

Review

Comparing Pregnancy Outcomes in Fetal Growth Restriction and Overgrowth in Women with Type 1 Diabetes: A Narrative Review

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Abstract

Background: Several scenarios of abnormal fetal growth develop during pregnancy in women with pregestational diabetes. A trend towards a higher incidence of large-for-gestational-age (LGA) births among women with type 1 diabetes (T1D) is observed worldwide despite novel approaches to maternal glycaemic control. Fetal growth restriction (FGR) is commonly recognised as a severe condition. However, the risk of adverse outcomes in LGA fetuses generally remains underestimated. **Methods:** We conducted a comparative analysis of pregnancy outcomes across different birthweight categories in pregnancies with T1D based on published data (2001–2026). **Results:** Interactions between disorders of early placentation, impaired nutrient transfer, fetal overnutrition, and genetic background result in alterations in birthweight and disproportionate fetal adipose tissue deposition. Both FGR and macrosomia are associated with increased risks of adverse perinatal outcomes. Although stillbirth occurs at both extremes of fetal growth, term LGA fetuses may remain particularly vulnerable because routine surveillance methods, including arterial Doppler assessment, fail to identify compromised fetuses with excessive growth. Accelerated fetal growth in T1D pregnancies, when placentation is initially impaired, may cause growth-restricted fetuses to appear appropriate for gestational age (AGA). Routine assessment may classify these vulnerable fetuses as being at low risk of perinatal complications. Disproportionate growth, even among infants classified as AGA, is associated with shoulder dystocia, birth trauma, and operative delivery. Neonatal hypoglycaemia and respiratory disorders remain frequent complications across the spectrum of abnormal fetal growth. **Conclusions:** Increased glucose variability may explain differences in placental and fetal growth trajectories and the high rate of pregnancy complications in patients with target glycated haemoglobin levels and routine capillary glucose monitoring results. Fetal overgrowth reflects impaired intrauterine development and should be considered a high-risk condition requiring careful consideration of the timing and mode of delivery.



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This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.**Keywords:** diabetes mellitus; type 1; pregnancy complications; fetal macrosomia; birth weight; fetal growth restriction; fetal growth; stillbirth

1. Introduction

Pregnancy complicated by pregestational diabetes (PGD) is often associated with accelerated fetal growth. Although major advancements in maternal glycaemic control during the preconception period and early pregnancy have reduced the incidence of

congenital malformations, early pregnancy loss, and stillbirths since the 1970s, pregnant women with type 1 diabetes (T1D) remain at substantially increased risk for maternal and neonatal complications [1–5].

Blood glucose control before and during conception, a comparative reduction in the prevalence of diabetic microangiopathies, and early pregnancy care improvements have led to a decrease in the prevalence of fetal growth restriction (FGR) originally caused by impaired placentation, although it remains a significant issue. Moreover, fetal overnutrition and growth acceleration in later gestation, associated with maternal hyperglycaemia and increased glucose variability, can shift growth-restricted fetuses toward an appropriate-for-gestational-age (AGA) weight trajectory. Desoye et al. (2023) emphasise that abnormal fetal growth in T1D pregnancies can develop with different scenarios, including normal or poor placentation accompanied by near-normal glucose levels or maternal hyperglycaemia during pregnancy, resulting in infants showing overgrowth and excessive adiposity even despite a normal birthweight [6].

The use of novel diabetes technologies, such as continuous glucose monitoring (CGM) and automated insulin delivery, is encouraged in women with T1D. It is especially recommended during the preconception period and pregnancy, as it facilitates improved glycaemic control and helps achieve near-normal glycaemic targets [7–9]. As CGM systems become more widely used, research employing this technology is yielding new data on the association between glycaemic variability and pregnancy complications, as well as on opportunities for their prevention [1,7,10–15]. Despite the increasingly widespread use of novel technologies, the incidence of fetal overgrowth has not decreased over recent decades. A large population-based study conducted in Sweden by Sandin et al. (2025) analyzed temporal trends in neonatal anthropometric outcomes among offspring of women with T1D ($n = 6485$), considering the spectrum of birthweight, gestational age-adjusted birthweight (standardised against national growth charts), and the ponderal index. Across the investigated time periods of 1998–2007 and 2008–2016, no substantial changes were observed in the distributions of birthweight, and the proportions of infants at both growth extremes (small for gestational age, SGA, below the 10th percentile; and large for gestational age, LGA, above the 90th percentile) remained similar irrespective of first-trimester maternal body mass index, with 60–62% of infants born LGA [16]. Similar results have been obtained in other population-based cohort studies. A study conducted in Spain by López-de-Andrés et al. (2020) identified consistently high rates of fetal macrosomia (birthweight over 4500 g) among pregnant women with T1D between 2009 and 2015 ($n = 5561$) [17]. Earlier, a study carried out in Scotland by Mackin et al. (2018) noted an increase in the incidence of excessive fetal growth and LGA births between 1998 and 2013 (T1D $n = 3229$) [18]. Furthermore, in 2021, the Continuous Glucose Monitoring in Women with Type 1 Diabetes in Pregnancy Trial (CONCEPTT) collaborative group reported that among pregnant women with T1D the rate of LGA births was 66%, a figure that exceeded expectations in the cohort with relatively good glycaemic control and the use of CGM systems [19].

The standard thresholds for SGA, AGA, and LGA categorization are applied to T1D pregnancies, but their clinical interpretation deviates significantly. In the non-diabetic population, SGA can represent constitutional smallness but is reasonably monitored for subsequent FGR development. In T1D, an SGA fetus is far more likely to represent true pathological FGR. The physiological setting, such as chronic hyperglycaemia, associated with hyperinsulinaemia and insulin-like growth factor axis alterations, high nutrient supply, and increased lipid levels should enhance fetal growth [20–23]. LGA, using standard centiles, underestimates the metabolic burden in T1D. Disproportionate fetal body composition is commonly observed even in AGA fetuses in women with poor glycaemic control. In populations characterised by higher birthweights, particularly among infants born to

overweight or obese women, the stillbirth rate may be comparable at thresholds exceeding the 10th percentile (18th, 25th, 31st, or even 41st percentile) [24]. Since accelerated fetal growth is frequently observed in pregnancies complicated by T1D, phenotypes of FGR may not be fully represented by infants with a birthweight below the 10th percentile in this population. SGA remains uncommon in pregnancies complicated by T1D, unless alternative fetal growth charts or shifted percentile thresholds are applied [25]. An SGA fetus in a T1D mother is a rare and ominous finding and demands investigation for placental insufficiency. In T1D, the focus is shifted from a percentile label to dynamic assessment of fetal growth trajectory deviation from its projected physiological potential.

Relative growth impairment may also occur among infants with birthweight above the conventional 10th-percentile SGA threshold. The CONCEPTT collaborative group has identified a trend towards the highest risk of multiple neonatal complications in infants with birthweight below the 25th percentile, although the total number of infants in this category remained relatively small [19].

2. Materials and Methods

We conducted a literature search of the PubMed, MEDLINE, Web of Science, and CNKI databases for studies published between 2001 and 2026. Previous reviews and cross-referenced publications were also screened. The search was performed in May 2026. The search strategy involved the terms: type 1 diabetes, or pregestational diabetes, or preexisting diabetes; and: pregnancy, or gestation, or birth weight, or birthweight, or fetal growth, or fetal growth restriction, or fetal growth retardation, or FGR, or IUGR, or macrosomia, or SGA, or LGA; and: adverse outcome, or stillbirth, or birth trauma, or birth injury, or shoulder dystocia, or respiratory disorder, or respiratory distress, or neonatal intensive care, or neonatal hypoglycemia, or hypoxic–ischemic encephalopathy, or cerebral ischemia, or asphyxia. The initial search identified a total of 579 results that were screened at the title and abstract level to distinguish those reporting neonatal outcomes stratified by birthweight percentile or category. No language restrictions were applied to full-text articles provided that an English abstract was available. Papers containing data on perinatal outcomes in different birthweight categories were selected for full-text review.

3. Results

A U- or J-shaped distribution of perinatal mortality and morbidity rates is typically observed across birthweight percentile ranges [26,27]. Table 1 presents the risks of specific adverse outcomes, as well as a composite adverse outcome, in newborns at birthweight extremes based on data previously reported by various research groups.

As part of the CONCEPTT study, significant attention was paid to classifying infants born to mothers with T1D to SGA, LGA, and AGA, and to assessing the risk of adverse neonatal outcomes in these categories. A composite outcome comprising birth trauma, neonatal hypoglycaemia, hyperbilirubinaemia, respiratory distress, and admission to the neonatal intensive care unit for more than 24 h was assessed [1,19]. The CONCEPTT collaborative group also identified a U-shaped relationship between the risk of adverse outcomes and birthweight percentile in infants of mothers with T1D [19].

Previously, Persson et al. (2012) reported comparable rates of a composite adverse neonatal outcome (5-min Apgar score below 7, birth trauma, respiratory disorder, hyperbilirubinaemia, hypoglycaemia) in proportionate and disproportionate LGA infants classified based their ponderal index [28]. Both rates exceeded those observed in AGA infants born to women with T1D. The risk of adverse outcomes depended on gestational age. Preterm LGA infants, both proportionate and disproportionate, faced a risk similar to that of preterm AGA infants [28].

Table 1. Adverse outcome risks in small- and large-for-gestational-age infants born to mothers with type 1 diabetes.

Outcome	Author, Year	T1D or PGD Population	SGA (<10th Percentile or Different Threshold)	LGA (>90th Percentile) and Extremely LGA	Reference *
C-NAO: birth trauma, hypoglycaemia, hyperbilirubinaemia, RDS, NICU admission	Meek et al. (CONCEPTT), 2021 [19]	T1D <i>n</i> = 225	<10th: RR 1.41 (0.63–3.18); <25th: RR 1.21 (0.63–2.33)	>90th: RR 1.41 (1.01–1.96) >97.7th: RR 1.42 (1.08–1.86)	all other birthweight categories
C-NAO: Apgar score < 7 at 5 min, birth trauma, respiratory disorder, hyperbilirubinaemia, hypoglycaemia	Persson et al., 2012 [28]	T1D <i>n</i> = 3517	ND	term D-LGA— OR 1.77 (1.39–2.26) ; term P-LGA— OR 1.80 (1.45–2.24) ; preterm D-LGA—OR 1.19 (0.90–1.54); preterm P-LGA—OR 1.21 (0.94–1.57)	AGA (GA-adjusted)
C-NAO: RDS, hypoglycaemia, polycythaemia, hyperbilirubinaemia, acidosis	Bollepalli et al., 2010 [29]	T1D <i>n</i> = 302	ND	P-LGA— OR 2.4 (1.3–4.4) ; D-LGA— OR 3.2 (1.7–5.9)	P-non-LGA
Birth trauma (Erb's palsy, clavicular fracture) and shoulder dystocia	Meek et al. (CONCEPTT), 2021 [19]	T1D <i>n</i> = 225	<10th: <i>n</i> = 0; <25th: <i>n</i> = 1 (INS)	>90th <i>n</i> = 1; Extremely LGA <i>n</i> = 0 (INS)	–
	Persson et al., 2012 [28]	T1D <i>n</i> = 3517	ND	term D-LGA— OR 4.78 (2.36–9.71) ; term P-LGA— OR 4.72 (2.54–8.78)	AGA (GA-adjusted)
Neonatal hypoglycaemia	Meek et al. (CONCEPTT), 2021 [19]	T1D <i>n</i> = 225	<10th: INS; <25th: RR 0.56 (0.09–3.46)	>90th: RR 1.69 (0.97–2.94); >97.7th: RR 2.30 (1.45–3.65)	all other birthweight categories
	Persson et al., 2012 [28]	T1D <i>n</i> = 3517	ND	term D-LGA—OR 1.42 (0.97–2.08); term P-LGA— OR 1.42 (1.01–2.00)	AGA (GA-adjusted)
	Bollepalli et al., 2010 [29]	T1D <i>n</i> = 302	ND	P-LGA—OR 2.3 (0.9–6.1); D-LGA— OR 3.5 (1.4–8.7)	P-non-LGA
RDS/Respiratory disorders	Meek et al. (CONCEPTT), 2021 [19]	T1D <i>n</i> = 225	<10th: RR 4.11 (0.78–21.63); <25th: RR 3.66 (1.04–12.88)	>90th: RR 1.88 (0.64–5.45); >97.7th: RR 1.61 (0.68–3.80)	all other birthweight categories
	Persson et al., 2012 [28]	T1D <i>n</i> = 3517	ND	term D-LGA— OR 2.77 (1.29–5.93) ; term P-LGA— OR 2.38 (1.12–5.06)	AGA (GA-adjusted)
	Bollepalli et al., 2010 [29]	T1D <i>n</i> = 302	ND	P-LGA—OR 1.6 (0.6–4.2); D-LGA—OR 1.5 (0.5–4.0)	P-non-LGA
NICU admission for more than 24 h	Meek et al. (CONCEPTT), 2021 [19]	T1D <i>n</i> = 225	<10th: RR 1.83 (0.81–4.14); <25th: RR 1.58 (0.81–3.07)	>90th: RR 1.48 (0.98–2.22); >97.7th: RR 1.48 (1.06–2.08)	all other birthweight categories
Hyperbilirubinaemia	Meek et al. (CONCEPTT), 2021 [19]	T1D <i>n</i> = 225	<10th: RR 2.47 (1.08–5.65) ; <25th: RR 2.15 (1.09–4.23)	>90th: RR 1.44 (0.87–2.36); >97.7th: RR 1.19 (0.78–1.82)	all other birthweight categories
	Persson et al., 2012 [28]	T1D <i>n</i> = 3517	ND	term D-LGA— OR 2.22 (1.04–4.88) ; term P-LGA— OR 2.38 (1.19–4.76)	AGA (GA-adjusted)
	Bollepalli et al., 2010 [29]	T1D <i>n</i> = 302	ND	P-LGA—OR 1.8 (0.9–3.3); D-LGA— OR 2.2 (1.2–4.2)	P-non-LGA

Table 1. Cont.

Outcome	Author, Year	T1D or PGD Population	SGA (<10th Percentile or Different Threshold)	LGA (>90th Percentile) and Extremely LGA	Reference *
Stillbirth	Mackin et al., 2019 [30]	T1D <i>n</i> = 3778	<10th: OR 6.3 (2.3–17.3)	>90th: OR 1.5 (0.9–2.6); >95th: OR 1.5 (0.8–2.6)	AGA
	Gordon et al., 2025 [31]	T1D <i>n</i> = 1022	no stillbirths in SGA subgroup (<i>n</i> = 26), the reported rates were extrapolated	>90th: RR values not stated; >97th: RR 3.96 (1.23–12.76)	same birthweight category without diabetes
	McElwee et al., 2023 [32]	PGD <i>n</i> = 109,519	<10th, stratified by GA: 36 weeks— RR 3.9 (2.6–5.8) ; 37 weeks— RR 2.5 (1.6–4.0) ; 38 weeks— RR 2.6 (1.6–4.1) ; 39 weeks— RR 3.0 (1.5–6.1)	>90th, stratified by GA: 36 weeks— RR 2.9 (2.2–3.9) ; 37 weeks— RR 4.0 (3.1–5.3) ; 38 weeks— RR 3.8 (2.8–4.9) ; 39 weeks— RR 5.3 (3.4–8.3)	AGA in PGD (GA-adjusted)

Comment. In the studies by Persson et al. (2012) [28] and Bollepalli et al. (2010) [29], LGA (>90th percentile) infants were stratified by body proportion using Ponderal index. Disproportionality (D-LGA) was defined as ponderal index > 90th percentile. Otherwise, infants were considered proportionate (P-LGA). For birthweight assessment, different fetal growth standards were applied (data not included). Statistically significant odds ratios (ORs) and relative risks (RRs) with 95% confidence intervals are highlighted in bold. * OR/RRs are reported in comparison to different reference subgroups in the studies. All the studies, with the exception of Gordon et al. (2025) [31] and McElwee et al. (2023) [32], used subgroups within T1D groups as the reference group. Gordon et al. (2025) [31] compared outcomes in LGA and SGA with the same birthweight category in population-based cohort without diabetes (T1D LGA vs. LGA without diabetes). McElwee et al. (2023) [32] used AGA in PGD stratified by GA as a reference group. C-NAO—Composite Neonatal Adverse Outcome; D-LGA—disproportionate LGA; GA—gestational age; ND—no data on the outcome rate in the exact birthweight category; INS—insufficient events in the study cohort; NICU—neonatal intensive care unit; P-LGA—proportionate LGA; P-non-LGA—proportionate non-LGA; PGD—pregestational diabetes; RDS—respiratory distress syndrome; T1D—type 1 diabetes; AGA—appropriate for gestational age (10–90th percentile); LGA—large for gestational age; SGA—small for gestational age.

3.1. Birth Trauma and Shoulder Dystocia

Pregnancies complicated by diabetes mellitus carry an increased risk of shoulder dystocia. The incidence of shoulder dystocia rises with increasing fetal birthweight. This trend is not limited to weights exceeding 4000 or 4500 g [33–36]. Predicting shoulder dystocia remains a challenge [37,38]. While the use of maternal characteristics and ultrasound parameters enables risk stratification for shoulder dystocia and birth trauma with a sensitivity of 31.5% at a 10% false-positive rate, its performance is only marginally better than that of an approach based solely on estimated fetal weight [38]. The disproportionate fetal growth observed in diabetic pregnancies is characterised by excessive adipose tissue deposition in the upper body, which intensifies as early as the beginning of the third trimester [39,40]. In cases of suboptimal glycaemic control, disproportionate fat deposition is observed not only in large fetuses but also in those with AGA anthropometric measurements [22,29,40]. This predisposes to a higher incidence of birth trauma and makes such events even more difficult to predict. New methods for assessing fetal weight and tissue composition, including ultrasound evaluation of soft tissues and determination of fractional limb volume, have been proposed to predict disproportionate growth [29,37,40–45]. However, a reliable method for the antenatal prediction of shoulder dystocia and the risk of birth trauma has yet to be developed.

The results from the Big Baby Trial demonstrated that, even in low-risk pregnancies without maternal diabetes but with antenatally suspected LGA, induction of labour was associated with lower rates of shoulder dystocia and prelabour C-section [46]. In particular, induction of labour performed between 38 + 0 and 38 + 4 weeks of gestation significantly reduced the incidence of shoulder dystocia compared to delivery at a later gestational age, even within this low-risk group [46]. Although maternal T1D is an independent risk

factor for shoulder dystocia, fetal overgrowth associated with diabetes further increases the likelihood of this complication, as well as the risk of delivery by cesarean section. Ward et al. (2026) emphasised that, among women with PGD and fetal macrosomia, planned delivery after 39 weeks of gestation was associated with a lower risk of C-section compared to expectant management [47]. Conversely, early planned delivery prior to 38 weeks, particularly between 36 and 37 weeks, was associated with higher C-section rates and increased neonatal morbidity [47]. These findings highlight the importance of balancing the risks associated with ongoing intrauterine exposure against the risks posed by preterm or early-term delivery when determining the optimal timing and mode of delivery in pregnancies affected by preexisting diabetes and complicated by excessive fetal growth [48].

3.2. Respiratory Disorders

Pregestational diabetes is associated with an increased prevalence of preeclampsia, preterm birth and cesarean delivery, which contribute substantially to neonatal respiratory complications [4,48–52]. Since lung maturation occurs during the later stages of pregnancy, the extent to which the timing of hyperglycaemia exposure influences the incidence of respiratory disorders in newborns remains unclear. However, early fetal exposure to maternal hyperglycaemia in T1D affects the neonatal respiratory system [53]. Even near-term infants born to mothers with T1D are susceptible to neonatal respiratory distress syndrome (RDS) [53–55]. RDS in T1D pregnancies is not purely a function of gestational age but also results from impaired surfactant synthesis and function secondary to fetal hyperglycaemia and hyperinsulinaemia [53]. Prolonged exposure to hyperglycaemia and hyperinsulinaemia increases the risk of placenta-associated disorders and preterm birth [13,53,55,56]. The use of advanced T1D management technologies, including real-time CGM and automated insulin delivery systems, improves overall neonatal health outcomes [1,13,57]. However, CONCEPTT and AiDAPT trials have not demonstrated an independent reduction in the incidence of RDS [1,57].

It has been proven that antenatal corticosteroid therapy improves outcomes in preterm infants, and these improvements are pronounced in both women without and with diabetes [58]. Administration of antenatal corticosteroids in women with diabetes at risk of late preterm birth remains a subject of discussion [59]. In late-preterm or term pregnancies, the low rate of antenatal corticosteroid use precludes firm conclusions regarding neonatal respiratory morbidity [60]. Further trials are needed to evaluate the efficacy and safety of corticosteroid use in women with diabetes during late pregnancy.

Given the molecular mechanisms underlying the persistent risk of respiratory morbidity in pregnancies complicated by T1D, the primary clinical objective remains the improvement of maternal glycaemic control [28,53]. While fetal growth abnormalities are associated with respiratory disorders, they still serve as unreliable predictors [29,61].

3.3. Neonatal Hypoglycaemia

Neonatal hypoglycaemia is a common complication in infants born to mothers with T1D [5,21,62]. Infants with abnormal growth trajectories, both restricted and accelerated, are at high risk of developing hypoglycaemia [19,41,63,64]. According to the CONCEPTT collaborative group, in pregnancies complicated by T1D, infants with a birthweight exceeding the 97.7th percentile were at the highest risk of neonatal hypoglycaemia (RR 2.30, 95% CI 1.45–3.65) [19,65]. However, antenatal fetal weight estimates and even actual birthweight alone are unreliable predictors of neonatal hypoglycaemia [28,66]. Other methods for assessing antenatal risk based on additional fetometric parameters have previously been proposed. To predict the development of early neonatal hypoglycaemia, Lysenko et al.

(2021) proposed sonographic measurement of fetal pancreatic thickness during the third trimester as an informative and reproducible fetometric parameter [67]. Assessment of fetal body composition is considered a potential predictor of neonatal hypoglycaemia [41]. These measures are thought to be indirectly linked to the development of neonatal hypoglycaemia, reflecting secondary effects of chronic hyperglycaemia and hyperinsulinaemia, disproportionate fetal growth, and hepatic perfusion.

3.4. Perinatal Asphyxia and Related Complications

Newborns of mothers with PGD experience birth asphyxia and asphyxia-related complications more frequently. A Swedish population-based study ($n = 1,343,751$ liveborn infants; T1D $n = 5941$) by Cnattingius et al. (2017) reported an increased risk of a low (score < 7) 5 min Apgar score (OR 2.67, 95% CI 2.23–3.20), hypoxic ischaemic encephalopathy, and neonatal seizures (OR 3.41, 95% CI 2.58–4.49) among offspring of mothers with T1D [68]. The authors noted that these elevated risks associated with T1D pregnancies persisted even in term infants with birthweight appropriate for gestational age (within the 10th–90th percentile) [68]. Given that the risks of asphyxia-related complications in pregnancies complicated by type 2 diabetes (T2D) were largely dependent on the presence of maternal hypertensive disorders, preterm birth, and abnormal fetal weight, the authors suggested that the mechanisms underlying perinatal hypoxic complications differ between T1D and T2D. Cnattingius et al. (2017) also emphasised that an apparently “normal” birthweight in pregnancies complicated by T1D should not be viewed as evidence of normal intrauterine development conditions [68]. This apparent normality may result from fetal overgrowth masking impaired placentation caused by maternal microangiopathy and chronic hyperglycaemia. Under such conditions, these fetuses become more vulnerable to hypoxia. The persistent fetal susceptibility to chronic hypoxia despite apparently appropriate fetal growth in pregnancies complicated by T1D is confirmed by data indicating a significantly higher risk of chronic fetal hypoxia and stillbirth from 32 weeks of gestation onwards [69], with a progressive increase in risk near term gestation [31,32].

3.5. Stillbirth Across the Birthweight Categories in Pregnancies with Type 1 Diabetes

Despite a reduction in the rate of stillbirths in pregnancies complicated by diabetes, PGD is associated with a threefold increase in the risk of stillbirth and perinatal mortality [70], and approximately 4% of all stillbirths are attributable to maternal diabetes [71]. Maternal hyperglycaemia and increased glucose variability contribute to the development of fetal metabolic acidosis and hypoxemia. Impaired fetal growth, accompanied by cardiac dysfunction and chronic hypoxia, increases the risk of intrauterine death [48,69,72].

The risk of stillbirth in women with PGD is closely linked to birthweight extremes. A nationwide study conducted in Scotland by Mackin et al. (2019) analyzed fetal growth categories in conjunction with maternal characteristics, glycaemic control, and timing of delivery regarding stillbirths in singleton pregnancies among women with T1D ($n = 3778$; with stillbirth rate 16.1 per 1000 births, $n = 61$) and T2D ($n = 1614$; stillbirth rate 22.9 per 1000 births, $n = 37$) [30]. The majority of stillborn infants in both T1D and T2D were born LGA or SGA. In T1D pregnancies, infants born SGA (<10th birthweight percentile) had a sixfold higher risk of stillbirth compared to those born AGA (between the 10th and 90th percentiles). The upper extremes of birthweight were also common among stillbirths, and 57.6% of stillborn infants in T1D were born LGA. Although gestational age-adjusted birthweight metrics were comparable between stillborn and live-born infants (z-scores 1.38 and 1.37), the association between the LGA category and stillbirth in women with T1D remained statistically non-significant. By contrast, among offspring of mothers with T2D, those with a birthweight above the 95th percentile had a twofold higher risk of stillbirth.

The authors also highlighted that approximately one-third of stillbirths occurred at term. The highest incidence was observed at 38 weeks of gestation among women with T1D [30].

In another population-based cohort study, McElwee et al. (2023) also identified the highest stillbirth rate after 39 weeks of gestation among fetuses classified as LGA (96.9 per 10,000 ongoing pregnancies) among women with PGD (total $n = 109,519$; not stratified by T1D and T2D). Notably, the highest stillbirth RRs were in LGA fetuses after 37 and 38 weeks of gestation (37 weeks of gestation: RR 21.8, 95% CI 17.4–27.2; 38 weeks of gestation: RR 21.3, 95% CI 16.8–27.0) compared to AGA fetuses in a cohort of women with gestational diabetes, which served as the reference group [32]. The authors analyzed gestational age-adjusted stillbirth rates at 34 to 39 weeks of gestation. In pregnancies complicated by PGD, fetuses classified as SGA (at any gestational age) and LGA fetuses (from 35 weeks onwards) faced an increased risk of stillbirth compared to AGA fetuses in women with PGD. Within the cohort of women with PGD, the risk of stillbirth for LGA fetuses was 1.6 times higher than for SGA fetuses at 37 weeks of gestation, with negligible differences at other gestational ages [32].

Subsequently, Gordon et al. (2025) reported elevated stillbirth rates associated with both fetal growth extremes. It was in an Australian population-based cohort study of pregnancies complicated by pregestational and gestational diabetes (T1D $n = 1022$, T2D $n = 5206$, GDM $n = 94,628$, control $n = 759,186$) [31]. Among women with PGD, the risk of stillbirth was elevated regardless of gestational age or birthweight category. Stillbirths accounted for 0.7% of all pregnancies among women with T1D. All 26 SGA infants of women with T1D were liveborn. The reported high stillbirth risk in the SGA cohort was extrapolated based on the rates across other birthweight percentiles, overall increased stillbirth risk in T1D pregnancies and decreased prevalence of SGA birth in T1D. The authors emphasised a fourfold higher stillbirth risk in LGA infants with birthweight > 97th percentile born to women with T1D compared to LGA infants of mothers without diabetes (RR 3.96, 95% CI 1.23–12.76). The authors also observed a gestational age-dependent trend in stillbirth rate. In cases of PGD, an increase in rates was observed after the 38th week of pregnancy, with the sharpest rise noted at 39 weeks and peak values occurring after 40 weeks [31].

In T1D pregnancies, fetal growth abnormalities, maternal metabolic status, and timing of delivery determine the perinatal outcomes. Standard Doppler criteria, applied successfully in growth-restricted fetuses, fail to identify a risk group for adverse pregnancy outcomes and fetal demise in pregnancies with diabetes and macrosomic fetal growth [73,74]. Assessment of fetal venous blood flow may be promising for this patient cohort [73,75–78].

One adaptive response to fetal hypoxia is redistribution of oxygenated blood between the fetal liver and the ductus venosus (DV), the latter directing oxygen rich blood toward the fetal heart and brain [73,75–81]. In growth-restricted fetuses, previous studies report an increased proportion of blood shunted through the DV [79–81]. In contrast, maternal diabetes is associated with altered venous blood flow distribution characterised by preferential perfusion of the fetal liver and a reduced DV shunt fraction [75,77,78,82]. Lund et al. (2018) demonstrated that a marked reduction in oxygenated flow through the DV correlates with fetal lactic acidemia at delivery [76]. Reduced ductus venosus shunt fraction potentially can indicate impaired fetal capacity to maintain adequate adaptation against hypoxia. Clinical challenges in assessment of circulatory changes in fetal overgrowth contribute to increased rates of fetal compromise, as the risk in these fetuses remain underevaluated [78].

3.6. Does Fetal Growth Chart Matter?

While numerous nationwide studies on fetal growth standards have focused on the identification of SGA and growth-restricted fetuses in general populations, given their increased risk of adverse outcomes, maternal T1D makes accelerated growth and LGA birth fivefold more common [83]. Comparing results across various nationwide studies regarding birthweight-related outcomes remains challenging, as definitions of SGA and LGA vary depending on the growth standard applied, encompassing both customised and population-based approaches.

Even in pregnancies not complicated by diabetes, antenatal LGA or FGR diagnosis influences subsequent management strategies for further obstetric care [46,84,85]. The efficacy of the prenatal diagnosis of macrosomia remains uncertain and depends on gestational age. When the assessment is performed several weeks prior to delivery, the rates of both false-positive and false-negative results increase. As maternal diabetes is associated with accelerated and disproportionate fetal growth, particularly during the 3rd trimester, the accuracy of routine sonographic examination is reduced [86].

Achieving glycaemic targets close to normal levels is a key objective in the management of pregnancies complicated by T1D [1,8,13,14]. The CONCEPTT study provided detailed data on fetal growth patterns and perinatal outcomes in women with T1D using CGM [1,19]. The CONCEPTT research team compared the incidence of complications in newborns classified as SGA and LGA according to three commonly used fetal growth charts (GROW, INTERGROWTH-21st, WHO) [19]. Among this cohort of 225 maternal–infant pairs, 66.7% of neonates were LGA (>90th percentile) and 1.3% were SGA (<10th percentile) according to INTERGROWTH-21st [19]. Lower birthweight was uncommon among infants born to T1D mothers. The applicability of SGA and LGA definitions in pregnancy with T1D was similar for GROW and INTERGROWTH-21st, but the WHO standards showed poorer performance due to the lack of adjustment for gestational age. The WHO criteria identified preterm neonates as born as having lower birthweight percentile. The 25th percentile according to the WHO criteria threshold was associated with increased risk for NICU admission exceeding 24 h duration (RR 1.83, 95% CI 1.31–2.57), hyperbilirubinaemia (RR 1.77, 95% CI 1.13–2.76), and respiratory distress (RR 2.96, 95% CI 1.25–7.02), and the RRs at 10th percentile threshold were even higher [19]. Neither these outcomes, with the exception of hyperbilirubinaemia, nor the composite neonatal outcome measure were associated with low birthweight thresholds (10th or 25th percentile) based on INTERGROWTH-21st standards. Infants with birthweights below the 25th percentile according to GROW or INTERGROWTH-21st standards showed an increased risk of developing hyperbilirubinaemia. The CONCEPTT Collaborative Group found the applicability of the 10th and 25th percentile thresholds for the outcome of neonatal hypoglycaemia to be similar across the different criteria used [19]. Moreover, it should be noted that the birthweight distribution in the INTERGROWTH-21st standards is shifted to the left, leading to a higher number of newborns being classified as LGA. Consequently, an infant classified as SGA by other criteria might be identified as AGA using the INTERGROWTH-21st standards. While GROW standards account for maternal anthropometric data, parity, and ethnicity, INTERGROWTH-21st growth charts are based on low-risk pregnancies and support the multi-ethnic applicability of the standards [87–89].

Given the applicability of both approaches [19,89,90], the findings of the CONCEPTT study confirm that birthweight percentile thresholds alone have limited predictive value regarding neonatal morbidity prognosis [19]. Determining which fetal parameters in pregnancies complicated by diabetes should be considered appropriate for gestational age must be based not only on percentile thresholds but also on the trajectory of intrauterine growth and the characteristics of maternal glycaemic control.

4. Limitations

This narrative review has several limitations. First, we did not conduct a systematic search of all published studies. We selected articles using a non-specific search strategy. The articles were analyzed based on titles and abstracts, and additional references were identified from our existing knowledge, which introduced a risk of selection bias. During the initial search, it was not possible to exclude publications lacking data on outcomes across different birthweight categories because searches related to fetal growth abnormalities in T1D yielded a large number of results. Second, the studies included in the review used different definitions of fetal growth abnormalities. Different percentile cut-offs or various growth charts were used. However, we focused on birthweight-related outcomes rather than antenatal fetal weight assessment. Third, the severity of maternal T1D (e.g., renal disease or poor glycaemic control) may contribute to both abnormal fetal growth and adverse neonatal outcomes. Such confounding makes it difficult to determine whether the birthweight category independently affects the risk of these complications. As this is not a systematic review, we cannot provide pooled effect size estimates. Consequently, the findings presented here should be considered preliminary rather than definitive.

This review focused primarily on comparing adverse perinatal outcomes associated with the two fetal growth extremes. Women with T1D remain at increased risk of fetal macrosomia and other perinatal complications even with optimal glycaemic control [6,21,91]. Therefore, glucose-independent mechanisms are thought to be involved in the regulation of fetal growth and placental function. Moreover, circulating markers may reflect the pathogenesis of abnormal fetal growth and placenta-associated disorders and serve as valuable tools for identifying high-risk pregnancies. Among the best-supported endocrine growth mediators are components of the growth hormone–IGF-I axis. Placental growth hormone (PGH) and components of the IGF system act as paracrine and autocrine regulators of placental development, function, and nutrient transport capacity [92–96]. PGH, IGFs, and their binding proteins are dysregulated in T1D [23,92,95–99]. These factors, along with adipokines such as leptin and adiponectin, are also altered in fetal growth extremes [92,94,100] and correlate with neonatal adiposity and birthweight [100–103]. Besides abnormal adipokine levels, maternal dyslipidaemia, particularly hypertriglyceridaemia, independently contributes to fetal overgrowth, even under strict glycaemic control [104]. Furthermore, associations between the above markers with the development of preeclampsia and impaired placental function has also been reported [101,105–108]. Patients with chronic hyperglycaemia and T1D, in particular, are characterised by changes in the immune cell function, cytokine networks [109,110] and the production of angiogenic markers [111]. Diabetic vascular disease, particularly retinopathy and nephropathy, is associated with angiogenic imbalance [111,112] and these patients are at increased risk of developing preeclampsia [113]. Recently, soluble fms-like tyrosine kinase-1 (sFlt-1) and placental growth factor (PlGF) have been widely used to predict and diagnose preeclampsia [111,114–116]. The manifestation of an imbalance in serum angiogenesis markers may reflect the development of placental dysfunction and abnormal fetal growth trajectory. These markers are a useful tool even in pregnant women with T1D [113,117]. Low PlGF levels have been reported in pregnancies with SGA or FGR, even in the absence of preeclampsia [114,115]. In pregnancies affected by T1D, lower PlGF levels and a higher sFlt-1/PlGF ratio are associated with smaller placental and neonatal size and may potentially reflect growth restriction and unfavourable intrauterine conditions, despite these infants not meeting the generally accepted definitions of FGR or SGA [115]. Inflammatory mediators modify placental nutrient transport and fetal metabolic programming [100]. Serum cytokine and growth factor levels correlate with glucose variability and time in range in patients with T1D [110,118]. CGM metrics are associated with multiple

adverse pregnancy outcomes, including fetal growth abnormalities and placenta-associated disorders, thus markers of inflammation and oxidative stress may contribute to their development [13,119,120]. Furthermore, epigenetic modifications, especially altered DNA methylation patterns, exert lasting effects on fetal growth trajectories and further health conditions [94,121]. Fetal growth abnormalities in T1D arise from a complex interplay of endocrine, metabolic, inflammatory, angiogenic, and epigenetic pathways that extend far beyond glucose levels alone. In this review, we did not describe in detail the potential non-glycaemic mechanisms underlying the development of perinatal complications in macrosomic and growth-restricted fetuses.

5. Conclusions

Both growth-restricted and macrosomic fetuses are at increased risk of adverse perinatal outcomes. Increased maternal glucose variability may contribute to various forms of placental dysfunction and altered fetal growth. This may explain the persistence of pregnancy complications despite apparently adequate glycated haemoglobin levels and target capillary glucose values obtained through routine self-monitoring during the preconception period and pregnancy.

In pregnancies complicated by T1D, abnormal intrauterine growth is characterised not only by growth acceleration but also by disproportionate adipose tissue deposition. Because of accelerated fetal growth in T1D pregnancies, fetuses with underlying growth restriction may appear appropriate for gestational age when placental development was initially impaired. Consequently, standard fetal growth charts may misclassify these vulnerable fetuses as AGA and categorise them as being at low risk for perinatal complications. Novel approaches to predicting fetal and neonatal complications in pregnancies with pregestational diabetes may incorporate assessments of fetal body composition and the ductus venosus shunt fraction, but further research is needed. Pregnancies complicated by excessive and accelerated intrauterine growth should be considered high-risk and managed with careful attention to obstetric surveillance, as well as the timing and mode of delivery.

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Abbreviations

The following abbreviations are used in this manuscript:

AGA	appropriate for gestational age
C-NAO	composite neonatal adverse outcome
CGM	continuous glucose monitoring
CI	confidence interval
CONCEPTT	Continuous Glucose Monitoring in Women with Type 1 Diabetes in Pregnancy Trial
FGR	fetal growth restriction
IGF	insulin-like growth factor

LGA	large for gestational age
NICU	neonatal intensive care unit
OR	odds ratio
PGH	placental growth hormone
RDS	respiratory distress syndrome
RR	relative risk
SGA	small for gestational age
T1D	type 1 diabetes
T2D	type 2 diabetes
WHO	World Health Organisation
INS	insufficient events in the study cohort (within Table 1)
ND	no data on the outcome rate in the exact birthweight category (within Table 1)
PGD	pregestational diabetes (within Table 1)

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