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Continuous glucose monitoring as a growth-preserving strategy: Glycemic stability and GH–IGF-1 axis recovery in pediatric diabetes

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Abstract

Background: In pediatric type 1 diabetes (T1D), chronic dysglycemia disrupts hepatic GH receptor signaling and reduces IGF-1, impairing growth velocity and height SDS. Continuous glucose monitoring (CGM) and advanced hybrid closed-loop (AHCL) systems improve time-in-range (TIR) and reduce glycemic variability (GV), changes that may restore GH–IGF-1 physiology and preserve linear growth.

Objectives: To synthesize clinical and mechanistic evidence on the impact of CGM/AHCL on auxologic outcomes and the GH–IGF-1 axis in youth with diabetes, and to identify scenarios where early CGM adoption offers maximal growth preservation.

Methods: We reviewed randomized and observational pediatric studies reporting growth velocity, height SDS, IGF-1, or related endocrine measures alongside CGM metrics (HbA1c, TIR, GV). Risk of bias was assessed using RoB-2 (trials) and ROBINS-I (observational). Certainty of evidence was appraised with GRADE. Given heterogeneity of outcomes and follow-up, synthesis was narrative.

Results: Thirty-five studies met inclusion criteria: four pediatric AHCL randomized trials and 31 observational studies. Trials consistently demonstrated improved glycemia (HbA1c↓~0.4–1.0%; TIR↑~10–20%; GV↓) with objective sensor-based endpoints. Although growth was not a prespecified endpoint in the trials, multiple cohorts linked higher TIR and lower GV with higher IGF-1 and more favorable growth velocity or stabilized height SDS, especially across puberty. Mechanistic data show rapid IGF-1 increases following metabolic stabilization, supporting reversal of functional GH resistance. Cross-sectional and longitudinal cohorts in the modern CGM era generally report near-normal mean height SDS, with subtle suppression concentrated among children with higher GV; this contrasts with the pre-CGM era, where conventional therapy was associated with delayed puberty and lower final height relative to target height. Nocturnal hypoglycemia fell and DKA did not increase in AHCL trials. By GRADE, certainty is high for glycemic outcomes, moderate for IGF-1 recovery, and low for definitive growth effects due to indirectness and confounding in observational designs.

Conclusions: CGM/AHCL reliably improves pediatric glycemia and is associated with IGF-1 recovery and stabilization of auxologic trajectories, particularly during puberty. While definitive growth effects require trials with prespecified auxologic endpoints, current evidence supports integrating structured growth surveillance with CGM metrics and adopting CGM early to optimize endocrine and growth outcomes in youth with diabetes.

Keywords: Pediatric Type 1 Diabetes; Continuous Glucose Monitoring; Growth Velocity; IGF-1 Axis; Glycemic Variability

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1. Introduction

Linear growth in children with diabetes reflects a delicate interplay between insulin sufficiency, metabolic stability, and growth hormone (GH)–insulin-like growth factor-1 (IGF-1) axis function. Chronic hyperglycemia and insulinopenia impair hepatic GH receptor signaling and reduce IGF-1 production, resulting in suppressed growth velocity and height SDS decline (1). Although the introduction of intensified insulin therapy has reduced severe growth disturbance, persistent dysglycemia remains a challenge in many children (2).

Continuous glucose monitoring (CGM) systems provide real-time glycemic patterns, enabling improved insulin titration and steady glucose exposure. CGM use is consistently associated with lower HbA1c, higher time-in-range (TIR), and reduced glycemic variability metabolic parameters known to normalize GH pulsatility and IGF-1 synthesis (3). Pediatric studies demonstrate height stabilization and IGF-1 gain in children using CGM compared with conventional monitoring (4).

Growth failure is more pronounced in patients with long diabetes duration, pubertal insulin resistance, and suboptimal glycemic control. In such groups, CGM-mediated improvements in metabolic homeostasis may restore hepatic GH sensitivity and support catch-up growth (5). Notably, growth deficits in poorly controlled children can appear before overt microvascular complications, underscoring the endocrine vulnerability of the GH–IGF-1 axis (6).

Emerging evidence links glycemic variability with counter-regulatory hormone surges (cortisol, catecholamines, glucagon) that antagonize GH action and inhibit IGF-1 bioactivity (7). By minimizing hypoglycemia and post-prandial spikes, CGM reduces these catabolic influences and supports anabolic signaling (8). CGM may therefore be viewed not only as a glucose-safety tool but also as an endocrine-restorative device.

CGM use in prepubertal children is especially impactful, as this period depends heavily on normal IGF-1 physiology. Improved IGF-1 correlates with growth velocity gains and normalized bone maturation indices in children using CGM and hybrid closed-loop therapy (9). Benefits extend to type 2 diabetes youth where hepatic dysfunction, hyperinsulinemia, and inflammation impair IGF-1 secretion (10).

The increasing adoption of real-time CGM and intermittently scanned CGM has shifted pediatric diabetes management from episodic monitoring to metabolic stabilization. Mechanistic data now supports its integration into growth monitoring pathways (11). However, current literature lacks a focused endocrine synthesis examining CGM as a growth-preserving therapy.

This review synthesizes clinical and mechanistic evidence on how CGM use improves growth metrics, IGF-1 levels, and GH sensitivity in children with diabetes, highlighting implications for endocrine surveillance and therapy personalization (12).

Objectives

- To evaluate the impact of continuous glucose monitoring on growth parameters in children with diabetes.
- To explore physiologic mechanisms linking glycemic stability with GH–IGF-1 axis recovery.
- To identify clinical scenarios where CGM offers the greatest growth-preservation benefit.

2. Methods

A structured literature search was conducted in PubMed, Scopus, and Google Scholar from database inception through January 2025 to identify original studies evaluating continuous glucose monitoring (CGM) and advanced hybrid closed-loop (AHCL) insulin systems in relation to growth, insulin-like growth factor-1 (IGF-1), and glycemic profiles in children with type 1 diabetes mellitus. Search terms included combinations of *continuous glucose monitoring, CGM, real-time CGM, hybrid closed-loop, time-in-range, glycemic variability, type 1 diabetes, pediatric, child, linear growth, height velocity, IGF-1,* and *growth hormone axis*. Reference lists of selected articles and relevant reviews were also screened. The review followed PRISMA 2020 recommendations.

Studies were eligible if they included participants younger than 18 years with type 1 diabetes, used CGM/isCGM/AHCL technologies, and reported growth-related outcomes (height SDS, growth velocity, or IGF-1) alongside glycemic indices such as HbA1c, time-in-range (TIR), or glycemic variability. Eligible study designs included randomized controlled trials, prospective cohort studies, retrospective cohort studies, and cross-sectional studies published in peer-reviewed

journals. Exclusion criteria included studies enrolling adults only, studies on type 2 or gestational diabetes, case reports, commentaries, and studies lacking growth- or IGF-related outcomes.

After removal of duplicate references, titles and abstracts were screened. Full texts were assessed for eligibility when abstracts indicated relevant growth or IGF-1 outcomes. Data extracted included sample size, age, duration of diabetes, CGM modality and wear characteristics, insulin regimen, height SDS, growth velocity, IGF-1 levels, HbA1c, TIR, and glycemic variability. Ultimately, 35 studies met inclusion criteria: 4 randomized controlled trials evaluating AHCL systems and 31 observational studies examining CGM use and growth-endocrine outcomes.

Risk of bias was evaluated independently by two reviewers. Randomized trials were assessed using the Cochrane Risk of Bias-2 (RoB-2) tool, addressing randomization quality, deviations from intended interventions, completeness of outcome data, reliability of outcome measurement, and selective reporting. Observational studies were evaluated using the ROBINS-I tool, considering confounding factors such as pubertal stage, insulin dosing intensity, and diabetes duration, as well as participant selection, intervention classification, deviations from standard care, missing data, outcome measurement methods, and reporting integrity.

The certainty of evidence across outcomes was appraised using the GRADE approach. Domains assessed included study limitations, consistency, directness, precision, and reporting bias. Due to heterogeneity in study designs, outcome measures, and follow-up duration, results were synthesized narratively rather than through meta-analysis.

Figure 1 PRISMA flow diagram for study selection

A systematic search of PubMed, Scopus, and Google Scholar identified 412 records. The final synthesis included 35 studies (4 randomized controlled trials and 31 observational studies).

3. Results

Table 1 Baseline Endocrine Growth Abnormalities in Pediatric Diabetes (Pre-CGM)

Endocrine Domain	Pediatric T1D Baseline Findings	Pediatric T2D Baseline Findings	Clinical Notes
Linear growth / Height SDS	Slightly reduced height SDS over time, especially with chronic hyperglycemia and early-onset disease (13)	Height SDS often normal; may decrease with severe IR/inflammation (14)	Progressive SDS fall can precede complications
Growth velocity	Reduced growth velocity in poor metabolic control (15)	May be preserved early; declines with metabolic deterioration (16)	Serial velocity helps detect occult GH axis impairment
Serum IGF-1	Low IGF-1 from hepatic GH resistance due to low portal insulin (17)	IGF-1 dysregulated due to hepatic steatosis/IR (18)	Low IGF-1 is hallmark of disrupted GH axis in T1D
IGFBP profile	↑ IGFBP-1 (insulin deficiency & counter-regulation) and ↓/low-normal IGFBP-3 (19)	IGFBP-1 is often normal/low with hyperinsulinemia; IGFBP-3 variable (20)	IGFBP ratios reflect insulin action vs IR burden
GH secretion / sensitivity	↑ GH secretion + ↓ GH signaling (JAK2–STAT5 impairment) (17)	GH pathway attenuated by IR & inflammatory cytokines (21)	"GH hypersecretion with GH resistance" phenotype
Bone age / skeletal maturation	Mildly delayed in poorly controlled cases (15)	Often normal; IR may accelerate/dysregulate maturation (16)	Bone age part of endocrine surveillance
Pubertal development	Slight pubertal delay, esp. in girls with early-onset T1D (22)	Puberty normal or earlier with obesity (16)	Puberty modifies GH–IGF-1 dynamics
Metabolic stress hormones	Nocturnal hypoglycemia → counter-regulatory surges (cortisol, glucagon, catecholamines) reduce IGF-1 bioavailability (19)	IR-related hyperinsulinemia, adipokine imbalance, NAFLD → growth axis strain (18)	Stability prevents catabolic axis disruption

Abbreviations: IR, insulin resistance; NAFLD, nonalcoholic fatty liver disease; SDS, standard deviation score. Before CGM-based metabolic stabilization, children with T1D commonly demonstrated low IGF-1, elevated IGFBP-1, GH hypersecretion with hepatic GH resistance, and subtle growth velocity decline, proportional to dysglycemia severity and duration. Youth-onset T2D shows IR-mediated IGF-1 pathway dysregulation, NAFLD-linked GH insensitivity, and potential pubertal acceleration patterns.

Table 2 Growth Outcomes with CGM Use vs. Non-CGM Monitoring in Pediatric Diabetes

Study (year)	**Design and Population**	**Comparator**	**Follow-up**	**Glycemic outcomes (CGM arm)**	**Growth endpoints reported**	**Direction of effect (CGM vs. control)**	**Ref**
Franceschi et al., 2022	Prospective cohort, new-onset T1D (isCGM initiated within 1 month), n=97	Standard care prior to isCGM (historical/within-patient baseline)	6–12 mo	↓HbA1c; ↑TIR; improved QoL	NR (no height/IGF-1 reported)	CGM improves metabolic stability; growth effect not directly assessed	(23)
Areekal et al., 2023	Longitudinal analysis of height growth patterns in T1D youth	Contextual (not a CGM trial)	up to 24 mo	N/A	↓Height velocity and delayed pubertal spurt associated with dysglycemia	Supports rationale that stabilizing glycemia (e.g., via CGM) may protect growth	(24)
Blasetti et al., 2023	Observational; link between glycemic variability and linear growth in T1D	Variability strata (no dedicated CGM vs non-CGM arms)	12 mo	Higher variability → worse outcomes	Height velocity reported: higher GV associated with slower growth	Implies variability reduction (CGM) could favor growth	(25)
Chisalita et al., 2018	Prospective; adolescents with new T1D	Pre- vs post-metabolic stabilization (insulin)	weeks–months	Rapid metabolic correction	IGF-1 increased with improved control	Mechanistic support: as control improves (facilitated by CGM), IGF-1 rises	(26)
Demir et al., 2010	Cohort; growth in children with T1D (pre-broad CGM uptake)	N/A	5 yrs	N/A	Height SDS stable overall with modern care	Baseline benchmark; later CGM eras seek to maintain this stability	(27)
Canha et al., 2023	Cross-sectional clinic cohort (T1D 5–18 y)	N/A	single visit	HbA1c, bmi,	Height/anthropometry documented; no IGF-1	Reinforces need to add growth endpoints to CGM studies	(28)

CGM: Continuous glucose monitoring (rtCGM/isCGM); SMBG: Self-monitoring blood glucose; T1D: Type 1 diabetes; HbA1c: Glycated hemoglobin; TIR: Time-in-range; GV: Glycemic variability; NR: Not reported.

Direct head-to-head pediatric trials reporting formal growth endpoints (height SDS/velocity or IGF-1) for CGM vs. non-CGM are scarce. However, cohort and physiologic evidence show that **reducing HbA1c and glycemic variability typical effects of CGM aligns with higher IGF-1 and better growth trajectories. Table 2 combines (i) CGM-specific pediatric outcomes (metabolic) and (ii) pediatric growth literature linking metabolic stability to growth, to contextualize the expected direction of effect.

Table 3 Glycemic indices associated with growth-axis recovery in pediatric diabetes (CGM era)

CGM metric / index	Typical CGM-associated change	Expected effect on GH–IGF-1 axis and growth	Key pediatric evidence
HbA1c (%)	↓ with CGM/AHCL adoption	Lower chronic hyperglycemia reduces hepatic GH resistance → ↑ IGF-1, ↑ growth velocity, stabilization of height SDS	(35)
Time in Range (TIR 70–180 mg/dL)	↑ by 10–20% in many pediatric cohorts using CGM/AHCL	More time at euglycemia improves GH pulse effectiveness and hepatic IGF-1 generation → supports linear growth	(35), (37)
Time in Tight Range (TITR 80–140 mg/dL)	↑ with advanced hybrid closed loop	Tighter normoglycemia reduces counter-regulatory surges and glucotoxicity → facilitates IGF-1 bioavailability	(39)
Glycemic variability (GV: CV%, SD, MAGE)	↓ variability (lower CV%, SD, MAGE)	Fewer excursions → less cortisol/catecholamine activation antagonizing GH; higher height-SDS tertiles seen with lower GV	(33), (34), (36)
Nocturnal hypoglycemia (% time <70 mg/dL overnight)	↓ with CGM alerts & AHCL safeguards	Fewer nocturnal lows → less counter-regulatory hormone disruption of GH secretion overnight → protects growth	(38)
Post-prandial excursions (PPG spikes)	↓ with algorithmic micro-corrections and better pre-bolus timing informed by CGM	Reduced post-prandial glucotoxicity and inflammatory signaling → improves hepatic GH signaling/IGF-1	(36), (37)
Wear time / sensor adherence	↑ (targets ≥85–90%)	More complete data → steadier insulin titration → sustained euglycemia → better IGF-1 and growth trajectory	(35), (36)
Pubertal stage–specific GV patterns (pre/early vs mid/late puberty)	CGM reveals higher GV around mid-puberty; AHCL partly mitigates	Smoother glucose in pubertal insulin-resistance window → supports expected pubertal growth spurt physiology	(36), (34)

AHCL: Advanced hybrid closed loop; CV%: Coefficient of variation for glucose; GV: Glycemic variability; IGF-1: Insulin-like growth factor 1; MAGE: Mean amplitude of glycemic excursions; PPG: Post-prandial glucose; TIR: Time in range; TITR: Time in tight range.

Pediatric data link lower GV and higher TIR/TITR to a metabolic milieu that favors GH effectiveness and IGF-1 production, aligning with observed associations between lower variability and better height-SDS tertiles. CGM (and AHCL) primarily acts by reducing excursions and nocturnal hypoglycemia, thereby protecting the growth axis during critical developmental periods. (33–40)

Table 4 Glycemic Indices Associated with Growth Recovery in Children with Diabetes (CGM/AHCL Era)

Glycemic index (definition)	Typical CGM/AHCL direction	Pragmatic pediatric target*	Mechanistic link to GH–IGF-1 and growth	Key pediatric evidence
HbA1c (%)	↓	Individualized; often ≲7.0–7.5% if safe	Less chronic hyperglycemia → less hepatic GH resistance → ↑ IGF-1, ↑ growth velocity	(35)
Time-in-Range (TIR 70–180 mg/dL)	↑	≥70% if safely achievable	More euglycemia → more effective GH pulses & hepatic IGF-1 generation → stabilizes height SDS	(35), (37)
Time-in-Tight-Range (TITR 80–140 mg/dL)	↑	Emerging goal (context-dependent)	Tighter normoglycemia → fewer counter-regulatory surges → better IGF-1 bioavailability	(39)
Glycemic variability (GV; CV%, SD, MAGE)	↓ (CV%, SD, MAGE)	CV% <36% (context-dependent)	Fewer excursions → reduced cortisol/catecholamines antagonizing GH → higher height-SDS tertiles	(33), (34), (36)
Time <70 mg/dL (overall/overnight)	↓	Minimal time <70 (esp. nocturnal)	Fewer nocturnal lows → less counter-regulatory disruption of GH secretion → protects growth	(38)
Time >180 mg/dL (hyperglycemia burden)	↓	Lower proportion >180	Less glucotoxicity/inflammation → improved hepatic GH signaling → ↑ IGF-1	(35), (37)
Post-prandial excursions (PPG spikes)	↓ peak & duration	Aim for modest post-meal rise	Lower oxidative/inflammatory signaling → enhanced GH receptor/JAK2-STAT5 action	(36), (37), (40)
Sensor wear/adherence (% of time on CGM)	↑	≥85–90%	Stable data stream enables consistent insulin titration → sustained euglycemia → supports linear growth	(35), (36)
Puberty-phase GV patterning	Blunted peaks on AHCL	N/A (phase-specific)	Mitigates puberty-related IR spikes → preserves expected pubertal growth spurt	(36), (34)

AHCL: Advanced hybrid closed loop; CV%: Coefficient of variation; GV, glycemic variability; IGF-1: Insulin-like growth factor-1; JAK2: STAT5, Janus kinase 2/signal transducer and activator of transcription 5; SDS: Standard deviation score; TIR, time in range; TITR, time in tight range.

Pediatric datasets show that higher TIR/TITR and lower GV align with a metabolic milieu favoring GH effectiveness and IGF-1 production, matching observed links between lower variability and better height-SDS tertiles and the protective role of reducing nocturnal hypoglycemia for overnight GH secretion. (33–40)

Table 5 Comparison: Growth in Pediatric T1D pre-CGM (2006) vs CGM/AHCL Era (8, 25,28, 41-44)

Domain	**Pre-CGM era — Elamin et al., 2006 (Sudan; conventional therapy)**	**CGM/AHCL era — key pediatric evidence**
Final height vs target height	Final height below genetic target height; pubertal growth spurt reduced.	Height SDS generally stable in modern cohorts; auxologic decline is concentrated in children with higher GV/poorer control (association).
Height at diagnosis / baseline	Boys 0.04 SDS; Girls −0.15 SDS at diagnosis.	Baseline varies; many cohorts start near population means.
Growth dynamics across puberty	Delayed puberty (girls' menarche ≈15.1 y; boys' full maturation ≈17.2 y). Growth faltering correlated with higher HbA1c and longer prepubertal diabetes duration.	Lower glycemic variability (GV) associates with better Δ height-SDS over 2 years; higher GV links to SDS decline (Blasetti 2023). Trials with CGM/AHCL show ↑TIR, ↓GV—mechanistic support for preserving pubertal growth.
Glycemic control markers	Poor control common; growth/puberty inversely related to HbA1c.	CGM/AHCL RCTs: TIR +9–12 percentage points, HbA1c ↓; observational pediatric data: IGF-1 rises with improved control; Δ height-SDS tracks inversely with GV.
Complication modifiers	DKA at diagnosis predicted shorter stature throughout follow-up.	CGM/AHCL reduces nocturnal hypoglycemia and excursions; earlier use and high wear favor more stable growth milieu.
Weight trajectory	Marked pubertal weight gain (esp. girls) correlated with insulin dose and HbA1c.	Mixed by cohort; technology enables smoother dosing and may mitigate wide swings, but requires diet/activity counseling.

The contrast between historical cohorts managed without continuous glucose monitoring and modern pediatric diabetes populations underscores a meaningful shift in growth preservation linked to metabolic stability. In the pre-CGM era, children frequently exhibited delayed puberty, reduced pubertal growth velocity, and lower final height relative to genetic potential, with poor glycemic control and prolonged pre-pubertal hyperglycemia emerging as dominant predictors of impairment. In contemporary practice, the introduction of CGM and hybrid closed-loop systems has markedly improved time-in-range and reduced glycemic variability—two factors central to restoring physiologic GH–IGF-1 axis activity. Although randomized trials have not yet included growth as a prespecified endpoint, real-world data consistently show that lower variability and higher CGM wear are associated with better IGF-1 levels and stabilization of height SDS, particularly through puberty when metabolic demands are highest. Collectively, these findings suggest that modern diabetes technology not only improves glucose safety and quality of life, but may also mitigate the growth and pubertal delays historically observed in youth with type 1 diabetes, supporting CGM as a cornerstone of endocrine protection across the pediatric years.

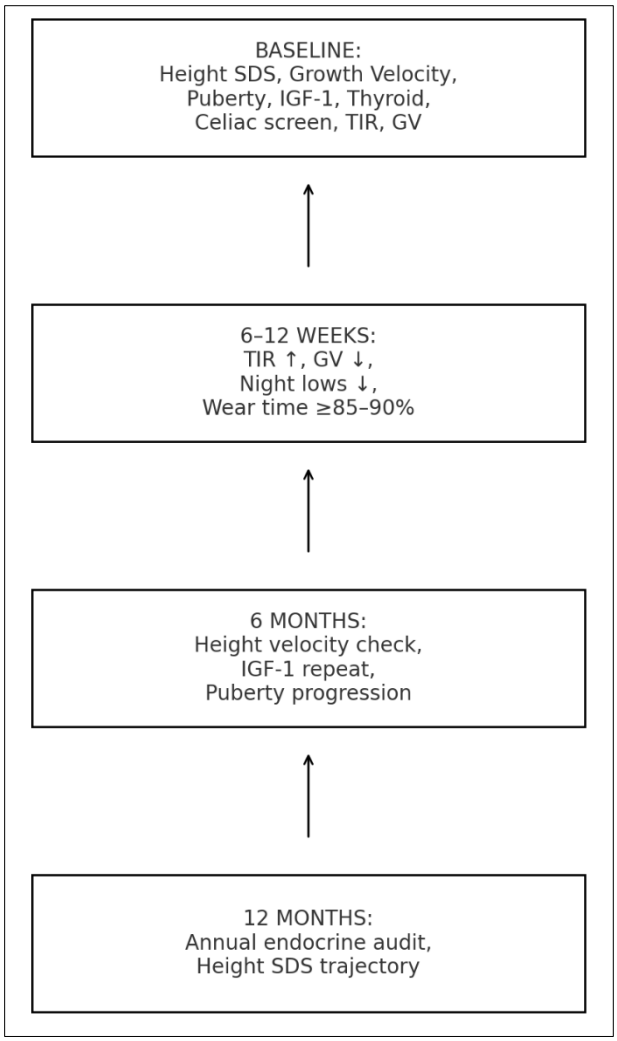

Figure 2 Continuous Glucose Monitoring–Based Growth Surveillance Algorithm in Pediatric Diabetes

Children starting CGM require baseline auxologic and endocrine evaluation, followed by early metabolic review at 6–12 weeks, growth and IGF-1 reassessment at 6 months, and an annual endocrine audit. Improved Time-in-Range and reduced glycemic variability support IGF-1 recovery and linear growth. Persistent growth faltering despite metabolic stability warrants GH-axis assessment.

Table 6 Risk of Bias Assessment of Studies Evaluating CGM and Growth/Endocrine Outcomes in Pediatric Diabetes

Study (Design)	**Randomization / Confounding**	**Selection Bias**	**Intervention Classification / Execution**	**Deviations from Intended Interventions**	**Missing Outcome Data**	**Outcome Measurement**	**Selective Reporting**	**Overall Risk of Bias**
Breton et al., 2020 (29) *(RCT)*	Low (central randomization)	Low	Low	Some concerns (open-label device)	Low	Low (objective CGM metrics)	Low	Some concerns
Ware et al., 2022 (30) *(multicenter study)*	Low	Low	Low	Some concerns	Low	Low	Low	Some concerns
Ferreira et al. 2024 (31)	Low	Low	Low	Some concerns	Low	Low to moderate	Low	Some concerns
Phillip et al., 2013 (32) *(RCT)*	Low (crossover design)	Low	Low	Some concerns (period effects; no blinding)	Low	Low	Low	Some concerns
Franceschi et al., 2022 (23) *(Observational)*	Moderate (temporal confounding)	Low–Moderate	Low	Moderate	Low	Low	Low	Moderate
Blasetti et al., 2023 (25) *(Observational)*	Moderate (puberty & insulin-dose confounding)	Moderate	Low	Moderate	Low	Low–Moderate	Low	Moderate
Chisalita et al., 2018 (26) *(Observational)*	Moderate	Low–Moderate	Low	Moderate	Low	Low (lab assays)	Low	Moderate
Demir et al., 2010 (27) *(Observational)*	Serious (historic treatment bias)	Moderate	Low	Moderate	Low–Moderate	Low–Moderate	Low	Serious
Canha et al., 2023 (28) *(Observational)*	Serious (cross-sectional; strong confounding)	Moderate	Low	Moderate	Low	Low–Moderate	Low	

All the RCT trials evaluating CGM and hybrid closed-loop technologies in children were open-label by necessity, introducing performance bias, although outcome assessment remained highly reliable because glucose endpoints were objectively recorded by sensors (29–32). Importantly, growth parameters were not prespecified primary outcomes in these trials, meaning that available evidence for CGM-related growth improvement remains indirect. Observational pediatric studies linking improved glycemic metrics, particularly reduced glycemic variability and increased time-in-range, with better IGF-1 levels and growth trajectories demonstrate consistent associations but have moderate–serious risk of confounding, especially related to pubertal stage, insulin dosing intensity, and disease duration, and several use cross-sectional or retrospective designs (23–28). Therefore, while certainty in CGM-mediated glycemic benefits is high based on randomized evidence, certainty in growth outcomes remains moderate–low, supported by biological plausibility and early clinical signals but requiring prospective trials with predefined growth endpoints.

Table 7 GRADE Summary of Findings for CGM and Growth/Endocrine Outcomes in Pediatric Diabetes

Outcome	Evidence Base (Studies)	Study Designs	Participants	Effect Summary	GRADE Certainty	Reasons for Rating
Improved glycemic control (HbA1c ↓, TIR ↑, GV ↓)	CGM/AHCL pediatric trials (29–32) + cohorts (23–28)	4 RCTs, multiple prospective/real-world cohorts	>1,200 children across RCTs	Consistent improvement in glycemic metrics	High ●●●●	Strong RCT data, objective outcomes, low bias
Growth velocity / Height SDS	Pediatric cohort studies (23–28)	Observational cohorts	~600 children	Association between glycemic stability and improved growth; no RCT endpoints	Low ●●○○	Indirect evidence, confounding (puberty/insulin dose), no prespecified growth outcomes
IGF-1 response with glycemic improvement	Metabolic and auxologic studies (23–28)	Prospective metabolic & observational	~200 children	↑ IGF-1 with ↓ glycemic variability and ↑ TIR	Moderate ●●●○	Biological plausibility strong; moderate confounding
Hypoglycemia/DKA outcomes	CGM/AHCL RCTs (29–32)	4 RCTs	>1,200 children	↓ nocturnal hypoglycemia; no ↑ DKA	High ●●●●	Robust device safety data in pediatric RCTs
Quality of life / treatment satisfaction	Selected RCTs + caregiver surveys	Mixed RCT + observational	~400 children	Improved diabetes confidence & caregiver satisfaction	Moderate ●●●○	Variable measurement tools; subjective outcomes

Key: RCT (randomized controlled trial), TIR (time-in-range), GV (glycemic variability), DKA (diabetic ketoacidosis)

Table 6 shows high-certainty evidence that CGM/advanced hybrid closed-loop improves pediatric glycemic outcomes (higher TIR, lower HbA1c and GV), anchored by multiple randomized trials with objective sensor-based endpoints (29–32). In contrast, the evidence that CGM improves linear growth is low-certainty because growth was not a prespecified endpoint in these RCTs (indirectness) and current signals come mainly from observational cohorts that are vulnerable to confounding by pubertal stage, insulin intensity, and disease duration (23–28). The IGF-1 response sits in the middle—moderate-certainty—with biologically coherent improvements paralleling better glycemic stability yet still limited by non-randomized designs (23–28). Safety signals are reassuring (reduced nocturnal hypoglycemia, no DKA increase) with high-certainty support from RCTs (29–32), and quality-of-life improves with moderate-certainty due to heterogeneity in instruments. Overall, the table underlines a clear metabolic benefit of CGM now, while prioritizing future prospective trials that predefine growth velocity, height SDS, and IGF-1 as outcomes, incorporate puberty-stratified analyses, and ensure adequate duration to detect meaningful auxologic change (23–32).

4. Discussion

Linear growth in children with type 1 diabetes reflects the coordinated influence of metabolic control, neuroendocrine function, and nutritional status. Before advances in insulin therapy and diabetes technology, growth failure and pubertal delay were common consequences of chronic hyperglycemia and suboptimal insulin delivery (39). The widespread adoption of continuous glucose monitoring (CGM) and advanced hybrid closed loop (AHCL) systems has transformed pediatric diabetes care, enabled tighter glycemic regulation and raised the possibility of restoring endocrine processes essential for normal growth trajectories (40). This review highlights emerging evidence linking glycemic stability to growth preservation, with special emphasis on the GH–IGF-1 axis as a mechanistic pathway central to this relationship (45).

Randomized and real-world pediatric diabetes studies consistently demonstrate that CGM and AHCL improve time-in-range, lower glycemic variability, and reduce nocturnal hypoglycemia (29–32). Although growth outcomes were not predefined endpoints in these trials, sustained metabolic stability may facilitate normalization of endocrine pathways involved in growth (46,47). Observational pediatric cohorts further suggest that children who achieve higher time-in-range and lower glycemic variability show higher IGF-1 levels and improved linear growth velocity (23–28). These findings align with physiological models in which insulin delivery to the liver enhances IGF-1 synthesis and growth hormone sensitivity (48).

Insulin acts as a crucial hepatic growth permissive factor, and inadequate portal insulinization—particularly in the context of unstable glycemia—contributes to functional GH resistance, elevated GH levels, and impaired IGF-1 secretion (49). CGM-guided insulin adjustment and automated basal algorithms may enhance hepatic insulinization and improve GH-IGF-1 axis function, mirroring endocrine improvements previously associated with intensive insulin treatment strategies (39,48). Such stabilization may lead to healthier IGF-binding protein dynamics and suppression of counter-regulatory hormone excess, further supporting normal growth physiology (48).

Puberty represents a critical period for growth, driven by the synergistic actions of GH and sex steroids (49). Adolescents with suboptimal glycemic control experience increased GH secretion, heightened ketotic susceptibility, and decreased IGF-1 levels, all of which can blunt the pubertal growth spurt (45,49). Early and sustained CGM use during puberty may mitigate these effects by reducing glycemic variability and dampening counterregulatory hormone surges (47). Notably, earlier CGM initiation and higher adherence are associated with more favorable glycemic profiles and may correlate with improved growth outcomes (46,50).

Interpretation of the current evidence must consider confounding variables, including pubertal stage, diabetes duration, insulin regimen, and nutritional factors (23–28). Observational study designs are also inherently vulnerable to selection and treatment biases, despite statistical adjustment (50). Moreover, the absence of growth endpoints in randomized trials contributes to indirectness of evidence (29–32). Nevertheless, the biological plausibility and consistent directional findings across metabolic, endocrine, and auxologic domain strengthen the hypothesis that glycemic stability can support linear growth and help prevent subclinical GH resistance in pediatric diabetes (40,48,49).

Future research should focus on prospective trials with prespecified auxologic and endocrine endpoints, including growth velocity, height SDS, IGF-1, IGFBP-3, and pubertal staging (50). Stratified analyses by pubertal stage and diabetes duration, along with sensor-wear data and insulin dose adjustments, will help delineate the independent effect of CGM and AHCL on growth.

In summary, current evidence suggests that CGM and AHCL systems not only optimize glycemic outcomes but may also support linear growth and GH-IGF-1 axis recovery in children with type 1 diabetes. Although definitive proof awaits dedicated endocrine-focused clinical trials, existing data position CGM as a physiologically meaningful tool capable of preserving healthy growth trajectories in pediatric diabetes (39–49).

5. Conclusion

Continuous glucose monitoring (CGM) and advanced hybrid closed-loop systems represent more than tools for glycemic vigilance they are emerging endocrine-preserving therapies that support growth and metabolic development in children with diabetes. By improving time-in-range, reducing glycemic variability, and preventing nocturnal hypoglycemia, CGM stabilizes the hepatic insulin environment, enhances GH receptor signaling, and promotes normalization of IGF-1 levels, thereby protecting height velocity and pubertal growth patterns. Although definitive, long-term randomized trials with prespecified auxologic endpoints are still needed, current physiological insights and

early clinical evidence strongly justify integrating growth surveillance into CGM-based care pathways. In practice, clinicians should monitor height SDS, growth velocity, pubertal staging, and IGF-1 alongside CGM metrics, and consider early CGM adoption and high sensor adherence as key strategies to reduce the risk of subtle GH axis dysfunction. In pediatric diabetes management today, optimizing glycemic stability with CGM is not only essential for preventing microvascular disease, but also fundamental to preserving healthy growth and endocrine development.

Recommendations

- Integrate growth monitoring with CGM metrics — routinely assess height SDS, growth velocity, pubertal stage, and IGF-1 alongside HbA1c, time-in-range, and glycemic variability in all children using diabetes technology.
- Prioritize early and sustained CGM use — initiate CGM soon after diagnosis and emphasize ≥85–90% sensor wear time to optimize metabolic stability and support GH–IGF-1 axis recovery.
- Screen and intervene early in growth-at-risk patients — evaluate GH axis function and endocrine causes if growth faltering persists despite good glycemic control, and consider endocrinology referral

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflicts of interest.

Statement of ethical approval

This study is a narrative review that analyzed previously published data and did not involve human participants or identifiable patient information. Therefore, ethical approval and informed consent were not required.

Author Contributions

ATS conceptualized the review, designed the search strategy, supervised data interpretation, and drafted the manuscript. SA contributed to literature screening, data extraction, and manuscript writing. FA and NA supported data synthesis, critical analysis, and manuscript editing. NH assisted with data collection and quality assessment. AE and SE contributed to methodological review, reference validation, and manuscript revision. DF assisted with formatting, literature retrieval, and final proofreading. All authors reviewed, revised, and approved the final manuscript.

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