Obesity Complicating Pediatric Type 1 Diabetes: Mechanistic Links, Clinical Assessment, and Therapeutic Challenges

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Abstract

Background: The coexistence of obesity and type 1 diabetes (T1D) in childhood has evolved from an apparent paradox into a common and clinically important phenotype. Excess adiposity may precede diagnosis, emerge during pubertal development, or follow insulin initiation and modern intensive diabetes management. In a child who depends on exogenous insulin, weight gain and insulin resistance form a bidirectional cycle: higher insulin exposure can favor energy storage, while adipose dysfunction, ectopic lipid deposition, inflammation, and pubertal hormonal changes increase insulin requirements. This phenotype, often described as double diabetes, may amplify cardiovascular and microvascular risk without producing a predictable relationship between body mass index and glycated hemoglobin. Clinical evaluation therefore must move beyond weight alone. Longitudinal body mass index trajectory, central adiposity, insulin dose, continuous glucose monitoring metrics, blood pressure, lipids, liver health, renal risk markers, pubertal status, medication exposures, physical activity, sleep, and psychological well-being should be interpreted together. The hyperinsulinemic-euglycemic clamp remains the reference method for insulin sensitivity but is impractical in routine care; estimated glucose disposal rate may assist risk stratification, whereas homeostatic model assessment of insulin resistance is generally unsuitable in established T1D because circulating insulin is exogenous. Management requires simultaneous protection from hypoglycemia and ketoacidosis, preservation of healthy growth, reduction of cardiometabolic risk, and avoidance of weight stigma or disordered eating. Insulin optimization, diabetes technology, individualized nutrition therapy, and safely planned activity remain foundational. Metformin may reduce insulin dose or adiposity in selected adolescents but does not provide durable glycemic benefit. Glucagon-like peptide-1 receptor agonists have proven efficacy for adolescent obesity outside T1D, yet pediatric T1D-specific evidence is insufficient; sodium-glucose cotransporter 2 inhibitors carry clinically important ketoacidosis risk. This review integrates current pediatric evidence into a practical framework for recognizing, assessing, and managing obesity complicating T1D while identifying major therapeutic and research gaps.

Keywords: type 1 diabetes; pediatric obesity; insulin resistance; double diabetes; cardiometabolic risk; adjunctive therapy

1. Introduction

Type 1 diabetes (T1D) is defined by immune-mediated loss of pancreatic beta-cell function and lifelong dependence on exogenous insulin. Its global childhood burden continues to rise, while advances in insulin analogues, glucose sensors, pumps, and automated insulin delivery have shifted therapeutic goals from survival toward near-normal glycemia, healthy development, and prevention of vascular disease. At the same time, children with T1D live within the same obesogenic environment as their peers, making excess weight an increasingly prominent part of routine diabetes care. [1,2]

The traditional image of a lean child with T1D is now incomplete. International registry data and pooled analyses indicate that approximately one third of affected children and adolescents have overweight or obesity, although prevalence varies by age, sex, geography, and reference standard. Because body mass index (BMI) can rise after diagnosis and through puberty, a single cross-sectional measurement may obscure an adverse trajectory that has developed during otherwise successful intensification of insulin therapy. [3-5]

Obesity in T1D is not merely a coexisting diagnosis. It changes insulin pharmacodynamics, complicates meal and exercise planning, increases treatment burden, and clusters with hypertension, dyslipidemia, fatty liver, and other manifestations of cardiometabolic risk. The resulting phenotype has been termed "double diabetes," but the label should not imply that every child with obesity has type 2 diabetes or measurable severe insulin resistance. A clinically useful approach is to treat obesity as a modifier of T1D biology and outcomes, requiring integrated assessment rather than diagnostic shorthand. [6,7]

This narrative review synthesizes evidence from pediatric registries, mechanistic studies, systematic reviews, clinical trials, and contemporary guidelines. It examines why obesity develops in youth with T1D, how excess adiposity and insulin resistance should be assessed, which outcomes are most concerning, and how available treatments can be used without compromising glycemic safety, growth, or psychological health. [7-9]

2. The Changing Epidemiology and Phenotype

The worldwide prevalence of overweight and obesity in pediatric T1D closely parallels, and in some settings exceeds, that of the background population. A recent systematic review and meta-analysis estimated a combined prevalence near 30%, while the international SWEET registry reported overweight or obesity in 31.8% of participants. Higher proportions are often observed among girls and older adolescents, consistent with sex-specific pubertal changes, sociocultural influences, and longer exposure to diabetes treatment. [3,4]

Longitudinal evidence is especially informative. International comparisons show that BMI z score frequently increases after diagnosis and may remain elevated across childhood. Predictors include higher baseline BMI, female sex, pubertal progression, diabetes duration, and aspects of insulin treatment, but effects are heterogeneous. Children may enter T1D care with overweight, regain weight rapidly after reversal of catabolism, or accumulate adiposity gradually; these pathways are biologically distinct and may require different interventions. [5,13]

BMI also incompletely characterizes the phenotype. Meta-analytic evidence suggests that youth with T1D may have higher total body fat than matched peers even when BMI differences are modest. Central fat and ectopic lipid are more closely related to insulin resistance than total mass, whereas increasing lean mass during growth is metabolically different from adipose gain. Consequently, the clinical question is not simply whether BMI exceeds a percentile threshold, but whether the child is developing excess or centrally distributed adiposity alongside rising insulin needs and cardiometabolic abnormalities. [10,14]

Lifestyle exposures are interwoven with diabetes self-management. Fear of hypoglycemia can lead to defensive snacking, intentional maintenance of higher glucose levels, or avoidance of vigorous activity. Repeated treatment of sensor alarms with excessive carbohydrate may create substantial unrecognized

energy intake. Screen time, insufficient sleep, family food patterns, socioeconomic constraints, and limited access to safe activity affect children with and without diabetes, but T1D adds calculations, alarms, correction doses, and safety decisions to every behavior. [7,15]

3. Mechanistic Links Between Obesity and Type 1 Diabetes

3.1 Exogenous insulin, energy balance, and weight gain

Insulin is both an essential replacement hormone and a potent anabolic signal. Initiation of therapy reverses glycosuria, proteolysis, and lipolysis, restoring depleted fat and lean tissue. Thereafter, peripheral subcutaneous delivery creates a nonphysiologic insulin distribution: systemic tissues may be exposed to relatively high concentrations while portal insulinization remains lower than in normal physiology. When insulin exposure exceeds metabolic need, lipolysis is suppressed, nutrient storage is favored, and mild hypoglycemia or perceived risk may prompt additional carbohydrate intake. [8,9]

Intensive insulin therapy should not be avoided to prevent weight gain; sustained hyperglycemia is not an acceptable weight-control strategy. The practical challenge is to reduce avoidable overinsulinization while preserving target glucose. Mismatch may arise from overly aggressive basal dosing, stacking of correction boluses, inaccurate carbohydrate estimation, delayed postprandial dosing, or routine overtreatment of hypoglycemia. Pubertal dose escalation may persist after the precipitating growth phase, and high-fat meals can produce delayed hyperglycemia that encourages repeated corrections and subsequent lows. [12,13]

3.2 Adipose dysfunction and systemic insulin resistance

Adipose expansion becomes pathogenic when storage capacity is exceeded or fat is preferentially visceral. Enlarged adipocytes release more free fatty acids and proinflammatory mediators and less protective adipokine signaling. Lipid deposition in liver and skeletal muscle interferes with insulin signaling, reduces glucose uptake, and increases hepatic glucose production. In T1D, these processes occur on a background of absent endogenous insulin regulation, so compensation depends on larger exogenous doses that may further support weight gain. [9,10]

Clamp-based studies demonstrate that adolescents with T1D can have peripheral, hepatic, and adipose insulin resistance even when they are not obese, with additional impairment as adiposity increases. Poor glycemic control, chronic inflammation, oxidative stress, dyslipidemia, and altered growth-hormone signaling may contribute. Thus, insulin resistance in T1D is not synonymous with obesity, and obesity is not the sole explanation for a high insulin dose. Injection-site problems, missed doses, insulin degradation, intercurrent illness, glucocorticoid exposure, and inaccurate dosing must remain in the differential diagnosis. [10,11]

3.3 Puberty, sex, and developmental biology

Puberty produces a physiologic fall in insulin sensitivity driven partly by growth hormone, insulin-like growth factor 1, and sex steroids. Youth with intact beta-cell function compensate by increasing insulin secretion; those with T1D require larger administered doses. Appetite, autonomy, sleep timing, body composition, and activity also change during adolescence. Girls generally accrue a higher fat mass, whereas boys gain more lean mass, helping explain sex differences in BMI trajectories and insulin requirements. [6,11]

Developmental context is therefore indispensable when interpreting rising weight or dose. A transient dose increase during a growth spurt differs from persistent escalation accompanied by central adiposity, hypertension, or dyslipidemia. Menstrual irregularity, hyperandrogenism, or polycystic ovary syndrome may further intensify insulin resistance in adolescent girls. Conversely, restrictive eating, excessive exercise, or insulin omission can coexist with a normal or high BMI and must not be missed by an obesity-focused consultation. [7,9]

3.4 Double diabetes and the bidirectional cycle

The “accelerator hypothesis” proposes that increased body mass and insulin resistance accelerate beta-cell stress and may advance clinical expression of autoimmune diabetes in susceptible children. Evidence supports biologic plausibility but does not establish obesity as a universal cause of T1D. After

diagnosis, a clearer bidirectional cycle can operate: adiposity raises insulin demand; higher doses and hypoglycemia prevention behaviors favor positive energy balance; further weight gain then increases demand. Genetic susceptibility, family environment, and social determinants influence where a child enters and how rapidly the cycle progresses. [6,8]

Diabetes technology can interrupt or reinforce this cycle. Continuous glucose monitoring (CGM) and automated insulin delivery can reduce glycemic variability and fear of hypoglycemia, permitting safer activity and fewer rescue carbohydrates. However, automation cannot fully compensate for inaccurate meal announcements, energy-dense diets, or overtreatment of predicted lows. Improved glycemia may also reduce urinary calorie loss. Weight outcomes therefore depend less on the device itself than on settings, education, eating patterns, and the behavioral response to data. [12,15]

4. Clinical Assessment

Assessment should establish the pattern and drivers of weight gain, quantify current metabolic risk, and identify barriers to safe treatment. It should be longitudinal, family-centered, and free of blame. Weight-related language deserves explicit permission, and the clinical goal should be framed as improved health, strength, glucose stability, and quality of life rather than pursuit of a particular appearance. Table 1 summarizes a practical pediatric assessment. [1,32]

Table 1. Structured assessment of obesity in a child or adolescent with type 1 diabetes

Domain	What to assess	Interpretation and cautions
Growth and adiposity	Plot weight, height, BMI percentile and z score over time; document growth velocity and pubertal stage.	Consider waist-to-height ratio or waist circumference when feasible; body composition testing is reserved for selected clinical or research questions.
Diabetes and insulin	Review diabetes duration, total daily dose (units/kg/day), basal proportion, bolus timing, missed doses, injection/pump sites, hypoglycemia treatment, and recent steroid exposure.	A high dose is a clue, not a diagnosis of insulin resistance; first exclude delivery, adherence, illness, and dosing problems.
Glucose profile	Assess HbA1c together with CGM time in range, time above range, time below range, variability, and recurrent postprandial or nocturnal patterns.	Interpret in relation to age, individualized targets, hypoglycemia risk, and access to technology.
Cardiometabolic risk	Measure blood pressure correctly; assess fasting or nonfasting lipids as recommended, liver enzymes when indicated, and renal risk markers according to age and diabetes duration.	Evaluate suspected sleep apnea, metabolic dysfunction-associated steatotic liver disease, and polycystic ovary syndrome rather than using indiscriminate testing.
Lifestyle and environment	Explore food access, meal structure, sweetened beverages, activity, sedentary time, sleep, school demands, family routines, and neighborhood safety.	Identify diabetes-specific behaviors such as defensive snacking, exercise avoidance, or repeated overtreatment of sensor-predicted lows.
Psychological health	Screen for diabetes distress, depression, anxiety, weight stigma, binge eating, restriction, and intentional insulin omission.	Use confidential adolescent discussion and refer early when eating-disorder risk or unsafe insulin manipulation is suspected.

Note. Assessment frequency and laboratory thresholds should follow current pediatric diabetes and obesity guidance and be individualized to age, pubertal status, duration, and clinical findings. [1,11,17-19,22,24,25,32]

4.1 Anthropometry and body composition

BMI-for-age percentile and z score remain the practical screening standards, but serial values are more informative than an isolated classification. Height velocity must be reviewed because weight change during normal growth differs from disproportionate gain, and a falling height percentile may signal endocrine or systemic disease. Waist-to-height ratio can add information about central adiposity, although pediatric thresholds and measurement methods vary. Dual-energy X-ray absorptiometry,

magnetic resonance imaging, and research-grade body-composition techniques are not required routinely. [14,32]

4.2 Insulin sensitivity: what can and cannot be measured

The hyperinsulinemic-euglycemic clamp is the reference method for quantifying insulin sensitivity but is resource intensive and largely confined to research. Estimated glucose disposal rate (eGDR), derived from readily available clinical variables such as waist circumference or BMI, blood pressure or hypertension status, and HbA1c, correlates with clamp measurements and can stratify risk. Yet equations differ, cutoffs are not universally validated in children, and eGDR should support—not replace—clinical judgment. [16,17]

Homeostatic model assessment of insulin resistance (HOMA-IR) is commonly used in people who produce endogenous insulin, but it is generally inappropriate in established T1D because fasting insulin reflects injected insulin, absorption kinetics, assay cross-reactivity, and timing rather than beta-cell secretion. C-peptide-based approaches may have limited research relevance early after diagnosis in those with residual function. In routine care, the most useful signals are the trajectory of weight and central adiposity, total insulin dose in context, glycemic patterns, blood pressure, lipids, and associated conditions. [11,18]

4.3 Glycemia and insulin-delivery data

HbA1c alone cannot describe the interaction between weight and diabetes management. CGM should be examined for time in range, time above and below range, glucose variability, overnight stability, and the timing of excursions relative to meals and activity. Total daily insulin, units per kilogram, basal fraction, correction frequency, and automated-delivery logs can reveal excess basal insulin, missed meal boluses, recurrent rescue eating, or settings that no longer match physiology. Glycemic targets should remain ambitious but individualized, with safety prioritized during treatment changes. [19,20]

4.4 Cardiometabolic and psychosocial comorbidity

Blood pressure and lipid assessment are central because obesity and T1D independently increase vascular risk. Screening for albuminuria and retinopathy should follow diabetes duration and age guidance; obesity does not justify delaying established surveillance. Liver enzymes and evaluation for metabolic dysfunction-associated steatotic liver disease are appropriate when obesity, dyslipidemia, hepatomegaly, or other risk factors are present. Symptoms should prompt assessment for obstructive sleep apnea, and adolescent girls with menstrual irregularity or hyperandrogenism warrant evaluation for polycystic ovary syndrome. [22-24]

Psychological assessment is equally important. Weight stigma from families, peers, or clinicians can worsen distress and disengagement. Some adolescents restrict insulin to influence weight, whereas others experience binge eating, loss-of-control eating, or persistent fear of hypoglycemia. These behaviors can coexist and may be concealed during a standard dietary history. Private, nonjudgmental questioning and validated screening tools are preferable to assumptions based on HbA1c or body size, with timely collaboration among diabetes, mental-health, and eating-disorder professionals. [25,32]

5. Clinical Consequences

The relation between obesity and glycemic control is complex. Some pediatric cohorts associate higher adiposity with higher HbA1c or insulin dose, whereas others identify children with overweight who maintain excellent glycemia. Reverse causation also matters: glycosuria and insulin omission can lower weight while increasing HbA1c, and meticulous insulin replacement can increase weight while improving glucose. Clinicians should therefore avoid treating BMI as a surrogate for adherence or glycemic quality. [20,21]

The more consistent signal is clustering of cardiometabolic risk. Youth with T1D and overweight more often show hypertension, adverse lipid profiles, lower insulin sensitivity, and features of metabolic syndrome. Pediatric metabolic-syndrome definitions vary and should not become a barrier to action; each abnormal component is clinically relevant. Central adiposity, low eGDR, dyslipidemia, and elevated blood pressure may identify higher-risk youth even when HbA1c is near target. [22,23]

Over time, insulin resistance and excess adiposity may compound the vascular burden of hyperglycemia, contributing to albuminuria, retinopathy, endothelial dysfunction, and premature cardiovascular disease. Evidence linking childhood obesity directly to individual hard outcomes remains limited because events occur decades later, but risk-factor tracking from adolescence and adult T1D data support early prevention. Screening and treatment of blood pressure and lipid abnormalities should proceed according to pediatric T1D guidance rather than waiting for weight loss. [9,24]

Obesity also affects daily life: larger insulin doses, variable absorption, challenging infusion-set placement, exercise-related glucose swings, sleep-disordered breathing, orthopedic discomfort, and reduced participation in activity can increase burden. Repeated focus on weight may damage self-esteem or precipitate unsafe behavior. Meaningful outcomes therefore include less hypoglycemia-related eating, improved fitness and sleep, lower insulin burden, better time in range, and improved well-being—not only change on the scale. [7,15]

6. Therapeutic Challenges and Practical Management

Treatment has four simultaneous obligations: maintain safe glycemia, support linear growth and puberty, reduce cardiometabolic risk, and protect psychological health. A family-based, multicomponent plan is preferred, using person-first language and shared decisions. Rapid weight-loss prescriptions, severe carbohydrate restriction, and intentional insulin underdosing are unsafe. For many growing children, initial success may be weight stabilization while height increases, whereas adolescents with severe obesity may require a more intensive pathway. Table 2 summarizes the evidence and major cautions. [1,32]

Table 2. Therapeutic options and evidence gaps in pediatric type 1 diabetes complicated by obesity

Intervention	Potential role/evidence	Key challenge
Insulin and technology	Foundational; optimize basal/bolus balance, timing, sites, CGM alerts, and automated-delivery settings.	Prevents avoidable overinsulinization and rescue eating, but insulin must never be restricted for weight control.
Nutrition therapy	Recommended; individualized, family-based pattern emphasizing minimally processed foods, fiber, appropriate portions, and structured meals.	Must preserve adequate carbohydrate for growth and safe activity; avoid rigid rules and stigma.
Physical activity	Recommended; combine aerobic, resistance, and routine movement with individualized insulin/carbohydrate planning.	Hypoglycemia and delayed lows require preparation; fear of hypoglycemia should be treated as a modifiable barrier.
Metformin	Trials show modest reductions in insulin dose or adiposity in some adolescents, without sustained HbA1c improvement.	Not routine; gastrointestinal adverse effects are common. Consider only selected insulin-resistant adolescents with explicit goals.
GLP-1 receptor agonists	Effective for general adolescent obesity; adult T1D studies suggest weight and insulin-dose reductions.	Pediatric T1D evidence is insufficient; use would be off-label for T1D and requires specialist oversight, insulin adjustment, and ketone education.
SGLT2 inhibitors	Can lower glucose and weight in adults with T1D.	Clinically important risk of euglycemic ketoacidosis; not routine or approved treatment for pediatric T1D.
Metabolic surgery	May be considered under general pediatric severe-obesity criteria in exceptional cases.	Very limited T1D evidence; requires an experienced multidisciplinary center and lifelong nutritional and diabetes follow-up.

Note. No adjunct replaces insulin. Regulatory status varies by country; clinicians should verify current labeling and use pediatric endocrinology and obesity expertise for any pharmacologic or surgical escalation. [12,26-37]

6.1 Insulin optimization and diabetes technology

Insulin review is the first therapeutic step. Basal insulin should maintain stability during fasting rather than cover habitual snacks or missed boluses. Meal dosing should incorporate carbohydrate amount and, when needed, fat and protein effects; premeal timing can reduce postprandial excursions and corrective stacking. Infusion and injection sites require inspection for lipohypertrophy. Hypoglycemia should be treated with measured fast-acting carbohydrate and reassessed, limiting the common cycle of overtreatment, rebound hyperglycemia, and correction insulin. [12,19]

CGM and automated insulin delivery can facilitate safer weight-management behaviors by reducing severe lows, showing responses to meals and exercise, and permitting more precise adjustments. Alerts

should be individualized to avoid alarm-driven eating. Exercise modes, temporary targets, or insulin reductions should be planned rather than paired automatically with extra calories. Technology data should be reviewed with curiosity rather than judgment; the objective is to identify repeatable patterns that reduce both glycemic variability and unnecessary energy intake. [12,21]

6.2 Nutrition

Medical nutrition therapy should retain the principles of pediatric T1D care while creating a sustainable energy balance. Priorities include water instead of sugar-sweetened beverages, vegetables and fruit, whole grains and legumes, adequate protein, unsaturated fats, regular meals, and portion awareness for energy-dense foods. Carbohydrate counting remains necessary but does not by itself ensure dietary quality. Family meals, consistent access to healthy food, school planning, and involvement of a diabetes-experienced dietitian are more useful than assigning responsibility solely to the child. [15,26]

Very-low-carbohydrate or ketogenic diets are not routine pediatric T1D obesity therapy because of concerns about hypoglycemia, ketosis, nutrient adequacy, lipids, growth, and disordered eating. Modest changes that can be measured—fewer sweet drinks, less unplanned grazing, more fiber, and accurate treatment of lows—often offer safer leverage. Dietary goals should be revisited using glucose patterns, hunger, growth, cultural preferences, affordability, and family capacity rather than a universal calorie prescription. [26,32]

6.3 Physical activity, sedentary behavior, and sleep

Activity improves cardiorespiratory fitness, insulin sensitivity, blood pressure, lipid profiles, and mental health even when BMI changes little. Aerobic and resistance exercise should be combined with routine movement and reduced sedentary time. Because insulin cannot fall physiologically at exercise onset, glucose direction, active insulin, recent food, intensity, and duration must guide carbohydrate intake and dose adjustments. Individual plans should cover pre-exercise assessment, fast carbohydrate access, identification, hydration, and prevention of delayed nocturnal hypoglycemia. [27,28]

Youth with T1D are, on average, less active and more sedentary than peers, making fear of hypoglycemia an important treatment target rather than a reason to abandon exercise. Families benefit from rehearsing different strategies for spontaneous play, planned training, competition, and resistance activity. Sleep duration and regularity should also be assessed because insufficient sleep worsens appetite regulation, insulin sensitivity, and self-management and may indicate obstructive sleep apnea in a child with snoring or daytime somnolence. [15,28]

6.4 Psychological and behavioral support

Behavioral treatment is most effective when goals are concrete, collaborative, and compatible with diabetes demands. Motivational interviewing, problem solving, environmental restructuring, and family support can address food routines, activity, sleep, and device use. Clinicians should avoid moral language about glucose or food and should monitor whether frequent weighing or dietary tracking increases distress. Suspected binge eating, restrictive eating, body-image disturbance, depression, or insulin omission requires prompt specialized assessment; weight loss is never an adequate justification for unsafe insulin behavior. [25,32]

6.5 Adjunctive pharmacotherapy

Metformin is the most studied adjunct in overweight adolescents with T1D. Randomized evidence shows that it does not produce sustained improvement in HbA1c, although some participants experience modest reductions in insulin dose or adiposity. Gastrointestinal adverse effects are frequent. Routine addition solely to lower HbA1c is therefore not supported. A time-limited specialist trial may be considered for a carefully selected adolescent with marked insulin resistance or progressive adiposity, with predefined outcomes and discontinuation if benefit is absent. [29,30]

Glucagon-like peptide-1 (GLP-1) receptor agonists reduce appetite and produce substantial weight loss in adolescents with obesity who do not have T1D. Semaglutide and liraglutide trials establish efficacy for general pediatric obesity, but participants with T1D were not the basis for those conclusions. Extrapolation is therefore uncertain: delayed gastric emptying may complicate prandial insulin timing,

nausea may reduce carbohydrate intake unpredictably, and insulin reductions that are too rapid can increase ketosis risk. [33,34]

Adult T1D trials of liraglutide have shown weight and insulin-dose reductions but also safety signals, including more symptomatic hypoglycemia or hyperglycemia with ketosis at higher doses. Pediatric T1D-specific randomized data remain inadequate. If a GLP-1 receptor agonist is used under an obesity indication in a young person with T1D, this should occur through shared decision-making with experienced specialists, careful insulin titration, sick-day and ketone education, nutritional monitoring, and explicit recognition that the T1D application is not established routine care. [31,35]

Sodium-glucose cotransporter 2 (SGLT2) inhibitors can improve glucose and reduce weight in adults with T1D, but urinary glucose loss permits ketoacidosis at only modestly elevated glucose concentrations. Illness, fasting, low-carbohydrate intake, pump interruption, dehydration, exercise, and insulin reduction increase risk. Because euglycemic presentations may delay recognition and pediatric evidence is insufficient, these agents should not be used routinely in children or adolescents with T1D. Apparent glycemic or weight benefits do not outweigh an unmitigated ketoacidosis hazard. [1,36]

6.6 Metabolic and bariatric surgery

Metabolic surgery is not a treatment for T1D itself, but it may be considered for exceptional adolescents who meet established severe-obesity criteria and have major comorbidity despite intensive nonsurgical care. Published T1D experience is limited largely to small reports: substantial weight loss and lower insulin requirements may occur, while glycemic control can remain difficult and the risks of hypoglycemia, ketoacidosis, micronutrient deficiency, and altered carbohydrate absorption persist. Evaluation belongs in a high-volume multidisciplinary center with pediatric diabetes, obesity, surgical, nutritional, and mental-health expertise. [32,37]

6.7 A staged multidisciplinary pathway

A pragmatic pathway begins by confirming the growth trajectory, identifying insulin-delivery problems, and treating acute psychological or eating-disorder risk. The team then selects two or three feasible behavioral targets while optimizing insulin and CGM settings. Cardiometabolic abnormalities are treated directly and reassessed at defined intervals. Lack of progress should trigger escalation in support intensity and evaluation for secondary contributors, not blame. Adjunct medication or surgery is reserved for carefully selected patients after foundational care, with outcomes encompassing glycemia, insulin dose, fitness, comorbidities, quality of life, and safety. [7,31]

7. Research Gaps and Future Directions

Major gaps include a pediatric consensus definition of clinically meaningful insulin resistance in T1D, validation of eGDR across ages and ethnicities, and longitudinal studies connecting childhood adiposity phenotypes with adult vascular outcomes. Trials should measure body composition, central and hepatic fat, CGM outcomes, hypoglycemia treatment burden, insulin dose, cardiovascular markers, and patient-reported outcomes rather than relying only on BMI and HbA1c. Recruitment should include youth from diverse socioeconomic settings, where treatment burden and obesity risk often intersect. [8,16]

Pediatric T1D-specific studies are particularly needed for GLP-1 receptor agonists and emerging obesity medications, including protocols for insulin adjustment and ketone surveillance. Technology algorithms could potentially account for exercise, meal composition, appetite-modifying treatment, and changing insulin sensitivity, but unintended weight effects require evaluation. Implementation research should test integrated diabetes-obesity clinics and stigma-sensitive family interventions, with safety monitoring for disordered eating and inequitable access. [6,9]

8. Conclusion

Obesity has become a common and consequential modifier of pediatric T1D. Its clinical importance lies not in body size alone but in the interaction of adiposity, pubertal physiology, exogenous insulin, glycemic behaviors, and accumulating cardiometabolic risk. Assessment should follow longitudinal growth, central adiposity, insulin and CGM patterns, vascular risk factors, associated conditions, and psychological health. Clamp testing is impractical for routine care, eGDR can aid risk stratification, and

HOMA-IR is generally unsuitable once insulin is exogenous. Management should preserve intensive insulin replacement while reducing avoidable overinsulinization, improving food quality, enabling safe activity, and addressing sleep and distress. Metformin has limited selected use; GLP-1 receptor agonists remain insufficiently studied in pediatric T1D; SGLT2 inhibitors present an important ketoacidosis hazard; and surgery is reserved for exceptional severe obesity. The strongest current strategy is coordinated, family-centered care that pursues glycemic safety and cardiometabolic health together without stigma or unsafe weight-control practices.

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