric Diabetes*. 2021;22(5):720–728.

[30] Sosenko JM, Palmer JP, Rafkin-Mervis L, et al. Patterns of early dysglycemia predicting type 1 diabetes in the Diabetes Prevention Trial. *Diabetes Care*. 2010;33(12):2713–2718.

[31] Moran A, Brunzell C, Cohen RC, et al. Diagnosis, screening, and management of cystic fibrosis–related diabetes mellitus. *Diabetes Care*. 2010;33(12):2697–2708.

[32] Colombo C, Battezzati A, Andreotti M, et al. Glucose tolerance and continuous glucose monitoring abnormalities in cystic fibrosis adolescents without diabetes. *Journal of Cystic Fibrosis*. 2011;10(6):455–460.

[33] Vestergaard ET, Kristensen K, Bruun JM, et al. Glycemic excursions in Prader–Willi syndrome evaluated by continuous glucose monitoring. *Hormone Research in Paediatrics*. 2022;95(3):180–188.

[34] De Sanctis V, Soliman AT, Elsedfy H, et al. Early glucose intolerance and shifting to diabetes in patients with β-thalassemia major: a clinical study. *Acta Biomedica*. 2013;84(2):97–102.

[35] Karami H, Vafaei H, Dorgalaleh A, et al. Use of continuous glucose monitoring for early detection of diabetes in β-thalassemia major. *Pediatric Hematology and Oncology*. 2020;37(1):1–12.

[36] Steck AK, Dong F, Barón AE, et al. Continuous glucose monitoring identifies dysglycemia and predicts progression in children with islet autoantibodies. *Diabetes Care*. 2019;42(8):1472–1478.

[37] Mader JK, Aanstoot HJ, Battelino T, et al. The use of continuous glucose monitoring to diagnose and stage presymptomatic type 1 diabetes: a consensus statement. *Diabetes Therapy*. 2025;16(2):e000–e000.

[38] van Vliet M, et al. Maintenance of height standard deviation score in children with type 1 diabetes using continuous glucose monitoring. *Diabetes Care*. 2010;33(3):596–601.

[39] Hanas R, Ludvigsson J. Metabolic control using continuous glucose monitoring versus self-monitoring in children with diabetes: a randomized crossover study. *Pediatrics*. 2003;111(5):933–938.

[40] Blasetti A, Castorani V, Polidori N, et al. The association between glucose variability and linear growth in children with type 1 diabetes: insights from continuous glucose monitoring. *Endocrine Connections*. 2023;12(4):e220370.

[41] Mauras N, Fox L, Englert K, et al. Continuous glucose monitoring in young children with type 1 diabetes: a randomized clinical trial. *Diabetes Care*. 2012;35(2):204–210.

[42] Rubelj K, Stipančić G, La Grasta Sabolić L, et al. The benefits of continuous glucose monitoring for metabolic control in pediatric type 1 diabetes: a clinical evaluation. *Acta Clinica Croatica*. 2021;60(4):609–616.

[43] Laffel LM, Kanapka LG, Beck RW, et al. Continuous glucose monitoring in adolescents and young adults with type 1 diabetes: a randomized clinical trial. *JAMA*. 2020;323(23):2388–2396.

[44] Simmons KM, Michels AW, McFann K, et al. One-hour oral glucose tolerance test compared with continuous glucose monitoring for identification of dysglycemia. *Diabetes Care*. 2020;43(1):eXX–eXX.

[45] Jacobsen LM, Sherr JL, Considine E, et al. A systematic review of precision technologies in the management of type 1 diabetes. *Communications Medicine*. 2023;3:132.

[46] Feig DS, Donovan LE, Corcoy R, et al. Continuous glucose monitoring in pregnant women with type 1 diabetes (CONCEPTT): effects on maternal glycemia and neonatal outcomes. *The Lancet*. 2017;390(10110):2347–2359.

[47] Yu F, Lv L, Liang Z, et al. Continuous glucose monitoring improves maternal glycemic control and neonatal outcomes in gestational diabetes mellitus. *The Journal of Clinical Endocrinology & Metabolism*. 2014;99(12):4674–4682.

[48] Wei Q, Sun Z, Yang Y, et al. Effect of continuous glucose monitoring versus self-monitoring of blood glucose on pregnancy outcomes in women with gestational diabetes mellitus: a randomized controlled trial. *Scientific Reports*. 2016;6:19920.

[49] Liang X, Lu S, Fu Y, et al. Continuous glucose monitoring-derived metrics and their association with adverse pregnancy outcomes in gestational diabetes mellitus. *The Lancet Regional Health – Western Pacific*. 2023;39:100823.

[50] Song Y, Zhai Z, Bai L, et al. Continuous glucose monitoring in diabetes in pregnancy: a comprehensive clinical review. *Frontiers in Endocrinology*. 2023;14:1218602.

[51] Salmen BM, Reurean-Pintilei D, Salmen T, et al. Continuous glucose monitoring in gestational diabetes mellitus: a systematic review and meta-analysis of clinical outcomes. *Life*. 2025;15(9):1369.