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Subtypes of disordered eating and their diabetes-related and psychosocial concomitants in adults with type 1 diabetes

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ABSTRACT

Aims: To identify subtypes of disordered eating behaviors (DEB) in type 1 diabetes, describing their behavioral patterns, clinical features, psychosocial well-being, and diabetes-related complications.
Methods: Baseline data of the Disordered Eating Behaviors and Eating Disorders in Diabetes Type I (DEBBI) study were analyzed ($N = 645$). Participants completed questionnaires assessing DEB, diabetes-related outcomes, and psychosocial well-being. Latent profile analysis identified subtypes based on DEPS-R indicators, followed by comparisons of demographic, clinical, and psychosocial variables.
Results: Four distinct DEB subtypes were identified, that differed in intensity and behavioral characteristics: restrained eating (moderate DEB), disinhibited eating (moderate DEB), maintaining high glucose (severe DEB), and dual compensatory behaviors (severe DEB). All DEB subtypes reported significant poorer psychosocial well-being, including elevated diabetes distress, fear of hypoglycemia, depression, and anxiety. The severe DEB subtypes showed poorer diabetes self-management and higher HbA_{1c} levels.
Conclusions: The diversity of DEB presentations in type 1 diabetes underscores the need to examine specific behavioral patterns. The subtypes revealed distinct clinical features, behavioral patterns, and variations in self-management quality, demonstrating that harmful DEB extends beyond insulin purging alone. Even moderate forms of DEB were linked to significant psychosocial burden, highlighting the importance of early detection and intervention.

1. Introduction

The demands of living with type 1 diabetes extend far beyond glucose levels and insulin dosing—in the domain of eating and dietary behaviors, diabetes self-management touches upon the complex balance of hunger, satiety, and physical and emotional well-being, sometimes at the cost of disordered eating. Adolescents and young adults with type 1 diabetes have nearly five times the prevalence of eating disorders (ED) compared to their peers without diabetes,[1] with a strong association to bulimia nervosa and binge eating disorder.[2] However, due to the unique characteristics of type 1 diabetes, disordered eating can occur without fully meeting diagnostic criteria for specific ED. In such cases, non-specific diagnoses like "eating disorder, not otherwise specified" may be applied. To describe disordered eating outside of diagnostic categories, the term "disordered eating behaviors" (DEB) is commonly used in research and practice. Studies report that 21.1 % to 29.7 % of adolescents and young adults exhibit DEB,[3–5] with a recent meta-analysis estimating a prevalence of 24 %.[6]

A common diabetes-specific DEB is insulin purging,[6] intentionally reducing or omitting insulin to excrete glucose in the urine and minimize energy utilization. While this may lead to weight loss, it inevitably worsens metabolic control and increases the risk of diabetic ketoacidosis (DKA). Other DEB include compensatory behaviors such as self-induced vomiting, laxative abuse or excessive exercise, binge eating (sometimes in response to hypoglycemia), restrictive eating, emotional eating, and external eating (triggered by food stimuli irrespective of hunger or satiety).[7–10]

DEB in type 1 diabetes is multifactorially determined, influenced by both metabolic and psychosocial factors. A significant weight loss due to the catabolic state prior to diagnosis, followed by a rapid weight gain under insulin therapy, may contribute to body dissatisfaction.[3,34] Diabetes self-management—requiring carbohydrate counting, glucose monitoring, and insulin adjustments—can disrupt natural hunger and satiety cues, particularly when treating hypoglycemia.[18] Additionally, intensive insulin therapy has been associated with weight gain beyond the initial rehydration phase, potentially reinforcing weight

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concerns.[14,35] Other established risk factors include female gender, adolescence or young adulthood, anxiety, and depressive symptoms.[18] Diabetes-related psychosocial risk factors, like diabetes-related distress and fear of hypoglycemia have received less research attention regarding their role in DEB. While some studies have found associations between diabetes distress and several DEB,[11–13] others have not.[9] Fear of hypoglycemia has been identified as a potential barrier to physical activity,[14,15] which could be relevant in the context of stress management, weight control and body image. Moreover, fear of hypoglycemia may also lead to the intentional maintenance of high glucose levels,[15] further complicating the interplay between DEB and diabetes self-management.

DEB and ED are closely associated with poorer metabolic control[3,5,9,16,17] and life-threatening consequences. Adolescents and young adults with type 1 diabetes and ED face a more than threefold increased risk of hospitalization due to DKA and a nearly sixfold increased mortality compared to their peers without ED.[1]

Despite growing evidence on DEB risk factors in type 1 diabetes, research is particularly limited regarding symptom severity, comorbid psychopathologies, and other diabetes-related concomitants. Broadley and colleagues emphasize the need for a more nuanced characterization of DEB (and eventually ED) in this population.[18] They advocate for an integrated approach that incorporates behavioral patterns, clinical features, and medical risks to inform tailored interventions and preventive strategies. In line with this perspective, the present study aims to identify distinct DEB subtypes in a large sample of adults with type 1 diabetes and to characterize these subtypes regarding their clinical features, (diabetes-specific) psychosocial well-being, and diabetes-related complications.

2. Materials and methods

2.1. DEBBI study

We used data of the Disordered Eating Behaviors and Eating Disorders in Diabetes Type I (DEBBI) online study that investigates DEB and its correlates in adults with type 1 diabetes.[19] The data collection took place between September 15, 2022, and July 31, 2024. The study includes four assessment points (baseline; follow-ups at 6, 12, and 18 months) and aims to (1) identify and characterize subtypes of DEB, (2) identify key psychosocial and diabetes-specific risk factors for DEB, and (3) explore the course of DEB over time. The study was approved by the Ethics Committee of the State Medical Chamber of Rhineland-Palatine, Germany (ID 2021-16040) and preregistered with the German Clinical Trials Register (DRKS00028833). Participants provided written informed consent. Details on the study protocol have been previously described.[19]

2.2. Participants and recruitment

Adults with a diagnosis of type 1 diabetes for at least 12 months were eligible to participate, independent of former or current DEB or ED. The survey was disseminated via social media, journals and other websites for people with diabetes and healthcare professionals, and offline in local diabetes clinics. Inclusivity in recruitment materials was emphasized to reduce potential selection biases, for example by explicitly inviting men that are often underrepresented in research into DEB, and utilizing a diverse set of channels for recruiting.

2.3. Measurements

The primary measure of *DEB* was the Diabetes Eating Problem Survey–Revised (DEPS-R), a 16-item questionnaire specific to diabetes-related behaviors, including insulin omission or underdosing and self-management problems related to eating and weight-management behaviors.[20,21] Items are rated on a 6-point Likert scale, with higher scores indicating more frequent DEB. A sum score (range 0–80) of $\geq$ 20 suggests clinically relevant DEB.

To assess the *deliberate induction of hypoglycemia*, participants were asked to answer the item "I have intentionally induced low glucose levels to consume fast-acting carbohydrates or foods that I otherwise would not 'allow' myself to eat", using a 6-point Likert scale (0 = never, 5 = often).

Diabetes self-management was assessed using the German version of the Diabetes Self-Management Questionnaire (DSMQ), which measures self-management across the four subscales glucose management, dietary control, physical activity, and healthcare use.[22] Items are rated on a 4-point Likert scale, with higher scores indicating more effective self-management (range 0–10). The scale demonstrated excellent internal consistency in our sample.

Diabetes-related distress was measured with the German version of the Problem Areas in Diabetes Scale (PAID), a 20-item questionnaire assessing the emotional burden related to diabetes. Items are rated on a 5-point Likert scale, and scores are summed and transformed into a total score (range 0–100). A higher score indicates greater distress, with scores $\geq$ 40 suggesting elevated distress.[23]

Fear of hypoglycemia was assessed using the German version of the Hypoglycemia Fear Survey-II (HFS-II), a well-validated instrument designed to measure fear of hypoglycemia.[24] For this study, we used the more concise HFS-II short form, which comprises 11 items.[25] Each item is scored on a 5-point Likert scale, with higher total sum scores indicating greater levels of fear of hypoglycemia (total score range 0–44).

Depressive symptoms were assessed using an 8-item version of the German Patient Health Questionnaire (PHQ) depression module, measuring the frequency of depressive symptoms over the past two weeks on a 4-point Likert scale.[26] Higher scores indicate greater depressive symptom severity (range 0–24).

Trait anxiety was measured using the trait subscale of the German State-Trait Anxiety Inventory (STAI-T).[27] The trait subscale includes 20 items rated on a 4-point Likert scale, assessing general levels of anxiety (range 20–80).

Reliability estimates for all scales are included in the Supplemental Material (Table S1).

2.4. Demographic and clinical data

Participants self-reported demographics, their most recent laboratory HbA_{1c} value, height, weight, type of insulin therapy, use of continuous glucose monitoring (CGM), the number of hypoglycemic or hyperglycemic events in the past 12 months that required external assistance and whether they had been diagnosed with an ED by a health care professional.

2.5. Statistical analysis

DEBBI baseline data were analyzed. Descriptive statistics were calculated to summarize demographic, diabetes-related and psychosocial variables. Preliminary analyses were conducted on the DEPS-R items to determine their suitability as indicators for a latent profile analysis (LPA). LPA was selected because it enables the identification of subgroups within the sample based on shared patterns of DEB. Following the LPA, comparisons between the derived profiles were performed. The LPA was conducted using the tidyLPA package[28] in R version 4.2.2. Other analyses were conducted using IBM SPSS Statistics Version 29.0.2.0.

2.5.1. Indicator selection and preliminary analyses

The characterization of DEB was based on the DEPS-R as it is designed and widely used to assess DEB in type 1 diabetes. However, several DEB, such as insulin purging, are captured by multiple items. To enhance the differentiation of behaviors, cognitions and consequences of DEB for the LPA, the DEPS-R items were assigned to six distinct domains: restrained eating (item 2), eating-related loss of control (items 3,

5, 14, 15), intentional vomiting (item 8), self-management problems (items 7, 12), preoccupation with thinness and/or weight (items 1, 6, 11, 16), and maintaining high glucose (items 4, 9, 10, 13). The structural validity of these domains and indicators were tested via confirmatory factor analysis (CFA), and the reliabilities of the domain scales were assessed using Cronbach's α if they included multiple items.

2.5.2. Main analyses

LPA model selection was guided by several fit indices, including the Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), entropy, minimum and maximum classification probabilities, smallest and largest class sizes, and Bootstrap Likelihood Ratio Test (BLRT). Differences between profiles on the LPA indicators were tested using analysis of variance (ANOVA) with Games-Howell post-hoc tests, as the assumption of homogeneity of variances was violated. Bonferroni corrections were applied, and Hedge's g was calculated as effect size (accounting for differences in group sizes).

Subsequent analyses examined differences between profiles on demographic, clinical, and psychosocial variables. Due to the small number of participants who identified their gender as "diverse", these cases ($n = 4$) were excluded from the analysis comparing gender differences. This decision was taken to ensure robustness of the results while acknowledging the importance and representation of this group.

For categorical variables, Fisher's exact tests were used, with Monte Carlo simulations (10,000 iterations) for variables with more than two levels. For continuous variables, none of which met the assumption of normality, Kruskal-Wallis tests were performed as a non-parametric alternative to ANOVA, followed by pairwise Mann-Whitney *U* tests for post-hoc comparisons. Effect sizes were calculated as phi (ϕ) for categorical variables and Hedges' g for continuous variables, accounting for group sizes differences.

2.5.3. Missing data

The analyses included complete records. However, BMI could not be calculated for 22 participants, as weight and height were optional questions. BMI was analyzed for the descriptive statistics of the full sample, and to compare differences between profiles. Here, no imputation methods were applied, and these analyses were conducted with the available cases.

3. Results

3.1. Participants

$N = 645$ participants with type 1 diabetes completed the baseline survey. Participants mean age was 42.94 (SD 15.84) years. 70.16 % ($n = 459$) identified as women. The DEPS-R cut-off score of ≥ 20 was met by 32.71 % ($n = 211$) of the sample. Sixty-four participants (9.92 %) reported an ED diagnosis by a healthcare professional. Further demographic and clinical characteristics are presented in Table 1.

Table 1
Sample characteristics.

N = 645	
Age, *mean* (*SD*), years	42.94 (15.84)
Women	459 (71.16)
Highest education level achieved	
Primary school or no formal education	1 (0.16)
Secondary school	56 (8.86)
High school	86 (13.33)
Vocational training	182 (28.22)
Bachelor's degree or higher	313 (48.53)
Other	7 (1.09)
Employment status	
Employed full-time	283 (43.88)
Employed half-time	111 (17.21)
Marginally or irregularly employed or retired	32 (4.96)
Seeking employment	13 (2.02)
Household and family responsibilities	105 (16.28)
In school, college or vocational training	101 (15.66)
Relationship status	
Single	143 (22.17)
In a relationship	189 (29.30)
Married	296 (45.89)
Other	17 (2.64)
Household composition	
Single-person household	136 (21.09)
Living with roommates, friends	34 (5.27)
Living with partner, family	475 (73.64)
Diabetes duration, *mean* (*SD*), years	22.13 (15.16)
Insulin therapy	
CT	6 (0.93)
MDI	262 (40.62)
CSII	197 (30.54)
AID	180 (27.91)
CGM use	587 (91.01)
HbA_{1c}, %, *mean (SD)*	6.94 (1.11)
HbA_{1c}, mmol/mol, *mean (SD)*	52.34 (12.08)
BMI*, kg/m^2, *mean (SD)*	26.29 (5.52)
Disordered eating, DEPS-R	
Total DEPS-R score, *mean (SD)*	16.65 (13.45)
DEPS-*R* < 20	434 (67.29)
DEPS-R ≥ 20	211 (32.71)
Eating disorder diagnosis	64 (9.92)
Anorexia nervosa	16 (2.48)
Bulimia nervosa	20 (3.10)
Binge-eating	24 (3.72)
Eating disorder not otherwise specified	20 (3.10)

Note. Data reported as *n* (%) unless otherwise indicated. CT, conventional insulin therapy; MDI, multiple daily injections; CSII, continuous subcutaneous insulin infusion; AID, automated insulin delivery. CGM, continuous glucose monitoring; HbA_{1c}, glycated hemoglobin A_{1c}; BMI, body mass index; DEPS-R, Diabetes Eating Problem Survey–Revised. *$n = 623$.

3.2. Preliminary results

The structural validity of the six DEPS-R indicators was evaluated using CFA. The model achieved acceptable fit based on comparative fit indices (CFI/TLI), residual-based measures (RMSEA/SRMR), and χ^2 values (Supplemental Table S2). Factor loadings indicated adequate to good item representation within factors (Supplemental Table S3). Intercorrelations indicated moderate to strong associations between the subscales without evidence of redundancy (Supplemental Table S4). Internal consistency values ranged from $\alpha = 0.68$ to $\alpha = 0.85$, reflecting acceptable to good reliability.

3.3. Latent profile analysis

LPA can be conducted at a Cohen's d of 0.8 with a minimum sample size of $N = 500$ when at least 10 indicators are used to define latent classes.[29] Given that our model includes six indicators, our final sample of $N = 645$ provided a robust basis for identifying distinct profiles. Several LPAs were conducted, testing solutions with 1–8 classes to determine the optimal parameterization model and number of profiles representing DEB subtypes. A latent profile model that assumes equal variances across profiles and a common covariance structure between indicators across all profiles[28] demonstrated the best fit. Table 2 provides an overview of the fit indices, entropies, class sizes, and classification probabilities for each solution.

AIC and BIC decreased substantially from the 1-class to the 6-class solution, suggesting that additional classes improved model fit. However, after the 6-class solution, the reduction in these criteria became minimal. Additionally, the BLRT remained significant for all solutions

Table 2
Latent profile analysis fit indices for 1 to 8 classes.

Classes	AIC	BIC	Entropy	Minimum classification probability	Maximum classification probability	Smallest class size	Largest class size	BLRT *p* value
1	9542.2	9662.9	1.000	1.000	1.000	645	645	.010
2	9477.8	9629.7	0.747	0.892	0.939	202	443	.010
3	8536.4	8719.7	0.907	0.889	1.000	27	482	.010
4	8550.0	8764.6	0.575	0.000	1.000	0	474	.010
5	8245.6	8491.4	0.896	0.671	1.000	17	464	.010
6	7828.2	8105.3	0.696	0.250	1.000	15	401	.010
7	7842.2	8150.6	0.636	0.000	1.000	0	381	.570
8	7813.3	8153.0	0.634	0.000	1.000	0	387	.030

Note. AIC, Akaike information criterion; BIC, Bayesian information criterion; BLRT, Bootstrap likelihood ratio test.

except the 7-class model ($p = 0.570$), suggesting that models with up to six profiles were statistically superior to their respective lower-class counterparts.

Entropy values, which reflect the clarity of class separation, ranged from 0.575 to 0.907. The entropy for the 5-class solution was 0.896, indicating good class separation, while the 6-class solution showed a notable drop to 0.696. The minimum classification probability also decreased considerably from 0.671 in the 5-class solution to 0.250 in the 6-class solution signaling lower classification stability and less reliable profile assignments in the 6-class model. Furthermore, the 6-class solution artificially split the "minimal or no DEB" profile into two indistinguishable groups. We opted for the 5-class model as it provided clear profile distinctions with higher classification reliability.

3.4. Latent profiles of DEB and their demographic, diabetes-related and psychosocial characteristics

Fig. 1 displays the five identified latent profiles of DEB as standardized means on the DEB indicators. Means on demographic, diabetes-related and psychosocial characteristics are provided in Table 3. Details for LPA indicator variables and demographic, diabetes-related and psychosocial variables, along with results and effect sizes of post-hoc comparisons, are provided in the Supplemental Material (Tables S5, S6). The five profiles significantly differed in demographic, diabetes-related and psychosocial variables (Table 3). No significant differences were found for the number of late complications ($p = 0.452$), education level ($p = 0.310$), employment status ($p = 0.154$), relationship status ($p = 0.153$), household composition ($p = 0.180$) and insulin therapy ($p = 0.126$).

3.4.1. Profile 1: minimal or no DEB

More than two thirds of the sample ($n = 464$) were categorized in profile 1, reporting minimal or no DEB. Scores on all six DEB indicators were consistently low, indicating healthy eating and no significant problems with diabetes self-management (Fig. 1). Participants in this group reported the lowest levels of diabetes-related distress, fear of hypoglycemia, depression and anxiety (Table 3).

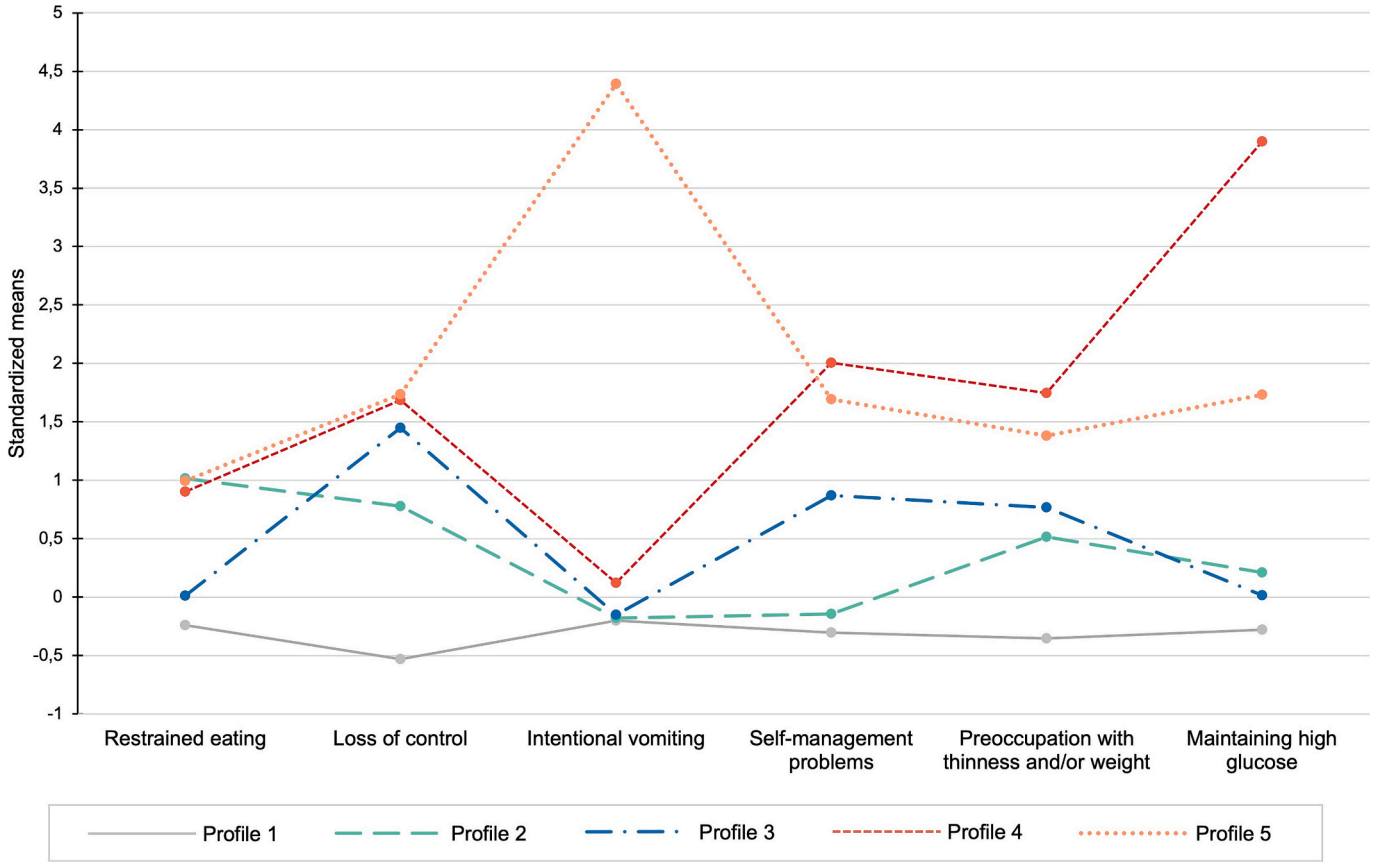

Fig. 1. Depiction of the five latent profiles defined by pattern of standardized means on six indicators of disordered eating behaviors (DEB).

Table 3
Demographic, diabetes-related and psychosocial characteristics across five profiles.

	Profile 1 $n = 464$	Profile 2 $n = 56$	Profile 3 $n = 82$	Profile 4 $n = 17$	Profile 5 $n = 26$
DEPS-R score (0.00–74.00)	$10.43\ (6.68)_a$	$26.04\ (7.43)_b$	$29.54\ (8.64)_b$	$50.24\ (14.34)_c$	$44.85\ (16.82)_c$
Age (18–88)	$45.34\ (16.14)_a$	$40.00\ (14.39)_{ab}$	$36.33\ (12.48)_b$	$33.76\ (15.17)_b$	$33.27\ (10.49)_b$
Women, *n* (%)	$300\ (64.66)_a$	$51\ (91.07)_b$	$68\ (82.93)_b$	$15\ (88.24)_{ab}$	$25\ (96.15)_b$
BMI (16.4–56.4)	$25.66\ (4.64)_a$	$27.55\ (6.78)_{ab}$	$29.60\ (7.25)_b$	$26.70\ (7.10)_{ab}$	$23.68\ (5.59)_a$
HbA_{1c}, %, (4.50–15.00)	$6.79\ (0.86)_a$	$6.70\ (1.00)_a$	$7.25\ (1.14)_b$	$9.22\ (1.87)_c$	$7.68\ (2.05)_{ab}$
HbA_{1c}, mmol/mol (25.68–140.44)	$50.70\ (9.39)_a$	$49.67\ (10.94)_a$	$55.72\ (12.41)_b$	$77.24\ (20.45)_c$	$60.49\ (22.44)_{ab}$
CGM use, *n* (%)	$428\ (92.24)_a$	$53\ (94.64)_a$	$72\ (87.80)_{ab}$	$11\ (64.71)_b$	$23\ (88.46)_{ab}$
Eating disorder diagnosis, *n* (%)	$14\ (3.02)_a$	$9\ (16.07)_b$	$18\ (21.95)_b$	$6\ (35.29)_{bc}$	$17\ (65.38)_c$
Deliberately inducing hypoglycemia (0.00–5.00)	$0.66\ (1.15)_a$	$1.23\ (1.69)_{ab}$	$1.49\ (1.48)_b$	$1.35\ (1.69)_{ab}$	$2.08\ (1.65)_b$
DSMQ score (0.36–10.00)	$7.48\ (1.31)_a$	$6.93\ (1.51)_a$	$5.27\ (1.66)_b$	$3.75\ (1.65)_c$	$4.98\ (1.73)_{bc}$
PAID score (0.00–100.00)	$26.87\ (19.75)_a$	$45.54\ (18.89)_b$	$52.24\ (19.47)_b$	$59.56\ (21.12)_b$	$58.17\ (20.68)_b$
HFS-II score (0.00–42.00)	$11.91\ (7.92)_a$	$17.09\ (9.09)_b$	$16.95\ (8.41)_b$	$18.06\ (8.36)_b$	$18.04\ (7.81)_b$
PHQ-8 score (0.00–24.00)	$6.85\ (4.77)_a$	$12.36\ (5.27)_b$	$13.52\ (5.40)_b$	$15.59\ (5.32)_b$	$16.19\ (5.91)_b$
STAI-T score (20.00–80.00)	$40.94\ (12.22)_a$	$52.54\ (11.65)_b$	$54.70\ (11.01)_b$	$56.94\ (11.05)_b$	$60.65\ (13.92)_b$
Severe hypoglycemic events in the last 12 months (0–20)	$0.25\ (1.45)_a$	$0.29\ (1.17)_{ab}$	$0.72\ (1.83)_b$	$0.12\ (0.49)_{ab}$	$0.96\ (2.24)_b$
Severe hyperglycemic events in the last 12 months (0–10)	$0.05\ (0.50)_a$	$0.04\ (0.19)_{ab}$	$0.06\ (0.24)_{ab}$	$0.18\ (0.39)_b$	$0.54\ (1.36)_b$

Note. Data reported as *mean* (*SD*) unless otherwise indicated. The first column contains the range of each variable for the complete sample (N = 645). Means sharing a subscript in a row indicate means that are not significantly different. BMI, body mass index; HbA_{1c}, glycated hemoglobin A_{1c}; CGM, continuous glucose monitoring.

3.4.2. Profile 2: restrained eating

Profile 2 ($n = 56$) is the first profile to exhibit notable DEB. It is primarily characterized by elevated levels of restrained eating (Fig. 1). Bulimic behaviors, such as insulin purging, play virtually no role in this group. While loss of control is also reported to some extent and preoccupation with thinness and/or weight is higher compared to profile 1, diabetes self-management does not yet appear to be significantly affected.

3.4.3. Profile 3: disinhibited eating

Compared to profile 2, profile 3 ($n = 82$) did not report restrained eating but higher loss of control (Fig. 1). This disinhibition does not appear to be compensated for by bulimic behaviors, such were virtually absent in this group. However, they deliberately induced hypoglycemia more often to be able to eat 'forbidden' foods. Participants in this group reported more diabetes management problems related to eating behaviors, which was also reflected in poorer DSMQ scores and higher mean HbA_{1c} levels compared to the previous two profiles. This group exhibited the highest mean BMI, which differed significantly from profile 1 (Table 3).

3.4.4. Profile 4: maintaining high glucose

The smallest profile ($n = 17$) displayed a broad spectrum of DEB (Fig. 1). Participants exhibited similar restrained eating as profile 2 and comparable loss of control to profile 3, but with markedly higher preoccupation with thinness and/or weight. The defining feature of this group is maintaining high glucose to promote weight loss. The frequent insulin restriction was reflected in substantial problems with diabetes self-management, including poorest self-management quality and highest mean HbA_{1c} levels across all profiles (Table 3). Simultaneously, this group reported a higher frequency of hyperglycemic events in the past 12 months. However, the number of severe hypoglycemic events did not differ significantly from the group without DEB.

3.4.5. Profile 5: dual compensatory behaviors

While participants in profile 5 ($n = 26$) also reported to maintain high glucose levels as a compensatory behavior, the defining feature of this group is intentional vomiting (Fig. 1). They also most frequently reported to intentionally induce hypoglycemia. Interestingly, compared to profile 4, DEB appears to be less reflected in glycemic control, as the mean HbA_{1c} levels do not differ significantly from profiles 1–3. Profile 5 reported the lowest mean BMI (although it differed significantly only from profile 4) and had the highest proportion of participants with a diagnosed eating disorder, although this proportion did not differ significantly from profile 4 (Table 3).

4. Discussion

Understanding the specific presentation of DEB in individuals with type 1 diabetes is crucial for a timely diagnosis and effective interventions. We identified four distinct DEB subtypes, highlighting the heterogeneity of these behaviors. Most participants (71.94 %) exhibited minimal or no DEB, the remaining individuals were distributed across what we classify as two moderate and two severe subtypes: restrained eating (moderate DEB), disinhibited eating (moderate DEB), maintaining high glucose (severe DEB), and dual compensatory behaviors (severe DEB). Moderate subtypes were characterized by restrained eating and feelings of loss of control related to eating without the use of compensatory behaviors, while severe subtypes presented a combination of cognitive preoccupation with thinness and/or weight and compensatory strategies to counteract loss of control. Eating disorder diagnoses were rarest in the group with minimal or no DEB and most frequent in the severe profiles. The subtypes corresponded with significant differences in clinical and diabetes-specific factors, including BMI, self-management quality, HbA_{1c} levels, and number of acute complications. The proportion of CGM users was smaller in more severe DEB subtypes. Individuals with DEB tended to be younger and were more likely to be women, consistent with established risk factors.[2,6] Other demographic variables (e.g., education level, employment status) did not differ across subtypes. All DEB subtypes, even those classified as moderate, were linked to substantial psychosocial burden, with heightened fear of hypoglycemia, clinically relevant diabetes distress (scores exceeding the cut-off of 40), and depressive and anxiety symptoms in the moderate to severe range. A rarely examined behavior, deliberate induction of hypoglycemia, was more common in profiles characterized by loss of control or compensatory behaviors.

With a prevalence of 32.71 %, the occurrence of DEB in our sample was slightly higher than the pooled prevalence of 27 % reported in a meta-analysis of studies using the DEPS-R.[6] Among participants with severe DEB, frequent insulin restriction was a prominent behavior. Specifically, 25 % of participants reported underdosing insulin at least "sometimes" slightly higher but comparable to the 21 % prevalence of insulin omission reported in a meta-analysis.[6]

Although less frequent, 4.2 % of participants in our sample reported intentional vomiting at least "sometimes". This aligns with prior findings from Colton et al.,[7] who reported a prevalence of 4.9–8.5 % for intentional vomiting among young women with type 1 diabetes, and Broadley et al.,[30] who similarly found a prevalence of 4.9 %. Intentional vomiting remains under-researched in the context of type 1 diabetes, yet in our study, it emerged as the defining feature of one of the severe DEB subtypes. Meta-analytic evidence indicates no significant difference in

the prevalence of intentional vomiting between individuals with type 1 diabetes and metabolically healthy controls,[2] underscoring the importance of considering intentional emesis in the diagnostic process. Our findings suggest that severe DEB may be present even in the absence of frequent insulin purging or poor metabolic control.

DEPS-R total score or scores exceeding the cut-off of 20 have been previously associated with higher HbA_{1c} and an increased risk for diabetes complications,[31] but do not necessarily indicate the severity of DEB.[32] While higher DEPS-R scores generally reflect more frequent and numerous eating disorder symptoms, scores alone do not capture their heterogeneity: while all DEB profiles exceeded the cut-off score, there were no significant differences in the mean DEPS-R scores between profiles 2 and 3 (26.04 ± 7.43 vs. 29.54 ± 8.64) or between profiles 4 and 5 (50.24 ± 14.34 vs. 44.85 ± 16.82), despite differences in behavioral patterns, self-management, and HbA_{1c} levels. Similarly, Merwin et al. identified four latent DEB profiles, characterized by low ED pathology, overeating, binge-eating, or bulimic behaviors, respectively.[32] These profiles corresponded not only to behaviors in real-life, as evidenced by ecological momentary assessment of binge eating frequency and insulin restriction, but also to HbA_{1c} levels and mean glucose values.[32] Our findings align with this research, underscoring the clinical utility of looking beyond the DEPS-R cut-off or total score. Examining individual DEPS-R domains and items (e.g., "I would rather be thin than to have good control over my diabetes", "I make myself vomit"[20]) could be a first step toward ensuring that an individual's unique experience and challenges are fully recognized and addressed.

Building on these findings, understanding specific DEB subtypes is not only diagnostically valuable but could also serve as a foundation for a more tailored diabetes care and psychosocial interventions, individualized to meet the needs of type 1 diabetes with distinct DEB patterns. This perspective is supported by an innovative integrated approach proposed by Ismail et al.,[33] who successfully combined diabetes and mental health treatments within a collaborative care model. Their proof-of-concept study demonstrated improvements in clinical outcomes, including HbA_{1c} levels, hospital admissions for DKA and severe hypoglycemia.[33] These promising results underline the importance of an early characterization of DEB, as detailed behavioral insights can provide a key starting point for personalized and effective intervention strategies.

Several limitations of this study warrant consideration. First, the online recruitment and data collection likely introduced some self-selection bias, and individuals with greater digital literacy may have been overrepresented despite additional efforts to recruit participants offline through clinics and diabetes care centers. A potential selection bias may also be reflected in the relatively high proportion of eating disorder diagnoses (9.92 %), which substantially exceeds, for example, the 1.6 % prevalence found among female adolescents in the Diabetes Patienten Verlaufsdokumentation (DPV) registry.[16] Second, all data were self-reported and not corroborated by objective measures such as clinical records or healthcare provider assessments, which likely has introduced some recall or reporting biases. Third, the demographics of our sample impose additional constraints on the generalizability of the results. Participants identified predominantly as female, were highly educated, and most were in a committed partnership and living with their partner or family. Additionally, comparisons between the DEB profiles should be interpreted with caution due to the small group sizes of the severe subtypes. While Fisher's Exact Test was employed for categorical variables to account for the small group sizes, the limited statistical power likely reduced the sensitivity to detect significant differences. For instance, while there was a clear trend toward a higher mean number of severe hyperglycemic events in profile 4 (0.54, SD 1.36) compared to profile 2 (0.04, SD 0.19), this difference was not statistically significant. Future studies with larger and more diverse samples are needed to explore the broader applicability of these findings and to examine how demographic and socioeconomic factors may influence the presentation and impact of DEB in type 1 diabetes. Furthermore, it remains unclear whether the moderate DEB profiles identified in this study represent stable subtypes or potential precursors to more severe disordered eating. Early identification and monitoring of behaviors such as restrained eating could be paramount, as these may progress to more severe forms of DEB over time. Future analyses will explore the stability and potential transitions between DEB subtypes with DEBBI follow-up data.

Despite these limitations, the present findings inform clinical practice. Our study underscores the heterogeneity of DEB in type 1 diabetes and highlights the need to look beyond DEPS-R sum scores to consider specific behavioral patterns. The identified DEB subtypes differ in their clinical features, behavioral characteristics, and diabetes self-management, implying that hazardous DEB in type 1 diabetes is not synonymous with insulin purging alone. Even moderate forms of DEB were associated with significant psychosocial distress, emphasizing the importance of an early detection and tailored interventions.

CRediT authorship contribution statement

Lilli-Sophie Priesterroth: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Writing – original draft. **Jennifer Grammes:** Conceptualization, Writing – review & editing. **Thomas Kubiak:** Conceptualization, Resources, Supervision, Writing – review & editing.

Prior presentation

Not applicable.

Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used *DeepL* in order to improve readability and language. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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Declaration of competing interest

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jdiacomp.2025.109067.

Data availability

The datasets analyzed in the current study are available from the corresponding author upon request.

References

1. Gibbings NK, Kurdyak PA, Colton PA, Shah BR. Diabetic ketoacidosis and mortality in people with type 1 diabetes and eating disorders. *Diabetes Care*. 2021;44:1783–1787.

2. Dean YE, Motawea KR, Aslam M, Pintado JJL, Popoola-Samuel HAO, Salam M, et al. Association between type 1 diabetes mellitus and eating disorders: a systematic review and meta-analysis. *Endocrinol Diabetes Metab*. 2024;7, e00473.
3. Propper-Lewinsohn T, Gillon-Keren M, Shalitin S, Elran-Barak R, Yackobovitch-Gavan M, Fayman G, et al. Disordered eating behaviours in adolescents with type 1 diabetes can be influenced by their weight at diagnosis and rapid weight gain subsequently. *Diabet Med*. 2023;40, e15166.
4. Troncone A, Affuso G, Cascella C, Chianese A, Zanfardino A, Iafusco D, et al. Prevalence and multidimensional model of disordered eating in youths with type 1 diabetes: results from a nationwide population-based study. *J Pediatr Psychol*. 2023;48:731–739.
5. Nip ASY, Reboussin BA, Dabelea D, Bellatorre A, Mayer-Davis EJ, Kahkoska AR, et al. Disordered eating behaviors in youth and young adults with type 1 or type 2 diabetes receiving insulin therapy: the SEARCH for diabetes in youth study. *Diabetes Care*. 2019;42:859–866.
6. Niemelä PE, Leppänen HA, Voutilainen A, Möykkynen EM, Virtanen KA, Ruusunen AA, et al. Prevalence of eating disorder symptoms in people with insulin-dependent-diabetes: a systematic review and meta-analysis. *Eat Behav*. 2024;53, 101863.
7. Colton PA, Olmsted MP, Daneman D, Farquhar JC, Wong H, Muskat S, et al. Eating disorders in girls and women with type 1 diabetes: a longitudinal study of prevalence, onset, remission, and recurrence. *Diabetes Care*. 2015;38:1212–1217.
8. Baechle C, Hoyer A, Stahl-Pehe A, Castillo K, Toennies T, Lindner LME, et al. Course of disordered eating behavior in young people with early-onset type I diabetes: prevalence, symptoms, and transition probabilities. *J Adolesc Health*. 2019;65:681–689.
9. Huisman SD, Hendrieckx C, Bot M, Pouwer F, Nefs G. Prevalence, associations and health outcomes of binge eating in adults with type 1 or type 2 diabetes: results from diabetes MILES – the Netherlands. *Diabet Med*. 2023;40, e14953.
10. Schober E, Wagner G, Berger G, Gerber D, Mengl M, Sonnenstatter S, et al. Prevalence of intentional under- and overdosing of insulin in children and adolescents with type 1 diabetes. *Pediatr Diabetes*. 2011;12:627–631.
11. Araia E, King RM, Pouwer F, Speight J, Hendrieckx C. Psychological correlates of disordered eating in youth with type 1 diabetes: results from diabetes MILES Youth—Australia. *Pediatr Diabetes*. 2020;21:664–672.
12. Powers MA, Richter SA, Ackard DM, Craft C. Diabetes distress among persons with type 1 diabetes: associations with disordered eating, depression, and other psychological health concerns. *Diabetes Educ*. 2017;43:105–113.
13. Embaye J, Bassi G, Dingemans AE, Doesborg L, Snoek FJ, De Wit M. Associations between disordered eating behaviour, diabetes distress and emotion regulation strategies in adults with type 1 diabetes: results from a dutch-italien cross-sectional study. *Diabet Med*. 2023;40, e15122.
14. Driscoll KA, Corbin KD, Maahs DM, Pratley R, Bishop FK, Kahkoska A, et al. Biopsychosocial aspects of weight management in Type 1 diabetes: a review and next steps. *Curr Diab Rep*. 2017;17:58.
15. Martyn-Nemeth P, Schwarz Farabi S, Mihailescu D, Nemeth J, Quinn L. Fear of hypoglycemia in adults with type 1 diabetes: impact of therapeutic advances and strategies for prevention - a review. *J Diabetes Complicat*. 2016;30:167–177.
16. Reinehr T, Dieris B, Galler A, Teufel M, Berger G, Stachow R, et al. Worse metabolic control and dynamics of weight status in adolescent girls point to eating disorders in the first years after manifestation of type 1 diabetes mellitus: findings from the diabetes Patienten Verlaufsdokumentation Registry. *J Pediatr*. 2019;207:205–212.
17. Wisting L, Skrivarhaug T, Dahl-Jørgensen K, Rø Ø. Prevalence of disturbed eating behavior and associated symptoms of anxiety and depression among adult males and females with type 1 diabetes. *J Eat Disord*. 2018;6:28.
18. Broadley MM, Zaremba N, Andrew B, Ismail K, Treasure J, White MJ, et al. 25 Years of psychological research investigating disordered eating in people with diabetes: what have we learnt? *Diabet Med*. 2020;37:401–408.
19. Priesterroth L, Grammes J, Strohm EA, Kubiak T. Disordered eating behaviours and eating disorders in adults with type 1 diabetes (DEBBI): rational and design of an observational longitudinal online study. *BMJ Open*. 2022;12, e064863.
20. Markowitz JT, Butler DA, Volkening LK, Antisdel JE, Anderson BJ, Laffel LMB. Brief Screening Tool for Disordered Eating in Diabetes: internal consistency and external validity in a contemporary sample of pediatric patients with type 1 diabetes. *Diabetes Care*. 2010;33:495–500.
21. Saßmann H, Albrecht C, Busse-Widmann P, Hevelke LK, Kranz J, Markowitz JT, et al. Psychometric properties of the German version of the Diabetes Eating Problem Survey-Revised: additional benefit of disease-specific screening in adolescents with type 1 diabetes. *Diabet Med*. 2015;32:1641–1647.
22. Schmitt A, Gahr A, Hermanns N, Kulzer B, Huber J, Haak T. The Diabetes Self-Management Questionnaire (DSMQ): development and evaluation of an instrument to assess diabetes self-care activities associated with glycaemic control. *Health Qual Life Outcomes*. 2013;11:138.
23. Schmitt A, Reimer A, Kulzer B, Haak T, Ehrmann D, Hermanns N. How to assess diabetes distress: comparison of the problem areas in diabetes scale (PAID) and the diabetes distress scale (DDS). *Diabet Med*. 2016;33:835–843.
24. Schipfer M, Ehrmann D, Hermanns N, Kulzer B, Haak T. Evaluation des Fragebogens zu Unterzuckerungen bei Erwachsenen mit Typ-1-Diabetes (HFS). *Diabetol Stoffwechs*. 2016;19. s-0036-1580877.
25. Grabman J, Vajda Bailey K, Schmidt K, Cariou B, Vaur L, Madani S, et al. An empirically derived short form of the Hypoglycaemia Fear Survey II. *Diabet Med*. 2017;34:500–504.
26. Kocalevent RD, Hinz A, Brähler E. Standardization of the depression screener Patient Health Questionnaire (PHQ-9) in the general population. *Gen Hosp Psychiatry*. 2013;35:551–555.
27. Laux L, Glanzmann P, Schaffner P, Spielberger CD. *Das State-Trait-Angstinventar*. Weinheim: Beltz; 1981.
28. Rosenberg J, Beymer P, Anderson D, Van Lissa Cj, Schmidt J. tidyLPA: an R package to easily carry out latent profile analysis (LPA) using open-source or commercial software. *J Open Source Softw*. 2018;3:978.
29. Tein JY, Coxe S, Cham H. Statistical power to detect the correct number of classes in latent profile analysis. *Struct Equ Model Multidiscip J*. 2013;20:640–657.
30. Broadley MM, Bishop T, White MJ, Andrew B. The relationship between attentional bias to food and disordered eating in females with type 1 diabetes. *Appetite*. 2019;140:269–276.
31. Ryman B, MacIsaac J, Robinson T, Miller MR, Herold GP. Assessing the clinical utility of the diabetes eating problem survey-revised (DEPS-R) in adolescents with type 1 diabetes. *Endocrinol Diabetes Metab*. 2019;2, e00067.
32. Merwin RM, Dmitrieva NO, Moskovich AA, Warnick JL, Goebel-Fabbri AE, Topor LS, et al. Profiles of disordered eating behaviour in type 1 diabetes using the DEPS-R and behaviour and glycaemic outcomes in a real-life setting. *Diabet Med*. 2024;41, e15314.
33. Ismail K, Turner D, Brown J, Rosenthal M, Ayis S, Oliver N, et al. An integrated diabetes and mental health intervention for people with type 1 diabetes and severe disordered eating: a prospective proof-of-concept cohort study. *Lancet Diabetes Endocrinol*. 2024;12:442–444.
34. Young-Hyman DL, Davis CL. Disordered eating behavior in individuals with diabetes: importance of context, evaluation, and classification. *Diabetes Care*. 2010;33:683–689.
35. Russel-Jones D, Khan R. Insulin-associated weight gain in diabetes-causes, effects and coping strategies. *Diabetes Obes Metab*. 2007;9:799–812.