The Intersection of Type 1 Diabetes and Disordered Eating: A Comprehensive Literature Review of Clinical Manifestations, Epidemiological Dynamics, and Multidisciplinary Management of Insulin Omission (Diabulimia)

Clinical Conceptualization, Terminology, and Pathophysiology

The co-occurrence of type 1 diabetes mellitus (T1D) and eating disorders (ED) represents a complex, highly dangerous hybrid pathology situated at the boundary of endocrinology and psychiatry.[1] Managing T1D requires continuous daily self-care, including frequent blood glucose monitoring, precise carbohydrate counting, and calculated administration of exogenous insulin.[3] However, this intensive focus on dietary restriction, portion sizes, and metabolic metrics can act as an iatrogenic catalyst for the development of disordered eating behaviors (DEB).[4]

Among these behaviors, the deliberate restriction or omission of insulin to control weight is a unique, highly dangerous purging practice.[6] Commonly referred to as "diabulimia" in popular literature [8], this phenomenon is also characterized as insulin-related disordered eating behavior (IRDEB) [11], eating disorder-diabetes mellitus type 1 (ED-DMT1) [10], or type 1 diabetes disordered eating (T1DE).[10] Although diabulimia is not officially recognized as a discrete diagnostic category in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) or the International Classification of Diseases (ICD-10), clinical consensus treats insulin omission as a specialized compensatory purging mechanism within existing diagnostics like bulimia nervosa, anorexia nervosa, or other specified feeding or eating disorders (OSFED).[1]

Terminology	Clinical Definition	Pathophysiological and Behavioral Rationale	Source Citations
Insulin-Related Disordered Eating	Umbrella term for all disordered eating	Avoids diagnostic confusion with	[11]

Behavior (IRDEB)	behaviors involving the manipulation of insulin doses.	standard eating disorders and encompasses diverse clinical presentations.	
Active IRDEB	Deliberate manipulation of insulin doses with the goal of achieving hypoglycemia or hyperglycemia to serve disordered eating motivations.	Encompasses active purging behaviors; directly links metabolic manipulation with eating pathology.	[11]
Passive IRDEB	Disengagement from appropriate diabetes self-care practices (e.g., failure to correct high glucose levels) driven by disordered eating motivations.	Avoids blame-associated connotations of "intentionality" while recognizing clinical avoidance patterns.	[11]
Insulin Restriction	Active or passive removal of insulin from a prescribed dose for weight control.	Can be partial or total; avoids the clinical connotations of bulimia nervosa.	[11]
Insulin Inflation	Active or passive addition of insulin beyond the prescribed dose to permit binge eating or compensate for perceived overeating.	Avoids classifications like Factitious Disorder while capturing unique diabetic purging cycles.	[11]

Diabulimia (ED-DMT1 / T1DE)	Colloquial and clinical term for intentional insulin omission to influence body shape, size, or weight.	Portmanteau of diabetes and bulimia; highly recognized in patient support communities and clinical settings.	[9]

The physiological mechanism of diabulimia is driven by deliberate metabolic decompensation.[7] Restricting or omitting insulin deprives the body of the hormone required to transport glucose into skeletal muscle and adipose tissue for cellular metabolism.[14] This lack of insulin leads to severe hyperglycemia, often exceeding $600\ \mathrm{mg/dL}$ ($33\ \mathrm{mmol/L}$).[10]

To clear this extreme glucose load, the kidneys excrete large amounts of glucose in the urine (glycosuria).[7] This process dumps massive quantities of calories, resulting in rapid weight loss.[7] Concurrently, the lack of cellular glucose triggers a starvation-like, catabolic state.[7] The body rapidly breaks down muscle proteins and fatty acids to sustain baseline cellular function.[1] This catabolic breakdown produces acidic ketone bodies, which accumulate in the bloodstream and can lead to diabetic ketoacidosis (DKA).[7]

A significant clinical irony is that many patients learn this purging mechanism directly from their initial diabetes education.[9] During education at diagnosis, clinicians explain that insulin deficiency causes weight loss through glycosuria and ketone-driven fat breakdown.[9] For patients with underlying body dissatisfaction, this clinical explanation inadvertently provides a guide on how to purge calories through insulin manipulation.[9]

Epidemiology, Demographics, and Vulnerability Profiles

Epidemiological studies demonstrate that individuals with T1D face a significantly higher risk of developing disordered eating behaviors and clinical eating disorders than their peers without diabetes.[2] Large-scale cohort evaluations suggest that approximately one-third of adolescents and young adults with T1D experience disordered eating.[14]

A notable gender disparity exists within this population.[7] Adolescent females with T1D are twice as likely to develop eating disorders as non-diabetic females.[7] Furthermore, the practice of deliberate insulin omission is concentrated primarily among adolescent girls and young adult women.[1]

Population Cohort	Key Epidemiological Metrics	Clinical Findings and Patterns	Source Citations

Pediatric & Adolescent T1D Cohorts	Prevalence of eating disorders ranges from **20.8% to 48.0%**; pooled meta-analytic prevalence is **11.0%**.	Disordered eating behaviors are up to **5 times more prevalent** in youth with T1D than in peers without diabetes.	[14]
Adolescent Females with T1D	Approximately **10.3%** admit to active insulin omission; **7.4%** omit specifically to lose weight.	Rates of eating disorders range from **20.0% to 50.0%**; bulimia nervosa is the most common clinical diagnosis.	[7]
Adolescent Males with T1D	Approximately **1.4%** admit to insulin omission; **9.0%** experience general eating disorders.	Considerably lower rates of active purging, though up to **40.0%** report skipping an insulin injection at least once.	[10]
Adult T1D Outpatient Cohorts	Prevalence of positive DEPS-R screens ($\geq$) is **25.0% to 30.1%**.	Women measure blood glucose more frequently than men, yet show higher rates of positive screens.	[22]
Comparative Cohorts (T1D vs. T2D on intensive insulin)	Positive DEPS-R screens found in **25.0%** of T1D vs. **55.0%** of T2D patients.	BMI is similar between high-risk and low-risk T1D groups, but is significantly higher in high-risk T2D groups.	[23]

This heightened vulnerability is driven by developmental trajectories and clinical management requirements.[6] Prior to a T1D diagnosis, patients often experience severe, unexplained weight

loss due to metabolic decompensation.[7] Initiating intensive insulin therapy quickly restores cellular glucose uptake and hydration, leading to rapid weight gain that often exceeds pre-diagnosis levels.[6]

For adolescents transitioning through vulnerable developmental stages, this rapid increase in body weight can trigger severe body image distress and a drive for thinness.[6] This distress is further compounded during puberty, when female students experience a natural rise in insulin requirements that can accelerate weight gain.[5]

Psychological comorbidities further increase this vulnerability.[6] Rates of major depressive disorder and generalized anxiety are doubled in T1D populations.[6]

Among girls with T1D, those diagnosed with depression score significantly higher on the Eating Disorder Examination than those without depression (75% vs. 45%).[6] Additionally, generalized anxiety is highly prevalent, with approximately 21% of children and adolescents with T1D screening positive.[6]

Clinical Consequences, Prognosis, and Multi-System Failure

The metabolic consequences of prolonged or intermittent insulin omission are severe, leading to significant multi-system complications and a highly compromised prognosis.[7] Physically, the patient experiences an immediate rise in glycated hemoglobin (HbA_{1c}) levels, extreme glycemic variability, and clinical dehydration.[7]

In the short term, insulin deficiency causes unregulated lipolysis, producing an accumulation of acidic ketone bodies in the bloodstream.[7] This state rapidly progresses to diabetic ketoacidosis (DKA), a life-threatening medical emergency requiring immediate hospitalization.[7]

In the long term, chronic hyperglycemia accelerates microvascular and macrovascular damage.[7] Complications develop much earlier in patients who engage in insulin omission compared to those with standard T1D management.[15]

Clinical Complication	Pathophysiological Mechanism	Clinical Timeline to Development	Prognosis & Associated Mortality Risk	Source Citations
Diabetic Ketoacidosis (DKA)	Severe insulin deficiency causes uncontrolled breakdown of fatty acids, leading to	Acute, rapid onset; risk of recurrent hospitalizations is tripled in patients with concurrent	In-hospital mortality rates of DKA range from 13.0% to 30.0%, with significantly higher risk in	[7]

	metabolic acidosis and ketonemia.	T1D and ED.	pediatric populations.	
Retinopathy	Prolonged hyperglycemia damages retinal microvasculature, leading to microaneurysms, hemorrhage, visual occlusion, and blindness.	Accelerates rapidly: develops on average in **3.4 years** in patients practicing insulin omission, compared to **11.5 years** in those with T1D alone.	Threefold increase in development risk; linked to a severe reduction in long-term quality of life.	[7]
Neuropathy	Persistent hyperglycemia causes microvascular damage to peripheral nerves, resulting in sensory loss, pain, and limb amputation.	Disproportionately high: symptomatic in **55.5%** of active insulin restrictors in clinical cohort evaluations.	The 3-year survival rate for diabetic patients once neuropathy complications necessitate major clinical intervention is less than 17.0%.	[15]
Nephropathy	Glomerular hyperfiltration and structural microvascular damage lead to renal impairment and end-stage kidney failure.	Gradual progression, but heavily accelerated by frequent periods of metabolic decompensation.	Leading cause of mortality in T1D (lifetime incidence of ~50.0%); longitudinal cohorts show a **32.8%** direct mortality rate from nephropathy.	[7]

Cerebral Oedema	Rapid shifting of extracellular brain fluid, often precipitated during the rapid clinical correction of severe hyperglycemia or DKA.	Acute emergency occurring during intensive rehydration and insulin administration.	Extremely high mortality risk (**20.0% to 25.0%**); accounts for **60.0% to 90.0%** of all clinical deaths during pediatric DKA.	[15]
Overall Mortality	Multi-system failure resulting from accelerated microvascular disease, recurrent DKA, or infectious complications.	Elevated risk is evident over long-term observation.	**3.2-fold** increase in baseline mortality; up to **6-fold** mortality increase in adolescent cohorts with comorbid ED.	[7]
Life Expectancy Reduction	Combined burden of microvascular failure and acute metabolic crises.	Mean age of death is **45 years** in patients practicing insulin restriction, compared to **58 years** in those with standard T1D.	Direct reduction of **13 years of life**; severity of loss is directly correlated with the frequency of purging behaviors.	[8]

Behavioral Drivers, Technology Misuse, and Functional Nuances

The behavioral landscape of insulin omission is multi-faceted and extends beyond weight and shape concerns.[13] Qualitative research and clinical assessments identify several distinct behavioral drivers that maintain this dangerous practice:

- **Weight Control and Purging:** This is the primary driver originally identified by Stuart Brink, where patients learn that skipping insulin allows them to binge eat without gaining

weight.[1]

- **Deliberate Self-Harm:** Insulin omission is also used as a method of self-injury.[15] One study noted that self-destructive behavior was the primary cause in 28.0% of insulin omission cases, compared to 15.5% driven primarily by weight loss.[15]
- **Anxiety and Injection Phobias:** Fear of hypoglycemia and injection-related anxiety are prominent causes of insulin omission, particularly in pediatric and younger adolescent populations.[15]
- **Diabetes Burnout:** The relentless, 24/7 demands of T1D self-care can lead to physical and mental exhaustion, causing patients to disengage from treatment and passively omit insulin.[13]

Modern diabetes technologies, such as continuous glucose monitors (CGM) and continuous subcutaneous insulin infusion (CSII) pumps, can be a double-edged sword in this population.[5] While these advances offer improved glycemic control and lifestyle flexibility, they also introduce new opportunities for misuse.[16]

Insulin pumps allow for discrete and precise down-titration of basal rates or boluses, enabling patients to maintain elevated blood glucose levels with minimal effort.[16] Similarly, CGMs provide real-time glucose data, which some patients use to "safely" maintain hyperglycemia just below the threshold of severe DKA.[16]

Furthermore, the continuous presentation of glucose data around mealtimes can trigger food restriction to avoid postprandial excursions, while the constant use of carbohydrate databases can foster obsessive eating patterns.[16] Clinicians must also be aware of the potential misuse of adjunctive medications like amylin analogs (Symlin), which may be abused for their appetite-suppressant side effects.[16]

Screening Barriers and Diabetes-Specific Assessment Tools

Detecting disordered eating in patients with T1D is challenging, as behaviors are often well hidden and denied.[6] Clinicians must remain alert for key physical and biochemical markers of insulin omission:

- A persistently elevated, unexplained HbA_{1c} level.[14]
- High glycemic variability, characterized by unexplained fluctuations in blood glucose or wide shifts in continuous glucose data.[14]
- Recurrent, unexplained episodes of DKA requiring emergency admission.[14]
- Frequent, rapid fluctuations in body weight.[28]
- Secondary laboratory findings, such as iron deficiency or abnormal lipid profiles.[18]

Standard psychiatric screening tools, such as the Eating Disorder Examination Questionnaire (EDE-Q) or the SCOFF, are highly problematic in this population.[26] These general tools can over-pathologize behaviors that are clinically necessary for safe diabetes self-care, such as tracking carbohydrate intake, planning meals, and paying close attention to body weight.[26]

Conversely, they completely fail to capture diabetes-specific behaviors, such as the purging mechanism of insulin restriction.[30]
To address these limitations, diabetes-specific screening instruments and psychosocial scales have been validated to assess disordered eating and related stressors in this population:

Assessment Instrument	Target Domain & Description	Clinical Application & Scoring Metrics	Source Citations
Diabetes Eating Problem Survey – Revised (DEPS-R)	16-item self-report screening tool designed specifically to detect disordered eating behaviors in T1D.	Recommended for routine screening starting at age 10–12; a score $\geq$ indicates clinically significant risk.	[18]
Diabetes Eating Problem Survey (DEPS)	Original 28-item self-report measure of disordered eating behaviors in individuals with T1D.	Validated against clinical diagnoses in adult populations; updated by the DEPS-R for modern regimens.	[29]
Diabetes Distress Scale (DDS-2)	2-item scale used to evaluate the daily emotional burden and psychosocial stress of managing diabetes.	A score $\geq$ indicates high diabetes distress; highly correlated with insulin omission behaviors.	[31]
Diabetes Family Conflict Scale	Evaluates the level of family conflict surrounding 19 specific diabetes self-management tasks.	Completed by both youth and parents; higher scores indicate greater conflict, a known risk factor for DEB.	[29]
Blood Glucose	8-item survey	Higher scores	[29]

Monitoring Communication Questionnaire	assessing negative affect and emotional distress related to blood glucose monitoring.	indicate a greater degree of negative affect and anxiety surrounding metabolic tracking.	
Diabetes Quality of Life for Youth (DQOLY) - Eating Subscale	3-item subscale assessing dietary restriction, hiding diabetes to eat forbidden foods, and social eating limitations.	Used to measure the impact of diabetes management on psychosocial quality of life and peer interactions.	[29]
Problem Areas in Diabetes – Parent version (PAID-P)	20-item survey measuring the perceived burden of care experienced by parents of children with T1D.	Higher scores indicate greater parental burden, which can contribute to family conflict and adolescent distress.	[29]
Gold Score	Clinical screening tool used to assess a patient's awareness of hypoglycemia symptoms.	Evaluates hypoglycemia frequency, severity, and silent episodes; critical for identifying high-risk patients.	[3]

Despite clear international guidelines recommending annual screening, routine screening remains highly underutilized.[18] A review of pediatric T1D clinics indicates that most teams rely on unstructured, routine clinical interviews as their primary screening method, with fewer than 10% utilizing validated, diabetes-specific screening tools.[18] The primary barriers to implementing these standardized screens include a lack of clinical time during routine visits and a lack of staff confidence in using and interpreting screening tools (70% of clinics for each barrier).[18] Conversely, staff training in disordered eating has been identified as a key clinical enabler.[18]

Multi-Disciplinary Management, Treatment Models, and Systemic Disparities

Systemic Care Disparities

A critical barrier to treating concurrent T1D and eating disorders is the systemic fragmentation of healthcare services.[2] Nationwide epidemiological studies reveal a striking disparity: despite experiencing significantly higher risks of microvascular failure and mortality, T1D patients with eating disorders receive significantly *less* outpatient hospital treatment for their eating disorder than their peers without diabetes (a mean of 3.32 vs. 5.33 outpatient care visits per year).[2]
This treatment gap exists because standard eating disorder services often find diabetes too medically complex to manage safely, while standard diabetes clinics lack the psychological resources to address deep-seated eating pathology.[2] This fragmentation often leads to disengagement from care and poor clinical outcomes.[2]

The Multidisciplinary Team (MDT) and Psychological Care

To bridge this gap, clinical guidelines emphasize the necessity of an integrated, collaborative multidisciplinary team (MDT).[3] This team must maintain continuous communication to ensure that medical safety and psychological recovery are aligned.[8]
The core team includes an endocrinologist to oversee metabolic safety, a clinical psychologist or psychiatrist to deliver evidence-based psychological therapies, a diabetes specialist nurse to support practical self-management, and a specialist dietitian to guide nutritional rehabilitation.[3] Clinical psychologists are also crucial for training the wider MDT in motivational interviewing and early identification of vulnerable patients.[3]
Psychological therapy is the first-line treatment for eating disorders in T1D.[12] These therapies must be adapted to address the intersection of diabetes distress and eating pathology [12]:

- **Enhanced Cognitive Behavioral Therapy (CBT-E):** Highly effective for bulimic symptoms and purging behaviors, CBT-E is modified to target the cognitive overvaluation of weight and shape while addressing insulin omission as a form of purging.[28] It works to resolve the cognitive distortions surrounding insulin-induced weight gain and break the restriction-binge-purge cycle.[37]
- **Dialectical Behavior Therapy (DBT):** Particularly useful for patients experiencing severe emotional dysregulation, high distress intolerance, or using insulin omission as a form of deliberate self-harm.[36] DBT teaches mindfulness, distress tolerance, and emotion regulation skills to replace the functional use of insulin restriction during emotional crises.[36]
- **Family-Based Treatment (FBT):** Recommended for children and younger adolescents, FBT empowers parents to temporarily take over responsibility for meal planning and insulin administration, reducing the cognitive burden on the adolescent and preventing covert insulin restriction.[16]

Nutritional Rehabilitation and the "Insulin Edema" Barrier

Nutritional therapy in comorbid T1D and ED must balance medical safety with psychological recovery.[8] Standard, rigid diabetic diet plans that emphasize "forbidden foods" or strict

carbohydrate limits can exacerbate eating disorder pathology.[6]
Instead, guidelines recommend a weight-neutral, non-stigma-based approach, advocating for an **intuitive eating** framework.[33] This approach helps patients restore their interoceptive awareness of hunger and satiety cues, which are often severely disrupted by years of clinical dietary restraint and irregular glycemic patterns.[6]
A critical physiological challenge during early nutritional rehabilitation is the development of **insulin edema**.[8] When a patient who has been chronically omitting insulin restarts a therapeutic insulin regimen, the sudden restoration of physical hydration and glycemic stability causes acute, severe fluid retention.[8] This fluid retention leads to rapid swelling, physical discomfort, and bloating.[8]
To a patient with severe body dissatisfaction, this sudden change in physical appearance is often catastrophized as rapid fat accumulation, which can trigger panic and immediate relapse into insulin omission.[8] Proactive clinical education is essential to manage this risk.[8] Clinicians must prepare patients for the temporary nature of insulin edema, reassuring them that it represents water retention rather than adipose tissue accumulation.[8]

Integrated Service Models: The NHS T1DE Pilot Program

To address the systemic fragmentation of care, NHS England pioneered specialist, integrated Type 1 Diabetes Disordered Eating (T1DE) services.[10] Initiated as a pilot of two services in 2019, the program was expanded in 2022 to include five additional pilot services delivering a national integrated specification.[39]
The core clinical principle of the T1DE model is the integration of physical and mental health care through joint clinical appointments, integrated MDT meetings, and shared clinical supervision.[32] Data from rapid clinical evaluations of these integrated T1DE services, conducted by the NIHR-funded REVAL team at the University of Manchester, demonstrate strong evidence of clinical efficacy across both metabolic and psychological metrics at 6 months.[32]

- **Glycated Hemoglobin (HbA_{1c}):** Experienced a mean reduction of **0.97%** (dropping from a baseline of 11.2% to 10.2% at 6 months), which is highly clinically significant and dramatically lowers long-term microvascular risk.[32]
- **DEPS-R and DDS-2 Scores:** Showed significant improvements, reflecting a substantial reduction in active insulin restriction, food rules, and the daily emotional burden of diabetes distress.[32]
- **Anxiety and Depression (GAD7 & PHQ9):** Demonstrated statistically and clinically significant improvements, showcasing the benefit of delivering targeted psychotherapies directly alongside diabetes care.[32]

While these pilot services show strong clinical success, their long-term implementation faces significant structural challenges.[39] Key barriers include difficulties recruiting staff with specialized expertise in both diabetes and eating disorders, job insecurity due to fixed-term contracts, and funding barriers from local Integrated Care Boards (ICBs) once national pilot funding ends.[39]

Residential and Inpatient Treatment Models

For patients who are medically unstable, experiencing recurrent DKA, or unable to break the cycle of insulin omission in an outpatient setting, intensive inpatient or residential treatment is required.[8] Residential programs that integrate intensive medical monitoring, continuous nutritional rehabilitation, and daily structured psychotherapy show excellent clinical results.[33]
A clinical evaluation of a multidisciplinary residential treatment program for women with T1D and severe eating disorders demonstrated significant improvements across all metabolic and psychological dimensions over the course of treatment, while untreated cohorts showed no improvement or worsened over time.[33]

- **Glycated Hemoglobin (HbA_{1c}):** Decreased from an admission mean of **11.55%** (SD = 3.29) to a discharge mean of **9.00%** (SD = 1.99) ($p < .001$, effect size $\eta_p^2 = .59$).[33]
- **Fructosamine:** Decreased from an admission mean of **518.34** μ**mol** (SD = 151.95) to a discharge mean of **360.72** μ**mol** (SD = 69.04) ($p < .001$, effect size $\eta_p^2 = .67$).[33]
- **EDI-3 Bulimia Score:** Decreased from an admission mean of **48.07** (SD = 11.89) to a discharge mean of **30.93** (SD = 8.76) ($p < .001$, effect size $\eta_p^2 = .66$).[33]
- **EDI-3 Drive for Thinness:** Decreased from an admission mean of **46.35** (SD = 10.32) to a discharge mean of **30.52** (SD = 11.33) ($p < .001$, effect size $\eta_p^2 = .57$).[33]
- **EDI-3 Body Dissatisfaction:** Decreased from an admission mean of **45.28** (SD = 10.01) to a discharge mean of **36.55** (SD = 9.59) ($p < .001$, effect size $\eta_p^2 = .38$).[33]
- **General Psychological Maladjustment:** Decreased from an admission mean of **49.83** (SD = 12.04) to a discharge mean of **36.69** (SD = 11.62) ($p < .001$, effect size $\eta_p^2 = .53$).[33]

Conclusions

This literature review highlights the critical importance of recognizing and treating the intersection of type 1 diabetes and eating disorders. Deliberate insulin omission for weight loss—diabulimia—is a highly dangerous purging behavior that carries a 3.2-fold to 6-fold increase in mortality and reduces life expectancy by an average of 13 years.[7] Despite these severe risks, clinical detection is often delayed due to the secretive nature of the behavior and a lack of routine screening using diabetes-specific tools like the DEPS-R.[6]
To improve patient outcomes, clinical practice must move away from fragmented care.[2] Optimal management requires an integrated, multidisciplinary team that combines endocrinology, specialized psychological therapies (such as CBT-E and DBT), and weight-neutral nutritional support.[8] Data from specialized residential programs and integrated clinical pathways, such as the NHS T1DE pilots, demonstrate that combining physical and mental health care leads to

significant, long-term improvements in both metabolic control and psychological well-being.[32] Sustained clinical progress will depend on addressing systemic barriers to care, providing specialized training for healthcare professionals, and expanding access to these integrated treatment models.[8]

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