Synthesize the literature on the prevalence, clinical outcomes, and optimal management strategies for eating disorders concurrent with type 1 diabetes, specifically focusing on deliberate insulin omission for weight loss (diabulimia).

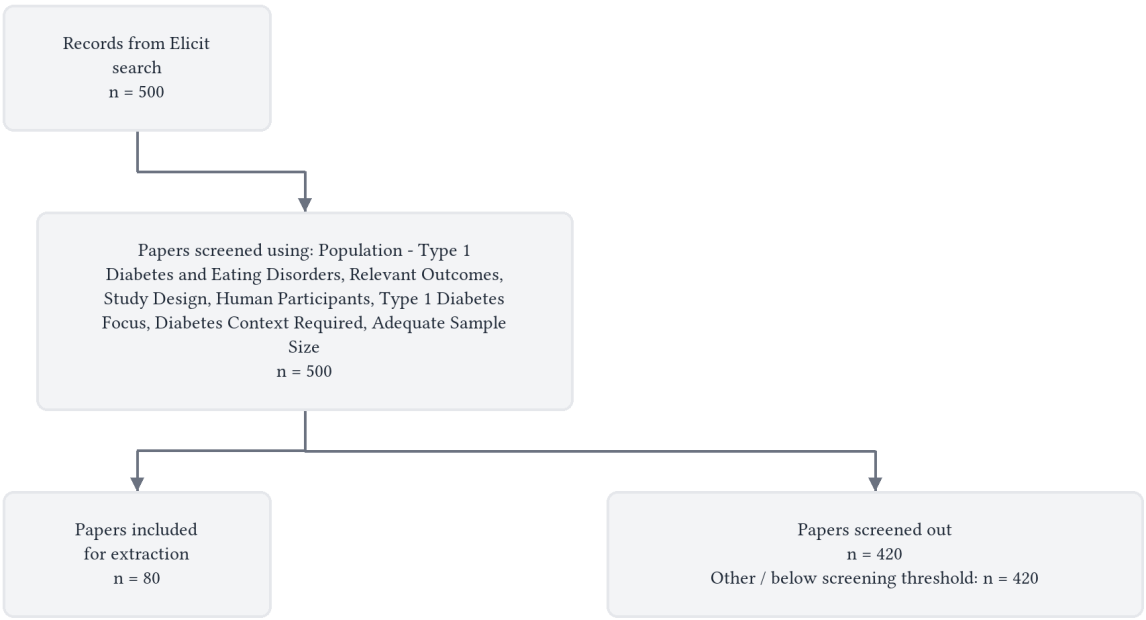
Eating disorders affect approximately twice as many individuals with type 1 diabetes as their non-diabetic peers, with deliberate insulin omission for weight loss occurring in roughly 10–40% depending on definition, and this behavior specifically drives the severe clinical consequences—including tripled diabetic ketoacidosis risk, accelerated microvascular complications, and up to sixfold increased mortality—yet evidence-based treatments remain scarce, with current best practice limited to routine screening using diabetes-specific tools like the DEPS-R and multidisciplinary care integrating endocrinology, dietetics, and mental health support.

Abstract

Eating disorders (EDs) and disordered eating behaviors (DEBs) are significantly more prevalent in individuals with type 1 diabetes (T1D) than in the general population, with large register-based cohorts from Scandinavia consistently showing approximately twofold elevated risk (HR 2.07, 95% CI 1.88–2.28) [1, 2]. Screening studies using diabetes-specific instruments such as the DEPS-R identify 21–48% of adolescents with T1D as having clinically significant DEBs [3–5], while the pooled prevalence of diabulimia specifically (deliberate insulin omission for weight loss) is estimated at 11% (95% CI 9–13%), with substantially higher rates in females [6]. Insulin omission prevalence ranges from 10% to 42% depending on definition breadth [5, 7], increases over time [8], and persists well into adulthood [9, 10]. Insulin manipulation is the central mediating behavior linking EDs to adverse T1D outcomes: diabetes-adapted questionnaires that capture insulin omission show significantly stronger associations with HbA1c (Hedges' g = 0.62) than generic instruments (g = 0.19) [11], and duration of severe insulin omission is the factor most closely associated with retinopathy and nephropathy [12]. Co-existing EDs confer more than triple the risk of diabetic ketoacidosis (HR 3.30, 95% CI 2.58–4.23) [13] and nearly sixfold increased mortality (HR 5.80, 95% CI 3.04–11.08) [13].

Key risk factors include female sex, higher BMI, depression, body dissatisfaction, diabetes-specific distress, and longer diabetes duration [9, 14–16]. Despite the severity of outcomes, treatment evidence remains limited: ED treatment outcomes in T1D patients are significantly poorer than in non-diabetic populations [17], and individuals with T1D paradoxically receive less outpatient ED treatment [2]. The Diabetes Body Project, a cognitive dissonance-based prevention program, is the most rigorously evaluated intervention to date, demonstrating small-to-medium acute effects on ED symptoms and diabetes-specific outcomes [18], but evidence for treating established EDs in T1D is scarce [19, 20]. Routine screening with diabetes-specific tools beginning in pre-adolescence and continuing through adulthood, integrated multidisciplinary care, and development of targeted interventions addressing insulin manipulation specifically are consistently recommended across the literature [21–23].

Flow Diagram



Paper search

We performed a semantic search across over 138 million academic papers from the Elicit search engine, which includes all of Semantic Scholar and OpenAlex.

We ran this query: "Synthesize the literature on the prevalence, clinical outcomes, and optimal management strategies for eating disorders concurrent with type 1 diabetes, specifically focusing on deliberate insulin omission for weight loss (diabulimia)."

The search returned 500 total results from Elicit.

We retrieved 500 papers most relevant to the query for screening.

Screening

We screened in sources based on their abstracts that met these criteria:

- **Population - Type 1 Diabetes and Eating Disorders**: Does the study include participants diagnosed with type 1 diabetes who also have eating disorders (including but not limited to deliberate insulin omission/restriction for weight control/diabulimia)?
- **Relevant Outcomes**: Does the study report at least one of the following outcomes: prevalence of eating disorders in type 1 diabetes populations, clinical outcomes associated with concurrent eating disorders and type 1 diabetes, or management/treatment strategies for this comorbid condition?

- **Study Design**: Is the study one of the following types: randomized controlled trial, cohort study, case-control study, cross-sectional study, case series with ≥5 cases, qualitative study, systematic review, or meta-analysis?
- **Human Participants**: Does the study involve human participants?
- **Type 1 Diabetes Focus**: Does the study include participants with type 1 diabetes (rather than focusing solely on type 2 diabetes)?
- **Diabetes Context Required**: Does the study examine eating disorders specifically in the context of diabetes (rather than examining eating disorders in populations without diabetes)?
- **Adequate Sample Size**: Is the study something other than an individual case report or case series with fewer than 5 cases?

We considered all screening questions together and made a holistic judgement about whether to screen in each paper.

At abstract screening, the number of papers excluded for each primary reason was:

- **Other / below screening threshold**: n = 420

Data extraction

We asked a large language model to extract each data column below from each paper. We gave the model the extraction instructions shown below for each column.

- **Study Design**:

  Extract basic study characteristics including: study design (systematic review, cross-sectional, longitudinal, case-control, etc.), setting (clinical, community, online survey), country/region, study period, and sample size. Note whether the study focuses specifically on diabulimia/insulin omission or broader eating disorders in type 1 diabetes.

- **Population & Prevalence**:

  Extract details about the study population and prevalence findings for eating disorders concurrent with type 1 diabetes, including:

- Demographics (age range, mean age, gender distribution, diabetes duration)
- Specific condition studied (diabulimia/insulin omission, broader eating disorders, or both)
- Prevalence rates with confidence intervals where available
- How prevalence was measured/defined
- Population subgroups with different prevalence rates (e.g., by age, gender, diabetes characteristics)

- **Clinical Outcomes**:

  Extract all clinical and psychological outcomes associated with eating disorders concurrent with type 1 diabetes, specifically focusing on diabulimia/insulin omission where reported. Include:

- Metabolic outcomes (HbA1c levels, glycemic control, diabetic ketoacidosis frequency)
- Diabetes complications (retinopathy, nephropathy, neuropathy, cardiovascular outcomes)
- Mental health outcomes (depression, anxiety, quality of life, body image)
- Mortality and morbidity data
- Short-term vs. long-term outcomes
- Severity/dose-response relationships where reported

- **Management Strategies**:

Extract details about screening, assessment, and treatment approaches for eating disorders concurrent with type 1 diabetes, including:

- Screening tools and methods (e.g., DEPS-R, clinical interviews)
- Treatment interventions (psychological, medical, multidisciplinary approaches)
- Intervention effectiveness and outcomes
- Recommended management strategies or guidelines
- Healthcare provider training or awareness interventions
- Setting and delivery method for interventions
- Duration and intensity of treatments

- **Risk Factors**:

  Extract risk factors and correlates associated with development or maintenance of eating disorders concurrent with type 1 diabetes, specifically diabulimia/insulin omission where reported. Include:

- Diabetes-specific factors (age at diagnosis, diabetes duration, treatment regimen, diabetes distress)
- Psychological factors (body dissatisfaction, depression, anxiety, perfectionism, coping mechanisms)
- Social factors (peer influence, family dynamics, healthcare relationships)
- Demographic factors (age, gender, socioeconomic status)
- Protective factors where identified

Results

Characteristics of Included Studies

The 80 included sources comprised a mix of primary studies and reviews spanning nearly four decades of research (1985–2026). Study designs included cross-sectional surveys, longitudinal cohort studies, case-control studies, randomized controlled trials, systematic reviews, meta-analyses, narrative reviews, qualitative studies, and scoping reviews. The majority focused broadly on eating disorders (EDs) and disordered eating behaviors (DEBs) in individuals with type 1 diabetes (T1D), with a subset specifically examining deliberate insulin omission or restriction for weight control (diabulimia). Populations ranged from pre-adolescent children to middle-aged adults, though the literature is heavily concentrated on adolescent and young adult females. Studies were conducted across numerous countries, including the United States, Canada, the United Kingdom, Germany, Austria, Norway, Denmark, Sweden, Finland, Italy, France, Spain, Portugal, Egypt, Tunisia, Saudi Arabia, Turkey, Israel, Brazil, Australia, and Ireland.

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
Christina Bächle et al., 2016	No	Cross-sectional [24]	Germany [24]	Youth aged 11–21 years [24]	819 [24]	DEB and insulin restriction [24]
Franciska Nilsson et al., 2020	No	Cross-sectional [16]	Denmark [16]	Children/adolescents aged 11–19 [16]	192 [16]	DEB in T1D [16]

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
Sohier Yahia et al., 2025	Yes	Cross-sectional [22]	Egypt [22]	Adolescents aged 12–18 [22]	350 [22]	EDs in T1D [22]
Patrick Ritz et al., 2026	No	Cross-sectional [25]	France [25]	Adults, median age 38 [25]	1113 [25]	ED and insulin omission [25]
Gisselle Campoverde et al., 2025	Yes	Systematic review and meta-analysis [6]	Multinational [6]	Adolescents aged 10–19 [6]	13 studies [6]	Diabulimia prevalence [6]
R. Peveler et al., 2005	No	Longitudinal [26]	Not specified [26]	Females aged 11–25 at baseline [26]	87 baseline, 63 follow-up [26]	EDs and insulin misuse [26]
J. Jones et al., 2000	Yes	Cross-sectional case-control [27]	Canada [27]	Females aged 12–19 [27]	356 T1D, 1098 controls [27]	EDs in T1D vs. controls [27]
E. Ip et al., 2023	No	Cross-sectional (online) [14]	Not specified [14]	Adults with T1D [14]	225 [14]	Diabulimia [14]
W. Polonsky et al., 1994	No	Cross-sectional [9]	Not specified [9]	Females aged 13–60 [9]	341 [9]	Insulin omission [9]
Kamila Mozga et al., 2025	Yes	Systematic review [28]	Not specified [28]	Children/adolescents with T1D [28]	3025 (occurrence), 612 (risk) [28]	EDs in T1D [28]
L. Wisting et al., 2013	Yes	Cross-sectional [3]	Norway [3]	Youth aged 11–19 [3]	770 [3]	DEB and insulin omission [3]
M. Takii et al., 2008	No	Cross-sectional [12]	Not specified [12]	Females with T1D and EDs [12]	109 [12]	Insulin omission and complications [12]
Y. Dean et al., 2024	Yes	Systematic review and meta-analysis [7]	Not specified [7]	T1D vs. controls [7]	Multiple studies [7]	EDs in T1D [7]
Kathryn S. Bryden et al., 1999	No	Longitudinal [29]	Not specified [29]	Adolescents aged 11–18 at baseline [29]	76 baseline, 65 follow-up [29]	EDs and insulin misuse [29]
N. Scheuing et al., 2014	No	Longitudinal (registry) [30]	Germany/Austria [30]	Aged 8 to <30 years [30]	52,215 [30]	EDs in T1D [30]
Vasileia Grylli et al., 2004	No	Cross-sectional [31]	Austria [31]	Adolescents, mean age 14.1 [31]	251 (199 assessed) [31]	EDs in T1D [31]

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
D. Neumark-Sztainer et al., 2002	No	Cross-sectional [32]	Not specified [32]	Adolescents with T1D [32]	143 [32]	DEB and insulin omission [32]
I. Engström et al., 1999	No	Case-control [33]	Not specified [33]	Females aged 14–18 [33]	89 T1D, matched controls [33]	EDs in T1D [33]
S. Nielsen, 2002	No	Meta-analysis [34]	Not specified [34]	Females with T1D [34]	Multiple studies [34]	EDs and insulin misuse [34]
A. Troncone et al., 2023	No	Cross-sectional [5]	Italy [5]	Youth aged 11–19 [5]	1562 [5]	DEB and insulin manipulation [5]
S. Sellami et al., 2020	No	Cross-sectional [35]	Tunisia [35]	Adolescents aged 11–18 [35]	102 [35]	EDs in T1D [35]
P. Colton et al., 2015	No	Case series [17]	Canada [17]	T1D with EDs [17]	100 [17]	ED treatment outcomes [17]
M. Olmsted et al., 2008	Yes	Longitudinal [15]	Canada [15]	Girls aged 9–13 at baseline [15]	126 [15]	Predictors of DEB onset [15]
A. Troncone et al., 2022	No	Cross-sectional [36]	Italy [36]	Adolescents aged 11–19 [36]	690 [36]	DEB in T1D [36]
F. Pinna et al., 2022	Yes	Cross-sectional [37]	Italy (Sardinia) [37]	Adults aged 17–55 [37]	172 [37]	Depression, EDs, and DEB in T1D [37]
M. Takii et al., 2002	No	Cross-sectional [38]	Not specified [38]	Females with T1D and BN [38]	79 [38]	Insulin omission subtypes in BN [38]
J. Jones, 2000 (thesis)	No	Cross-sectional [39]	Not specified [39]	Females aged 15 ± 2 [39]	356 T1D, 1098 controls [39]	EDs in T1D vs. controls [39]
T. Reinehr et al., 2019	No	Cohort (registry) [40]	Not specified [40]	Girls aged >6 months to <23 years [40]	31,556 [40]	EDs in girls with T1D [40]
Nicole K. Gibbings et al., 2021	Yes	Retrospective cohort [13]	Canada (Ontario) [13]	Ages 10–39 [13]	168 with EDs, 1680 controls [13]	DKA and mortality in T1D with EDs [13]

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
Kevin P Marks et al., 2023	No	Systematic review and meta-analysis [11]	Not specified [11]	Youth <29 years [11]	35 studies [11]	DEB–HbA1c association [11]
Saeed Yafei et al., 2023	Yes	Cross-sectional [41]	Saudi Arabia [41]	Youth aged 12–25 [41]	265 [41]	DEB and insulin restriction [41]
Leon Hirvelä et al., 2025	Yes	Longitudinal cohort [2]	Finland [2]	Aged <30 at T1D diagnosis [2]	11,055 T1D, 11,055 controls [2]	ED incidence and treatment in T1D [2]
N. D'Silva et al., 2026	No	RCT protocol [42]	Australia [42]	Women aged 15–30 [42]	80 planned [42]	ED prevention in T1D [42]
Ashley E. Tate et al., 2020	No	Longitudinal cohort (register) [1]	Sweden and Denmark [1]	Population-based [1]	>4.3 million [1]	ED risk in T1D and familial coaggregation [1]
Christopher M. Lawrence et al., 2024	No	Cross-sectional [4]	Australia [4]	Adolescents, mean age 15.2 [4]	50 [4]	DEB and diabetes technology [4]
Gülsüm Şahin-Bodur et al., 2021	No	Cross-sectional [43]	Not specified [43]	Adolescents aged 10–19 [43]	110 [43]	Diabulimia risk [43]
Tamar Propper-Lewinsohn et al., 2024	No	Cross-sectional [44]	Not specified [44]	Youth aged 13–21 [44]	147 [44]	DEB and glycemic variability [44]
Mareille H C L Hennekes et al., 2024	No	Multinational RCT [18]	Multinational [18]	Women aged 14–35 [18]	293 [18]	ED prevention (Diabetes Body Project) [18]
Magnus Sjögren et al., 2026	No	Retrospective cohort [45]	Sweden [45]	T1D diagnosed <15 years [45]	12,202 T1D, 48,484 controls [45]	Anorexia nervosa in T1D [45]
Mariana Palhão Verri (n.d.)	Yes	Cross-sectional [46]	Brazil [46]	Adolescents aged 10–19 [46]	123 [46]	EDs and diabulimia [46]

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
M. de Wit et al., 2025	No	Cross-sectional (baseline data from RCT) [47]	Not specified [47]	Women aged 14–35 [47]	293 [47]	ED and DS-DEB in T1D [47]
Margo E Hanlan et al., 2013	Yes	Review [21]	Not specified [21]	Adolescents/young adults [21]	Not applicable [21]	EDs and DEB in T1D [21]
M. B. Anesini et al., 2026	No	Systematic review [48]	Not specified [48]	Adolescents/young adults with T1D [48]	29 studies [48]	Intentional insulin omission [48]
Karol Piotrowski et al., 2026	No	Narrative review [49]	Not specified [49]	Adults with T1D [49]	Not applicable [49]	Diabulimia in adults [49]
Velimir Altabas et al., 2025	No	Systematic review [50]	Not specified [50]	T1D population [50]	19 manuscripts [50]	Diabulimia [50]
R. Nissim et al., 2002	No	Review [51]	Not specified [51]	Adolescent girls with T1D [51]	Not applicable [51]	EDs in T1D [51]
S. Kelly et al., 2005	No	Review [52]	Not specified [52]	Youth with T1D [52]	Not applicable [52]	EDs in T1D [52]
A. Goebel-Fabbri, 2009	No	Review [53]	Not specified [53]	Women with T1D [53]	Not applicable [53]	DEB and EDs in T1D [53]
Claire M. Peterson et al., 2015	Yes	Review (theoretical model) [54]	Not specified [54]	Youth with T1D [54]	Not applicable [54]	Risk model for DEB in T1D [54]
Natalia Kriese et al., 2026	Yes	Narrative review [55]	Not applicable [55]	T1D population [55]	23 articles [55]	Diabulimia [55]
D. Young-Hyman & C. Davis, 2010	Yes	Review [56]	Not specified [56]	T1D population [56]	>100 articles [56]	DEB in diabetes [56]
S. Herpertz et al., 1998	No	Cross-sectional [57]	Germany [57]	Adults with T1D and T2D [57]	341 T1D, 322 T2D [57]	EDs in T1D and T2D [57]
Maria Ana Falcão & Rita Francisco, 2017	Yes	Case-control [58]	Portugal [58]	Young adults aged 18–30 [58]	128 (55 T1D, 73 controls) [58]	DEB and diabulimia [58]

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
S. Coleman & N. Caswell, 2020	Yes	Qualitative (thematic analysis) [59]	UK [59]	Adults, mean age 32 [59]	45 [59]	Diabulimia lived experience [59]
A. Rydall et al., 1997	Yes	Longitudinal [8]	Canada [8]	Females, mean age 15 at baseline [8]	91 [8]	DEB and microvascular complications [8]
Stephen Poos et al., 2023	Yes	Systematic review (qualitative) [60]	Multinational [60]	T1D with diabulimia [60]	185 across 12 studies [60]	Diabetes distress and diabulimia [60]
Stephen Poos et al., 2024	No	Systematic review [61]	Not specified [61]	T1D with diabulimia [61]	185 across 12 studies [61]	Diabetes distress and diabulimia [61]
C. Fairburn et al., 1991	Yes	Cross-sectional [62]	UK [62]	Young adults aged 17–25 [62]	100 T1D, 67 controls [62]	EDs and insulin misuse [62]
R. Peveler et al., 1992	No	Cross-sectional [63]	Not specified [63]	Adolescents with T1D [63]	76 T1D [63]	EDs and insulin misuse [63]
S. Affenito & C. H. Adams, 2001	No	Case-control [64]	Not specified [64]	Females aged 12–19 [64]	Not specified [64]	EDs in T1D [64]
P. Colton et al., 2015a	Yes	Longitudinal [10]	Canada [10]	Girls, mean age 11.8 at baseline [10]	126 [10]	ED course over 14 years [10]
E. Mannucci et al., 2005	No	Meta-analysis [65]	Not specified [65]	Females with T1D [65]	748 T1D, 1587 controls [65]	EDs in T1D [65]
J. Steel et al., 1987	Yes	Retrospective cohort [66]	Scotland [66]	Young women, ages 16–25 [66]	208 [66]	EDs in T1D [66]
G. Rodin et al., 2002	No	Review [67]	Not specified [67]	Young women with T1D [67]	Not applicable [67]	EDs in T1D [67]
M. Pollock et al., 1995	No	Longitudinal [68]	Not specified [68]	Youth, ages 8–13 at onset [68]	79 [68]	EDs and insulin omission [68]
S. Nielsen & A. Mølbak, 1998	No	Meta-analysis [69]	Not specified [69]	Females with T1D [69]	Multiple studies [69]	EDs and T1D [69]

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
Rosemary Banting & Catherine Randle-Phillips, 2018	No	Systematic review [19]	Not specified [19]	T1D with EDs [19]	10 studies [19]	Psychological interventions [19]
M. Balfe et al., 2013	Yes	Qualitative [70]	Ireland [70]	Young adults aged 23–30 [70]	35 patients, 13 HCPs [70]	Weight loss concerns in T1D [70]
N. Custal et al., 2014	Yes	Comparative (matched) [71]	Spain [71]	Adults, mean age 25.3 [71]	40 (20 T1D, 20 non-DM) [71]	ED treatment outcomes in T1D [71]
Norma I García-Reyna et al., 2004	No	Cross-sectional [72]	Not specified [72]	Young adolescents [72]	98 T1D, 575 controls [72]	EDs in T1D [72]
Randi Birk & M. Spencer, 1989	No	Cross-sectional (survey) [73]	Not specified [73]	Females aged 13–45 [73]	550 (70% response) [73]	EDs and induced glycosuria [73]
Ruth Martin et al., 2023	No	Scoping review [74]	Not specified [74]	Adolescents/adults with T1D [74]	59 sources [74]	Dietitian role in ED/T1D [74]
A. Nip et al., 2019	Yes	Cohort (SEARCH study) [75]	United States [75]	Youth, mean age 17.7 [75]	2156 T1D, 149 T2D [75]	DEB in T1D and T2D [75]
G. Wagner & A. Karwautz, 2020	No	Review [20]	Not specified [20]	Adolescents with T1D [20]	Not applicable [20]	EDs in adolescents with T1D [20]
S. Philippi et al., 2013	Yes	Cross-sectional [76]	Brazil [76]	Ages 12–56 [76]	189 [76]	ED risk and insulin omission [76]
Emma Chad-Friedman et al., 2024	No	Review [23]	Not specified [23]	Adolescents with T1D [23]	Not applicable [23]	DEB risk factors and screening [23]
V. Cherubini et al., 2018	No	Cross-sectional [77]	Italy [77]	Adolescents aged 11–20 [77]	163 [77]	DEB in T1D [77]
P. Colton et al., 2004	No	Cross-sectional case-control [78]	Not specified [78]	Girls aged 9–14 [78]	101 T1D, 303 controls [78]	DEB in preteen girls [78]

Study	Full Text Retrieved?	Study Type	Country/Region	Population	Sample Size	Primary Focus
M. Biggs et al., 1994	No	Cross-sectional [79]	Not specified [79]	Women aged 16–40 [79]	42 [79]	Insulin withholding for weight control [79]
James I. Hudson et al., 1985	No	Cross-sectional (survey) [80]	Not specified [80]	Young women with T1D [80]	264 [80]	AN and bulimia in T1D [80]

Of the 80 sources, 26 had full texts retrieved. The remaining 54 were available as abstracts only. The studies included 30 cross-sectional or case-control primary studies, 9 longitudinal or cohort studies, 2 randomized controlled trials (one a protocol), 15 systematic reviews or meta-analyses, 6 narrative or scoping reviews, 2 qualitative studies, and several additional review articles. Sample sizes in primary studies ranged from 40 to over 52,000, with register-based cohorts from Scandinavia and Germany contributing the largest populations.

Effects

Prevalence of Eating Disorders and Disordered Eating Behaviors in Type 1 Diabetes

The literature consistently demonstrates elevated rates of both clinical EDs and subclinical DEBs among individuals with T1D compared to non-diabetic peers, though estimates vary considerably depending on the screening instrument, diagnostic method, population characteristics, and how broadly "disordered eating" is defined.

Prevalence of Clinical Eating Disorders

Study	Population	Instrument/Criteria	ED Prevalence in T1D	ED Prevalence in Controls	Relative Risk/OR
J. Jones et al., 2000	Females aged 12–19 [27]	DSM-IV interview [27]	10% DSM-IV; 14% subthreshold [27]	4% DSM-IV; 8% subthreshold [27]	OR 2.4 (95% CI 1.5–3.7) [27]
Vasileia Grylli et al., 2004	Adolescents, mean age 14.1 [31]	DSM-IV interview [31]	Girls: 11.5% clinical, 13.5% subsyndromal; Boys: 0% [31]	Not assessed [31]	Not applicable [31]
E. Mannucci et al., 2005	Females (meta-analysis) [65]	DSM-III-R/DSM-IV interview [65]	AN 0.27%, BN 1.73%, combined 2.00% [65]	AN 0.06%, BN 0.69%, combined 0.75% [65]	BN significantly higher ($p < 0.05$) [65]
N. Scheuing et al., 2014	Aged 8–<30 (registry) [30]	DSM-IV clinical diagnosis [30]	467 of 52,215 (0.9%) [30]	Not applicable [30]	Not applicable [30]

Study	Population	Instrument/Criteria	ED Prevalence in T1D	ED Prevalence in Controls	Relative Risk/OR
Ashley E. Tate et al., 2020	Population registers (Sweden, Denmark) [1]	Clinical diagnoses [1]	Not specified as prevalence [1]	Not specified [1]	Meta-analyzed: AED HR 2.07 (1.88–2.28), AN 1.68 (1.44–1.95), OED 2.44 (2.17–2.72) [1]
Leon Hirvelä et al., 2025	Aged <30 (Finland) [2]	Hospital records (ICD-10) [2]	175 incident cases [2]	75 incident cases [2]	IRR 2.35 (95% CI 1.80–3.09) [2]
Magnus Sjögren et al., 2026	Childhood-onset T1D (Sweden) [45]	ICD-10 (AN only) [45]	1.9% period prevalence (females) [45]	1.1% [45]	OR 1.64 (95% CI 1.31–2.06) [45]
P. Colton et al., 2015a	Girls followed 14 years [10]	Interview (DSM-IV-TR) [10]	32.4% current ED at age ~24; cumulative probability 60% by age 25 [10]	Not applicable [10]	Not applicable [10]
Sohier Yahia et al., 2025	Adolescents aged 12–18 [22]	DEPS-R + DSM-V interview [22]	22.6% [22]	Not applicable [22]	Not applicable [22]

The large register-based studies from Scandinavia provide robust population-level evidence that T1D approximately doubles the risk of any ED diagnosis, with hazard ratios consistently in the range of 2.0–2.5 [1, 2]. The meta-analysis by Mannucci et al. found that bulimia nervosa was significantly more prevalent in T1D females, while anorexia nervosa showed no significant elevation [65]. This pattern was partially echoed by Dean et al., who reported T1D was associated with bulimia nervosa (RR 2.80, 95% CI 1.18–6.65) and binge eating (RR 1.53, 95% CI 1.18–1.98) but not anorexia nervosa [7]. However, the Swedish nationwide cohort by Sjögren et al. did find a significantly elevated risk of anorexia nervosa (OR 1.64, 95% CI 1.31–2.06) [45], suggesting that the relationship may depend on study size and detection methods. The earlier meta-analysis by Nielsen and Mølbak found no support for increased anorexia nervosa but confirmed elevated bulimia nervosa (OR 2.9, 95% CI 1.03–8.4) and ED-NOS (OR ~2) [34].

Critically, the longitudinal study by Colton et al. demonstrated that the cumulative probability of developing an ED reached 60% by age 25 in girls with T1D, with a mean age of onset of 22.6 years and a high recurrence rate (53% by 6 years after remission) [10]. This underscores that cross-sectional point prevalence figures substantially underestimate lifetime burden.

Prevalence of Disordered Eating Behaviors (Screener-Based)

Study	Population	Instrument	DEB Prevalence	Gender Differences
L. Wisting et al., 2013	Youth aged 11–19, Norway [3]	DEPS-R ≥20 [3]	Females 27.7%, Males 8.6% [3]	Females > Males [3]
Franciska Nilsson et al., 2020	Youth aged 11–19, Denmark [16]	DEPS-R [16]	Overall 21%; Girls 34.1%, Boys 8.9% [16]	Females > Males [16]

Study	Population	Instrument	DEB Prevalence	Gender Differences
A. Troncone et al., 2023	Youth aged 11–19, Italy (n=1562) [5]	DEPS-R ≥20 [5]	29.7% [5]	Higher in females [5]
A. Troncone et al., 2022	Youth aged 11–19, Italy (n=690) [36]	DEPS-R ≥20 [36]	Overall 28.1%; Girls 35%, Boys 21% [36]	Females > Males [36]
V. Cherubini et al., 2018	Adolescents aged 11–20, Italy [77]	DEPS-R ≥20 [77]	Boys 27% (95% CI 17–38), Girls 42% (95% CI 31–53) [77]	Females > Males [77]
Saeed Yafei et al., 2023	Youth aged 12–25, Saudi Arabia [41]	DEPS-R ≥20 [41]	Overall 27.2%; Females 32.5%, Males 18.6% [41]	Females > Males [41]
Christopher M. Lawrence et al., 2024	Adolescents, mean age 15.2, Australia [4]	DEPS-R [4]	48% [4]	Females 75% vs. Males 31.6% [4]
A. Nip et al., 2019	Youth, mean age 17.7, US [75]	DEPS-R ≥20 [75]	T1D: 21.2%; T2D: 50.3% [75]	Females > Males [75]
Christina Bächle et al., 2016	Youth aged 11–21, Germany [24]	SCOFF [24]	Females: 28.2% SCOFF+ (no IR), 2.7% both; Males: 9.2% SCOFF+ (no IR), 1.9% both [24]	Females > Males [24]
S. Sellami et al., 2020	Adolescents aged 11–18, Tunisia [35]	Not specified [35]	33.3%; Girls 46.2%, Boys 20% [35]	Females > Males [35]
Tamar Propper-Lewinsohn et al., 2024	Youth aged 13–21 [44]	Self-report questionnaires [44]	42.1% [44]	Not specified [44]
Gülsüm Şahin-Bodur et al., 2021	Adolescents aged 10–19 [43]	DEPS-R ≥20 [43]	31.8% [43]	Not specified [43]
F. Pinna et al., 2022	Adults, mean age 36.8, Italy [37]	DEPS-R [37]	19.2% current DEB; 20.9% lifetime ED [37]	Females: 34.9% vs. Males: 7.0% [37]
M. de Wit et al., 2025	Women aged 14–35 [47]	DEPS-R ≥20 and diagnostic interview [47]	11% clinical ED; 45% elevated DS-DEB [47]	Women only [47]
Mariana Palhão Verri (n.d.)	Adolescents aged 10–19, Brazil [46]	EAT-26, BITE, DEPS-R [46]	27.6% (EAT-26), 47.9% (BITE), 60.2% (DEPS-R) [46]	Girls at higher risk [46]
S. Philippi et al., 2013	Ages 12–56, Brazil [76]	EAT, BITE, BES [76]	58.7% at risk [76]	Females > Males [76]

Across studies using the DEPS-R (the most commonly employed diabetes-specific screener), DEB prevalence in adolescents with T1D ranged from approximately 21% to 48%, with a consistent female predominance [3–5, 16, 36]. The pooled meta-analytic estimate from Campoverde et al. was 11% (95% CI 9–13%) for diabulimia specifically, with prevalence substantially higher in females (45%) than males (26%) [6]. The systematic review by Mozga et al. synthe-

sized data from 3,025 T1D patients and found 28.5% diagnosed with EDs, with females disproportionately affected (57.6% of cases) [28].

Prevalence of Insulin Omission/Restriction

Study	Population	Definition	Prevalence
W. Polonsky et al., 1994	Women aged 13–60 [9]	Self-reported intentional insulin omission [9]	31% any omission; 8.8% frequent [9]
L. Wisting et al., 2013	Youth aged 11–19 [3]	DEPS-R items [3]	31.6% restriction; 6.9% omission after overeating [3]
R. Peveler et al., 2005	Females aged 11–25 [26]	EDE interview + chart review [26]	35.6% (95% CI 25.7–46.6) [26]
A. Rydall et al., 1997	Females, mean age 15 [8]	Self-reported [8]	14% at baseline, 34% at follow-up [8]
Kathryn S. Bryden et al., 1999	Females aged 11–18 at baseline [29]	EDE interview [29]	30% of females [29]
C. Fairburn et al., 1991	Women aged 17–25 [62]	EDE interview [62]	37% of diabetic women [62]
R. Peveler et al., 1992	Adolescent girls [63]	EDE interview [63]	15% [63]
D. Neumark-Sztainer et al., 2002	Adolescent females [32]	AHEAD survey [32]	10.3% skipping; 7.4% reducing insulin [32]
Y. Dean et al., 2024 (meta-analysis)	Multiple populations [7]	Various [7]	10.3% (95% CI 8.1–13) [7]
E. Ip et al., 2023	Adults (online) [14]	Self-report of intentional omission for weight loss [14]	8.9% [14]
Saeed Yafei et al., 2023	Youth aged 12–25 [41]	DEPS-R items [41]	27.5% [41]
Patrick Ritz et al., 2026	Adults, median age 38 [25]	Single question [25]	21% (16% IOM without ED + 5% IOM with ED) [25]
A. Troncone et al., 2023	Youth aged 11–19 [5]	DEPS-R items [5]	42.4% insulin manipulation [5]
P. Colton et al., 2015a	Women, mean age ~24 [10]	Interview [10]	27% at time 7 [10]
Norma I García-Reyna et al., 2004	Young adolescents [72]	EDE + self-report [72]	9.1% [72]
M. B. Anesini et al., 2026 (review)	Adolescents/young adults [48]	Various [48]	20–45% [48]

Insulin omission or restriction prevalence varied from approximately 7% to 42% depending on how the behavior was defined and measured [5, 9]. When defined strictly as weight-motivated omission ("diabulimia"), meta-analytic estimates converge around 10–11% [6, 7]. However, when broader definitions encompassing any insulin restriction or manipulation are used, rates rise to 27–42% [5, 8, 62]. The longitudinal study by Rydall et al. demonstrated that insulin omission increases over time, rising from 14% at baseline to 34% at four-to-five-year follow-up [8]. Females are markedly more likely to omit insulin for weight loss, with Dean et al. reporting a relative risk of 14.21 (95% CI 2.66–76.04) for insulin omission in diabetic females compared to males [7].

Glycemic and Metabolic Outcomes

The association between DEBs (including insulin omission) and impaired glycemic control is among the most consistent findings across the literature.

Study	Population	HbA1c with DEB/ED	HbA1c without DEB/ED	Statistical Significance
J. Jones et al., 2000	Adolescent females [27]	9.4% (1.8) [27]	8.6% (1.6) [27]	p = 0.04 [27]
Franciska Nilsson et al., 2020	Youth aged 11–19 [16]	72.8 mmol/mol (8.8%) [16]	62.0 mmol/mol (7.8%) [16]	p < 0.001 [16]
L. Wisting et al., 2013	Youth aged 11–19 [3]	9.2% (DEB+); 9.0% (IR+) [3]	8.4% (DEB-); 8.3% (IR-) [3]	p < 0.001 [3]
N. Scheuing et al., 2014	Ages 8–<30 [30]	AN 8.61%, BN 9.11%, EDNOS 9.00% [30]	8.29% [30]	All p < 0.05 [30]
E. Ip et al., 2023	Adults (online) [14]	8.4% [14]	6.9% [14]	p = 0.014 [14]
A. Rydall et al., 1997	Females, mean age 15 [8]	11.1% (highly disordered) [8]	8.7% (nondisordered) [8]	p < 0.001 [8]
Christopher M. Lawrence et al., 2024	Australian adolescents [4]	8.2% / 67 mmol/mol [4]	6.9% / 51 mmol/mol [4]	p < 0.002 [4]
Norma I García-Reyna et al., 2004	Young adolescents [72]	9.7% (1.52) [72]	8.4% (1.5) [72]	p = 0.049 [72]
A. Nip et al., 2019	US youth, mean age 17.7 [75]	10.2% (2) [75]	8.9% (1.7) [75]	Not specified [75]

The meta-analysis by Marks et al. quantified the association: diabetes-adapted DEB questionnaires were associated with higher HbA1c with a Hedges' g of 0.62 (95% CI 0.52–0.73), while generic questionnaires showed a weaker, sometimes non-significant association (g = 0.19, 95% CI -0.17–0.55) [11]. This indicates that the insulin omission component captured by diabetes-specific instruments is a key driver of the DEB–HbA1c relationship. Lawrence et al. further demonstrated that individuals with elevated DEPS-R scores had lower continuous glucose monitor time-in-range (50.3% vs. 63.8%, p = 0.01) and increased meal bolus overrides on insulin pumps (7.9% vs. 3.8%, p = 0.047) [4]. In the French SFDT1 cohort, insulin omission combined with ED was associated with reduced time-in-range (OR 0.5, 95% CI 0.4–0.7) and increased time-above-range (OR 2.2, 95% CI 1.6–2.9) [25].

One notable exception was Herpertz et al., who found that neither EDs nor insulin omission influenced glycemic control [57], and Engström et al. found no relationship between EDI scores and HbA1c [33]. These contrasting findings are discussed in the synthesis section below.

Diabetic Ketoacidosis

Scheuing et al. reported DKA rates of 5.7 events per 100 person-years in T1D patients without EDs, compared to 12.1 (AN), 18.0 (BN), and 12.9 (EDNOS) events per 100 person-years in those with EDs [30]. Gibbings et al. found a DKA incidence of 112.5 per 1,000 patient-years in individuals with T1D and EDs versus 30.8 in those without, yielding an adjusted subdistribution hazard ratio of 3.30 (95% CI 2.58–4.23) [13].

Microvascular Complications

Study	Complication	Finding
S. Nielsen, 2002 (meta-analysis)	Retinopathy	OR 4.8 (95% CI 3.0–7.8) for co-existing ED with T1D [34]
S. Nielsen & A. Mølbak, 1998 (meta-analysis)	Retinopathy	OR 8.04 (95% CI 4.0–16.1) [69]
A. Rydall et al., 1997	Retinopathy	86% in highly disordered vs. 24% in nondisordered eating at 4-year follow-up [8]
M. Takii et al., 2008	Retinopathy, nephropathy	Duration of severe insulin omission most closely associated (OR 1.35 for retinopathy, 1.35 for nephropathy per year) [12]
N. Scheuing et al., 2014	Retinopathy	BN: 2.5-fold risk (95% CI 1.3–4.8); EDNOS: 1.4-fold (0.8–2.3) [30]
J. Steel et al., 1987	Retinopathy, nephropathy, neuropathy	11/15 had retinopathy, 6 nephropathy, 6 neuropathy; 4 developed acute painful polyneuropathy [66]
R. Peveler et al., 2005	Microvascular complications	Significant relationships between disordered eating, insulin misuse, and microvascular complications [26]

The evidence linking co-existing EDs/DEBs with accelerated microvascular disease is substantial. Takii et al. identified the duration of severe insulin omission as the factor most closely associated with both retinopathy and nephropathy in T1D females with clinical EDs, independent of diabetes duration [12]. This dose-response relationship between duration of insulin omission and complications severity provides mechanistic support for the clinical significance of diabulimia.

Mortality

Gibbings et al. reported all-cause mortality of 16.0 per 1,000 person-years in individuals with T1D and EDs versus 2.5 in those without, with an adjusted hazard ratio of 5.80 (95% CI 3.04–11.08) [13]. Sjögren et al. found a proportional mortality ratio of 20.4 (95% CI 6.6–47.6) for females with both T1D and anorexia nervosa compared to controls without either condition [45]. Peveler et al. noted that overall outcome in their longitudinal cohort was "poor; serious microvascular complications were common and mortality was high" [26]. Coleman and Caswell cited evidence that insulin restriction increases the risk of death by 3.2 times [59]. These figures place the co-occurrence of EDs and T1D among the highest-mortality psychiatric-medical comorbidities.

Mental Health and Psychosocial Outcomes

The comorbidity extends beyond metabolic consequences. Ip et al. found that individuals with diabulimia had significantly higher rates of major depressive disorder (40.0% vs. 11.5%, $p < 0.001$) [14]. Nip et al. reported markedly

more depressive symptoms in those with DEBs (CES-D scores 16.7 vs. 8.3) and poorer quality of life [75]. Pinna et al. found high rates of depression (35.5%) in adult T1D patients, with higher DEPS-R scores significantly increasing the odds of depression (adjOR 1.09, 95% CI 1.03–1.15) [37]. Troncone et al. demonstrated that both internalizing and externalizing symptoms were significantly associated with DEBs, explaining 37% of the variance in disordered eating [5]. The qualitative work by Coleman and Caswell revealed that individuals with diabulimia reported overwhelmingly negative experiences with healthcare professionals [59], while Balfe et al. documented depressive episodes and suicidal thoughts among young women with T1D and eating disturbances [70].

Treatment Outcomes

Treatment outcome data are sparse. Colton et al. reported that among 37 T1D patients attending an ED day hospital, only 18.8% achieved a good immediate outcome, 43.8% had intermediate outcomes, and 37.5% met ED diagnostic criteria at discharge—significantly poorer than outcomes in individuals without diabetes ($p = 0.002$) [17]. Custal et al. found higher dropout rates in T1D patients with EDs undergoing cognitive-behavioral therapy, with lower motivation to change [71]. Hirvelä et al. noted that individuals with T1D received less outpatient hospital treatment for EDs (mean 3.32 vs. 5.33 visits per year) despite their greater medical risk [2].

The multinational RCT of the Diabetes Body Project by Hennekes et al. showed significant improvements at posttest with small-to-medium effect sizes for ED symptoms ($d = -0.30$), diabetes distress ($d = -0.42$), diabetes-specific DEBs ($d = -0.70$), and body dissatisfaction ($d = -0.59$) compared to educational controls [18]. This represents the most rigorous prevention trial evidence to date, though longer-term follow-up data are awaited. An Australian adaptation (ADBP) is currently being tested in a clinic-based RCT [42].

Risk Factors and Correlates

Multiple interacting risk factors emerge across the literature.

Demographic and Clinical Risk Factors. Female sex is the most consistently identified risk factor, with females showing two-to-fourfold higher rates of DEBs across virtually all studies [3, 7, 16, 27]. Higher BMI or overweight status is strongly associated with DEB risk [15, 31, 33, 77]. Cherubini et al. found that overweight youth were six times more likely to screen positive on the DEPS-R [77]. Older age within adolescence is associated with higher DEB prevalence, with Wisting et al. documenting an increase from 8.1% in the 11–13 age group to 38.1% in the 17–19 age group [3]. Longer diabetes duration is a consistent correlate [8, 16, 22].

Psychological Risk Factors. Depression and depressive symptoms are strongly linked to DEB and insulin omission [14, 15, 53]. Ip et al. found that major depressive disorder increased the odds of diabulimia nearly fivefold (OR 4.87, 95% CI 1.31–18.22) [14]. Body dissatisfaction, weight and shape concerns, and low self-worth consistently emerge as predictors of DEB onset [15, 53, 54, 58]. Diabetes-specific distress was identified by Polonsky et al. as an independent predictor of insulin omission, alongside fear that improved glycemic control will lead to weight gain [9]. Poos et al. systematically identified diabetes distress themes across 12 qualitative studies of individuals with diabulimia, with over 40 participants meeting criteria from the Diabetes Distress Scale [60, 61].

Social and Family Factors. Neumark-Sztainer et al. found family cohesion was inversely associated with disordered eating in both females ($r = -0.52$) and males ($r = -0.41$) [32]. Family conflict was identified as an intensifier of disordered eating behaviors by Balfe et al. [70] and Yahia et al. [22]. Coleman and Caswell reported that past trauma, including emotional, physical, and sexual abuse, was a common theme among those with diabulimia [59].

Protective Factors. Physical activity was identified as protective, with Cherubini et al. finding that each added hour per week spent in physical activity decreased the probability of screening DEPS-R positive by approximately 20%

[77]. Family cohesion [32], parental involvement [28], and positive self-worth [15] were also noted as potentially protective.

Screening and Management

Screening. The DEPS-R is the most widely used diabetes-specific screening tool and was employed in the majority of recent prevalence studies [3, 4, 22, 41, 48, 77]. Marks et al. demonstrated that diabetes-adapted questionnaires are more strongly associated with HbA1c than generic instruments [11], supporting the recommendation to use diabetes-specific tools that capture insulin manipulation behaviors. The modified SCOFF (mSCOFF) was also recommended as a brief screening option [23, 60]. Yahia et al. proposed screening criteria: adolescents around age 13.6 years, at Tanner stage 3, with T1D duration >5 years, and/or HbA1c >7.5% [22]. Multiple reviews recommend that screening begin in the pre-adolescent period and continue through early adulthood [21, 51, 67, 78].

Treatment Approaches. The consistent recommendation across reviews is for multidisciplinary care integrating endocrinology, dietetics, and mental health [19, 21, 50, 55, 60]. Specific psychological treatments mentioned include cognitive-behavioral therapy (CBT) [28, 54, 55, 71], family-based therapy [21, 59, 67], and motivational enhancement interventions [71]. The Diabetes Body Project, a cognitive dissonance-based group prevention program, represents the most rigorously tested intervention, with significant acute effects on ED symptoms and diabetes-specific outcomes [18]. However, Banting and Randle-Phillips concluded from their systematic review that an individualized approach addressing both ED and T1D simultaneously appeared most effective, while noting a paucity of evidence on insulin omission as a treatment target [19].

Synthesis

The overarching finding across 80 sources spanning four decades is that EDs and DEBs are significantly more prevalent in individuals with T1D than in the general population, and that these behaviors—particularly insulin omission—are associated with substantially worse glycemic control, accelerated microvascular complications, and increased mortality. However, several areas of heterogeneity and apparent contradiction merit closer examination.

Reconciling Prevalence Estimates. The wide range of reported DEB prevalence (from ~8% to 60%) is best understood as reflecting differences in measurement approach rather than true population differences. Studies using diagnostic interviews and strict DSM criteria consistently yield lower prevalence (2–11% for full-syndrome EDs) [27, 31, 65], while diabetes-specific screeners like the DEPS-R with a cutoff of ≥20 identify 20–48% as screen-positive [3–5], and instruments that capture any form of insulin manipulation yield the highest figures (up to 42%) [5]. The meta-analytic estimate of 11% for diabulimia specifically [6] likely reflects the narrowest definition (deliberate insulin omission for weight loss), while estimates of 20–35% for broader insulin restriction [8, 9, 48] include a spectrum from occasional dose reduction to systematic omission. Registry-based studies report very low clinically recognized ED rates (0.9% in the German/Austrian DPV registry) [30], suggesting substantial under-detection in routine clinical practice.

The HbA1c–DEB Association: Explaining Discordant Findings. While the vast majority of studies report significant associations between DEBs and elevated HbA1c, two notable exceptions exist. Herpertz et al. found no association between EDs/insulin omission and glycemic control [57], and Engström et al. found no relationship between EDI scores and HbA1c [33]. These discrepant findings can be explained by measurement specificity. The Herpertz et al. study used a general assessment of EDs not designed to capture insulin manipulation behaviors. Engström et al. used the Eating Disorder Inventory (a generic tool), which does not assess insulin omission. The meta-analysis by Marks et al. directly demonstrated that generic questionnaires show a much weaker association with HbA1c ($g = 0.19$) compared to diabetes-adapted questionnaires ($g = 0.62$) [11]. In other words, it is specifically the insulin

manipulation component—not disordered eating attitudes per se—that drives the metabolic impact. Studies that fail to assess insulin omission will systematically underestimate the clinical significance of the ED–T1D comorbidity.

Gender Differences. While females consistently show higher overall DEB and ED prevalence, males are not unaffected. Bächle et al. found 5.3% of males reported frequent insulin restriction even without positive SCOFF screening [24], and Troncone et al. found 21% of boys screened positive on the DEPS-R [36]. Dean et al. reported markedly higher relative risks for insulin omission in females (RR 14.21) versus males [7], but the absolute prevalence in males is clinically relevant. Sellami et al. found 20% of boys with T1D had EDs [35]. These findings argue against screening only in female patients.

Temporal Dynamics. The longitudinal evidence makes clear that EDs in T1D are not merely an adolescent phenomenon. Colton et al. found new-onset EDs well into adulthood (mean onset age 22.6 years, cumulative probability 60% by age 25) [10], with high recurrence after remission (53% by 6 years) [10]. Rydall et al. showed insulin omission prevalence increasing from 14% to 34% over 4–5 years [8]. Peveler et al. documented that cumulative incidence of eating problems continued to increase after young adulthood [26]. Polonsky et al. found insulin omission across all ages (13–60 years), not limited to younger women [9]. Together, these findings indicate that both the onset and persistence of DEBs extend far beyond the often-assumed adolescent window, necessitating ongoing vigilance throughout the lifespan.

Insulin Omission as the Central Mediating Behavior. The evidence converges on insulin omission/restriction as the critical behavior linking EDs to adverse outcomes in T1D. Takii et al. demonstrated that among various ED behaviors, duration of severe insulin omission was the factor most closely associated with both retinopathy and nephropathy [12]. Marks et al. showed that diabetes-specific DEB instruments (which assess insulin manipulation) are more strongly associated with HbA1c than generic instruments [11]. The French SFDT1 study found that insulin omission—even in the absence of an ED—was associated with impaired glycemic control, while ED without insulin omission showed no such association [25]. Takii et al. further showed that among T1D females with bulimia nervosa, those who used insulin omission as a purging method (alone or in combination with vomiting/laxatives) had the worst metabolic control and highest complication rates, while those who purged through non-insulin means had relatively preserved metabolic outcomes but the highest psychological distress [38]. This subgroup analysis underscores that the metabolic toxicity of EDs in T1D is specifically mediated through insulin manipulation rather than through the eating pathology per se.

Treatment Gaps. The evidence reveals a concerning treatment gap. Hirvelä et al. found that despite greater medical risks, individuals with T1D received significantly less outpatient ED treatment than those without diabetes (3.32 vs. 5.33 visits per year) [2]. Treatment outcomes are poorer than in non-diabetic ED populations (18.8% good outcome vs. significantly better in non-diabetic patients, $p = 0.002$) [17]. Healthcare professionals report difficulty detecting and managing the dual condition due to lack of validated screening tools and integrated care guidelines [20]. Qualitative data from Coleman and Caswell revealed that patients with diabulimia had "overwhelmingly negative" experiences with health professionals, with many feeling misunderstood [59]. These findings point to a need for both better clinical tools and improved provider training. The Diabetes Body Project represents a promising prevention approach with demonstrated acute efficacy [18], but treatment evidence for established EDs in T1D remains remarkably limited, with Banting and Randle-Phillips identifying only 10 studies on psychological interventions [19] and Wagner and Karwautz noting that "studies on psychological treatments for the comorbidity of eating disorders and T1DM are scarce, especially for adolescents" [20].

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