

Disturbed eating behaviors in adolescents and emerging adults with type 1 diabetes:

A one-year prospective study

Short title: Disturbed eating behaviors in type 1 diabetes

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Abstract

Objective. Disturbed eating behaviors (DEB) are prevalent in youth with type 1 diabetes and are accompanied by an increased risk for complications, morbidity, and mortality. Prospective studies on DEB in the challenging transition to adulthood are scarce. This longitudinal study examined DEB over a 1-year period and investigated the directionality of effects linking DEB to diabetes-specific functioning and depressive symptoms in adolescents and emerging adults.

Research design and methods. 300 youth (14-25 years) with type 1 diabetes participated in a two-wave longitudinal study. Questionnaires measured DEB (Diabetes Eating Problem Survey-Revised; DEPS-R), self-management, diabetes distress, and depressive symptoms. HbA_{1c} values were obtained from physicians. Mixed analysis of variance and cross-lagged analysis were used to examine prospective changes and directionality of effects, respectively.

Results. Mean DEB remained stable in the total sample but significant individual differences were observed based on the cut-off score of the DEPS-R: 19% displayed persistent DEB, 8% increased, and 7.3% decreased in DEB over time. The remaining individuals scored low on DEB over time. These four groups were differentiated based on insulin restriction, omission, diabetes-specific functioning, and depressive symptoms. Cross-lagged analyses indicated that DEB predicted relative increases in depressive symptoms over time, whereas reciprocal associations with glycemic control were found.

Conclusions. This longitudinal study highlights the substantial impact DEB may have in the transition to adulthood, with a substantial portion of youth with type 1 diabetes being at risk for clinical DEB. Prospective pathways linking DEB to functioning were found, emphasizing the clinical relevance of assessing DEB over time.

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Type 1 diabetes constitutes a risk factor for developing disturbed eating behaviors (DEB), including unhealthy weight-management behaviors such as skipping meals, binge eating, and purging, as well as more severe eating disorders (1,2). DEB constitutes a substantial concern as up to 30% of adolescent girls with type 1 diabetes display DEB (3-5), many of whom are at risk for developing eating disorders (6,7). The magnitude of this problem in boys seems less extensive but still troublesome (2), with up to 10% reporting substantial DEB (4). Further, youth with type 1 diabetes may turn to insulin restriction or omission for manipulating their body weight, resulting in rapid weight loss (8,9). This phenomenon is quite prevalent as 20-40% of youth (and especially females; 2,10) engage in it (8,11,12), leading to long-term insulin misuse, morbidity, and even mortality (13,14). Consequently, a need for more attention to DEB in youth with type 1 diabetes has recently been highlighted as an important clinical guideline (15,16).

The strong focus on food in relation to blood glucose regulation and the dietary restraints possibly playing into perfectionistic attitudes may be causing DEB in type 1 diabetes (7,8,11,17-19). Given the societal pressure to obtain the body perfect ideal (20), especially adolescents seem to be at risk for developing DEB, as they are particularly vulnerable for body dissatisfaction (18,21). Meta-analysis has demonstrated that eating disorders and DEB (even at a subclinical level; 17,22) are associated with higher HbA1c-values and premature diabetes complications (4,23). In sum, DEB are of particular concern in youth with type 1 diabetes due to their health-compromising consequences (19).

Longitudinal studies that examine how DEB are associated with diabetes-specific functioning and depressive symptoms over time are limited, especially in the challenging transition to adulthood. Research has demonstrated that DEB tended to increase during this transition (10), necessitating a detailed examination of prospective implications toward youth

functioning. To inform clinical practice and increase our knowledge of DEB in type 1 diabetes, the present study addressed three innovative research objectives: (1) to examine DEB in adolescents and emerging adults over a time-span of 1 year (with a focus on gender differences); (2) to identify subgroups based on DEB (e.g., individuals displaying substantial vs. little DEB over time) and how these subgroups differ on demographic and clinical variables, diabetes-specific functioning, and depressive symptoms; and (3) to investigate the directionality of effects linking DEB to self-management, glycemic control, diabetes distress, and depressive symptoms over time.

In addressing these objectives, we made use of a diabetes-specific measure, the Diabetes Eating Problem Survey-Revised (DEPS-R; 24). Generic measures assessing DEB may not be appropriate (4,24), as such generic measures assess certain behaviors that are part of standard diabetes care (e.g., restricting the intake of carbohydrates). Such items may be misinterpreted by people with diabetes (12) and could lead to incorrect prevalence estimates (4,20). Further, the DEPS-R assesses insulin restriction and omission, allowing us to examine these behaviors in this prospective study.

Research Design and Methods

Participants and Procedure

Data were used from a larger longitudinal project in collaboration with the Belgian Diabetes Registry (25). Ethical approval was provided by the Institutional Review Board at University of Leuven. Dutch-speaking youth (14-25 years) with type 1 diabetes and without cognitive impairment completed questionnaires at home, resulting in four data-waves with 1-year time intervals. At T(ime) 1, questionnaires, informed consent forms, and stamped return envelopes were sent to 1,459 individuals. For minors, parents were provided with consent forms. 571 bundles were returned, of which 559 cases were eligible for analysis, given that no parental consent was obtained for 12 minors ($RR=40\%$). At T2, T3, and T4, these 559

individuals were invited to participate again, resulting in response rates of 422 (75%) at T2, 381 (68%) at T3, and 324 (58%) at T4. As DEB were assessed only at T3-T4, data were used from individuals participating at T3 and T4, totaling 300 individuals. These 300 individuals did not differ on gender, age, illness duration, and diabetes distress but had somewhat lower scores on depressive symptoms ($F(1,498)=5.545$; $p=.019$) and higher scores on self-management ($F(1,498)=10.764$; $p=.001$) as compared to the 259 individuals not included. For analyses involving glycemic control, we limited our analyses to individuals of whom we could obtain HbA_{1C}-values at T3 or T4 ($N=231$). For reading convenience, T3 and T4 will be referred to as T1 and T2.

Questionnaires

Disturbed eating behaviors. The DEPS-R (8,24) includes 16 items. Following Eilander et al. (8), the item referring to ketones was excluded as not all Belgian youth are familiar with the exact meaning of ketones. Items are answered on a 6-point scale ranging from 0 (*never*) to 5 (*always*) and address general (e.g., “I feel that my eating is out of control”) and diabetes-specific DEB in terms of insulin restriction and omission (e.g., “After I overeat, I skip my next insulin dose”). Total sum scores could range between 0-75; higher scores point to more DEB. Cronbach’s alpha was .87 at T1 and .86 at T2.

Self-management. The 14-item Self-Care Inventory (SCI; 25,26) was used. The SCI includes items that focus on blood glucose testing and monitoring, insulin and food regulation, exercise, and emergency precautions. Item 12 “Wearing a medic alert ID” was deleted, as this is not always part of standard treatment in Belgium. Individuals had to indicate how well they followed their diabetes regimen in the past month on a 5-point scale, ranging from 1 (*never do it*) to 5 (*always do this as recommended without fail*). A mean score was calculated with higher scores indicating better self-management. Cronbach’s alpha was .78 at T1 and T2.

Diabetes distress. Diabetes distress was assessed with the Problem Areas in Diabetes Scale (PAID; 27), measuring diabetes-related problems in four domains (emotions, food, self-management, social support) by means of 20 items to be rated on a 5-point scale ranging from 0 (*not a problem*) to 4 (*a serious problem*). A total score was calculated as the mean of the four domains; higher scores indicate more diabetes distress. Cronbach's alphas was .95 at T1 and T2.

Depressive symptoms. Depressive symptoms were measured by the 20-item Center for Epidemiologic Studies Depression Scale (CESD; 28). Each item asks how often participants had experienced depressive symptoms during the past week, using a 4-point scale from 0 (*seldom*) to 3 (*most of the time or always*). A total score was calculated (ranging from 0 to 60); higher scores indicate more depressive symptoms. Cronbach's alpha was .94 at T1 and .92 at T2.

Glycemic control. HbA1c-values that were closest to the date participants filled out the questionnaires (in a time-window of 3 months before or after completion of the questionnaires) were collected from medical records by contacting treating physicians. HbA1c-values were converted from DCCT-derived units (%) to IFCC-recommended units (mmol/mol). HbA1c-values below 7.0% or 53 mmol/mol are recommended (29).

Statistical Analyses

First, as a set of preliminary analyses, Pearson correlations were calculated. Second, to examine the prospective follow-up of DEB (Objective 1), mixed analysis of variance (ANOVA) was conducted with time as within-subjects factor, gender as between-subjects factor, and DEB at T1 and T2 as dependent variable. The interaction effect (TimeXGender) was entered to examine differential change over time for males versus females.

Third, based on the cut-off of 18 on the DEPS-R (suggesting DEB warranting further attention from clinicians; 4,8), four groups capturing mean-level change and stability were

created (Objective 2): *Low DEB* (<18 at T1 and T2); *Increasing DEB* (<18 at T1 and \geq 18 at T2); *Decreasing DEB* (\geq 18 at T1 and <18 at T2); and *Persistent DEB* (\geq 18 at T1 and T2). Multivariate analyses of variance (MANOVA with post-hoc Tukey HSD comparisons) and χ^2 -cross-tabulation were used to examine group differences on demographic variables (age, gender), clinical variables (illness duration, injection vs. pump therapy, Body Mass Index [BMI], glycemic control), self-management, diabetes distress, and depressive symptoms at T1 and T2. Further, mixed ANOVAs were used to investigate TimeXGroup interaction effects, possibly revealing different mean-level changes for glycemic control, self-management, diabetes distress, and depressive symptoms in the four DEB-groups. The model consisted of one within-subjects (time) and one between-subjects factor (DEB-group). Dependent variables were repeated measures of glycemic control, self-management, diabetes distress, and depressive symptoms.

Fourth, to examine the directionality of effects linking DEB to these variables (Objective 3), cross-lagged analyses were applied using Structural Equation Modeling in Mplus7 (30). Models were estimated using maximum likelihood with robust standard errors (MLR) to account for non-normality. Full information maximum likelihood was used to handle occasional missing data. In all models, within-time associations at T1 and T2 and auto-regressive coefficients were included when examining cross-lagged paths. Cross-lagged paths inform us as to whether variable X at T1 predicts relative or rank-order changes in variable Y at T2, controlling for all within-time associations and auto-regressive coefficients. All significant associations with clinical and demographic variables (gender, age, type of insulin therapy, illness duration) were controlled for by regressing variables at T1 and/or T2 on these variables at T1. We used standard fit indices. The Satorra-Bentler scaled (SBS)- χ^2 should be as small as possible; Root Mean Square Error of Approximation (RMSEA) should be less than .06; Comparative Fit Index (CFI) should exceed .95.

Results

Patient Characteristics

Table 1 summarizes patient characteristics at T1 for the full sample ($N=300$) and the restricted sample, excluding individuals without HbA_{1C}-values at T1 or T2 ($N=231$). These 231 individuals differed from those without HbA_{1C}-values at T1 or T2 ($N=69$) on age ($F(1,290)=6.341$; $p=.012$) and illness duration ($F(1,290)=24.006$; $p<.001$) at T1 (with individuals without HbA_{1C}-values being older and having a longer illness duration), but not on gender, BMI, DEB, diabetes distress, self-management, and depressive symptoms. At T1, 83% of the full sample were living with their parent(s) and 61.3% had a college/university degree or were currently in college/university. Mean HbA_{1c} in the restricted sample was 7.42 ($SD=0.95$) or 58 mmol/mol ($SD=10.4$).

Preliminary Correlational Analyses

As displayed in Table 2, DEB at T1 and T2 was positively related to BMI at T1 and T2 but unrelated to age and illness duration. Further, DEB at T1 and T2 was positively related to diabetes distress, depressive symptoms, and HbA_{1c}, and negatively to self-management at T1 and T2.

Objective 1: Prospective follow-up of DEB

Mixed ANOVA indicated that mean DEB remained stable ($F(1,294)=0.714$; $p=.399$) from T1 ($M=13.15$; $SD=10.43$) to T2 ($M=12.71$; $SD=9.87$). The TimeXGender interaction was not significant ($F(1,294)=2.078$; $p=.150$). Both males and females remained stable over time, with females (T1: $M=16.53$, $SD=11.34$; T2: $M=15.57$, $SD=10.49$) scoring substantially higher on DEB ($F(1,294)=48.661$; $p<.001$; $\eta^2=.142$) than males (T1: $M=8.71$, $SD=6.97$; T2: $M=8.96$, $SD=7.51$).

Objective 2: Identifying Four Groups of DEB

79 individuals (26.3%) at T1 and 81 individuals (27%) at T2 had a score of ≥ 18 on DEPS-R. Across T1-T2, 197 individuals (65.7%) were in the Low DEB-group (range DEPS-R scores between 0-17 at T1 and T2), 24 (8%) in the Increasing DEB-group (range DEPS-R scores between 3-17 at T1 and 18-40 at T2), 22 (7.3%) in the Decreasing DEB-group (range DEPS-R scores between 18-45 at T1 and 4-17 at T2), and 57 (19%) in the Persistent DEB-group (range DEPS-R scores between 18-59 at T1 and 18-50 at T2). Mean DEPS-R scores for these groups are displayed in Table 3. Cross-tabulation indicated that males and females were differentially distributed across these groups ($\chi^2(3)=27.028$; $p<.001$). Standardized residuals indicated that males were relatively (as compared to the total gender distribution) overrepresented in Low DEB (52.3% males vs. 47.7% females), whereas females were overrepresented in Persistent DEB (14.3% males vs. 85.7% females) and (less so) in Increasing DEB (31.8% males vs. 68.2% females).

MANOVA pointed to differences on BMI at T1 and T2. Univariate post-hoc comparisons (see Table 3) indicated that, as expected, at T1, Low DEB and Increasing DEB scored lowest on BMI, whereas, at T2, only Low DEB scored lowest (with Increasing DEB not differing from other groups). No group differences were found on type of insulin therapy (injection vs. pump) ($\chi^2(3)=1.942$; $p=.585$), age ($F(3,294)=0.124$; $p=.946$), or illness duration ($F(3,294)=0.286$; $p=.836$).

When exploring the two DEPS-R items assessing insulin restriction at T1, 63.7% indicated that they never restricted insulin, 21% almost never, and 15.3% at least sometimes. For insulin omission at T1, these percentages were 86.3%, 9.3%, and 4.3%, respectively. At T2, these percentages for insulin restriction were 71.7%, 18.0%, and 10.3%, and for insulin omission 87.7%, 8.7%, and 3.7%. There were no gender differences for insulin restriction or omission T1-T2 ($\chi^2(2)=1.524 - 5.062$; $ps=.080 - .467$). Further, insulin restriction and omission at T1 and T2 were differentially distributed across the DEB-groups ($\chi^2(6)=36.241 - 61.926$;

$p < .001$). All percentages can be found in Supplemental Table S1. For insulin restriction T1, 31.8% of Decreasing DEB and 36.8% of Persistent DEB indicated at least sometimes, whereas 8.1% of Low DEB did so. For insulin omission, these percentages were 18.2%, 10.5%, and 0.5%, respectively. For insulin restriction T2, 33.3% of Increasing DEB and 22.8% of Persistent DEB indicated at least sometimes, whereas only 4.1% of Low DEB did so. For insulin omission, these percentages were 20.8%, 8.8%, and 0.5%.

Objective 2: Linking the Four DEB-Groups to Diabetes-Specific Functioning and Depressive Symptoms

As expected, Table 3 shows that Low DEB scored lowest on diabetes distress, depressive symptoms, and HbA_{1C}, and highest on self-management at T1 and T2. Especially Persistent DEB had the least adaptive scores on these variables at T1 and T2. At T1, Increasing DEB scored higher on depressive symptoms and HbA_{1c} as compared to Low DEB, and lower on depressive symptoms and higher on self-management as compared to Persistent DEB. Decreasing DEB scored higher on diabetes distress and depressive symptoms and lower on self-management as compared to Low DEB. Decreasing DEB did not differ significantly on any variable from Persistent DEB at T1. At T2, Increasing DEB scored higher on diabetes distress, depressive symptoms, and HbA_{1C} as compared to Low DEB, whereas Decreasing DEB scored higher on depressive symptoms as compared to Low DEB. Decreasing DEB scored lower on diabetes distress as compared to persistent DEB. Increasing DEB did not differ significantly on any variable from Persistent DEB at T2.

Finally, mixed ANOVAs with TimeXGroup interactions revealed a significant interaction for self-management ($F(3,296)=9.174$; $p < .001$; $\eta^2=.085$). Whereas Low DEB and Persistent DEB remained fairly stable over time, Increasing DEB decreased and Decreasing DEB increased on self-management (mean values at T1-T2 are displayed in Table 3). No significant TimeXGroup interactions were found for diabetes distress ($F(3,296)=1.593$;

$p=.191$), depressive symptoms ($F(3,296)=0.431$; $p=.731$), and HbA_{1c} ($F(3,227)=0.821$; $p=.483$).

Objective 3: Directionality of Effects

Cross-lagged analyses indicated that all final models in which non-significant paths from control variables were trimmed had a good fit (range of indices: $df=2-6$; SBS- $\chi^2=3.674-5.144$ ($p=.133-.552$); RMSEA=.000-.059; CFI=.994-1.000). In all models, gender at T1 (0=male; 1=female) positively predicted DEB (β s ranging between .35 and .37; $p<.001$), diabetes distress ($\beta=.19$; $p<.001$), and depressive symptoms ($\beta=.21$; $p<.001$) at T1, and marginally negatively self-management ($\beta=-.11$; $p=.056$) at T1. Further, age at T1 negatively predicted self-management ($\beta=-.14$; $p=.010$) and HbA_{1c} ($\beta=-.14$; $p=.029$) at T1. With respect to cross-lagged associations (see Figure 1), DEB at T1 predicted relative increases in HbA_{1c} ($\beta=.12$; $p=.043$) and (marginally so) depressive symptoms ($\beta=.10$; $p=.066$). Reverse paths were significant for glycemic control, indicating that HbA_{1c} at T1 predicted relative increases in DEB ($\beta=.16$; $p=.001$). In the models of diabetes distress and self-management, no significant cross-lagged associations were found, with β s ranging between $-.07$ and $.06$ ($ps=.198-.857$).

Conclusions

The present study emphasized the need for a prospective follow-up of DEB in adolescents and emerging adults with type 1 diabetes. DEB were found to be unrelated to age and illness duration, indicating that DEB occurred at any age and at any stage in the illness trajectory. However, previous research indicated that, during adolescence, DEB was most prevalent in 17-19 year olds (4), and even higher prevalence rates have been found in emerging adulthood (10). Although no such age trends were found in the present study, findings again highlighted the substantial levels of DEB in the transition to adulthood. Similar to previous studies (4,19), especially individuals with a higher BMI were at risk for DEB. As the transition

to adulthood is characterized by an increasing concern about weight and body image, an increase in BMI may play into body dissatisfaction and DEB (10,18,20).

With respect to gender, especially females were at risk for DEB. This finding has been replicated repeatedly and signals certain vulnerabilities girls and women may experience, such as puberty-related increases in BMI, appearance-related conversations (so-called ‘fat talk’) in peer groups, and more weight concern and body image dissatisfaction (3,11,14). However, when looking at items assessing insulin restriction and omission, no significant gender differences emerged: males seemed to manipulate insulin administration at comparable rates as females. Previous research, however, indicated that especially females were at risk for insulin manipulation (2,10). As a possible explanation for these diverging findings, the DEPS-R items measuring insulin restriction and omission do not specify that such behaviors target weight loss. Other reasons may play into insulin administration, such as fear of hypoglycemia, negative affect toward injections, or interference with daily activities (4). Future research should examine such underlying motives to identify why male and female youth engage in insulin manipulation. Despite potential differences in these motives, the high occurrence of insulin restriction and omission found in the present study highlights the need for explicit attention to DEB in male youth as well, as DEB often go undetected in boys and men (21).

Extending previous research assessing DEB in this population, substantial individual differences over time were observed when using the cut-off score of 18 on the DEPS-R (8). Although this cut-off is not diagnostic, it signals individuals at-risk in need of further evaluation (4). Previous cross-sectional research indicated that about 15-18% of adolescents with type 1 diabetes scored above this cut-off (4,21). In the present study, 34.3% scored above this cut-off at either T1 or T2 or at both time-points: 19% scored above the cut-off at both time-points (Persistent DEB), 8% developed substantial DEB in-between T1 and T2 (Increasing DEB), and 7.3% seemed to recover from engaging in substantial DEB in-between T1 and T2 (Decreasing

DEB). When focusing on self-reported insulin omission and restriction, scores on these items seemed to signal the severity of DEB in the four groups (18). For instance, in the Low DEB-group, only a small minority had engaged in insulin restriction and virtually no one in insulin omission, whereas these percentages were substantially higher in the other DEB-groups.

The four DEB-groups were differentiated on diabetes-specific functioning and depressive symptoms as well. Overall, youth in Low DEB had the most adaptive profile, whereas youth in Persistent DEB showed the least adaptive profile in terms of glycemic control, self-management, diabetes distress, and depressive symptoms. As expected, findings for Increasing DEB and Decreasing DEB were dependent on the time-point at which diabetes-specific functioning and depressive symptoms were assessed. For instance, self-management seemed to co-develop with DEB over time, with self-management decreasing over time when DEBs increased, and vice versa. Such individual differences highlight the need for assessing DEB prospectively.

Finally, the cross-lagged findings substantially extended previous research and demonstrated that DEB (marginally) predicted relative increases in depressive symptoms over time. This prospective link with depressive symptoms merits special attention given the associations of depressive symptoms with worse self-management, glycemic control, and increased health care costs (31,32). Further, DEB and higher HbA1c-values were found to reinforce one another over time. The finding that DEB may contribute to higher HbA1c-values has been repeatedly forwarded and testifies to the adverse long-term consequences of DEB, necessitating early identification and intervention (4,9). However, the reverse pathway from glycemic control to DEB has not been documented systematically. Previous research indicated that higher HbA1c-values may play into the use of maladaptive coping strategies over time (33,34). Similarly, higher HbA1c-values may instigate some individuals to regulate their food intake more strictly, even to the point where such regulation turns into DEB (18). Other

individuals may become de-motivated by having higher HbA1c-values, shifting their attention away from a healthy diet and adaptive eating patterns. Executive function may also be an important variable in this respect, as lower executive function has been related to higher HbA1c-values and DEB in youth with type 1 diabetes (35,36). Hence, future research should examine this co-development of DEB and glycemic control with a specific focus on reciprocal pathways and mechanisms, paying attention not only to the psychological but also the physiological level (e.g., disrupted hunger and satiety sensations due to glycemic dysregulation; 11,14).

Limitations and Suggestions for Future Research

The present study had certain limitations. First, although the study was prospective, future research should assess these variables over longer periods using multiple assessments to examine developmental trajectories into adulthood. Intensive, short-term diary studies should be set up to identify possible mechanisms and processes playing into DEB. Second, no information was available as to whether youth received specific care in-between T1-T2 for DEB. Hence, it is difficult to know why some individuals displayed more changes in DEB as compared to others. Relatedly, data on hospital admission rates related to DEB were not available, although they could have informed us about the clinical relevance of our findings. Third, the sample was quite homogenous in nature, as all participants were sampled from the Belgian Diabetes Registry, spoke Dutch, and most of them were highly educated. Although our longitudinal sample was quite large, the generalizability of findings may be reduced as the final response rate (compared to the initial group that was invited) was relatively low. Given the relatively low mean HbA1c-value obtained for the present sample and given that the maximum score observed on the DEPS-R was 59 (on a scale from 0-75), our final sample could represent a selective, relatively well-adjusted group. Also in terms of depressive symptoms and self-management, the highest risk participants may not have been captured in our final sample.

Fourth, study variables were measured through self-reports (except for glycemic control), possibly confounding the results due to shared method variance. Relatedly, although the DEPS-R is a promising instrument for assessing DEB in individuals with diabetes, its sensitivity and specificity need further exploration (8). Finally, although HbA1c-values were obtained for the majority of participants, these values could not be obtained for participants without a medical visit during the study window. Moreover, there was a time window of three months in-between filling out the questionnaires and the measurement of HbA1c. Ideally, this measurement needs to be done at the same time as questionnaire completion.

Despite these limitations, the present findings may inform clinical practice. Given the prospective changes observed, it may be valuable to integrate the assessment of DEB and eating disorders in the vulnerable age group of adolescence and emerging adulthood into routine clinical care. DEB can emerge at any time during the illness trajectory and, for some individuals, the severity of DEB may change substantially over time. Further, not only do the dietary aspects of diabetes self-management make it more difficult to identify DEB (12,37), youth may also experience shame and guilt as a barrier for disclosing DEB (21,38). It may be helpful for clinicians to ask certain screening questions regarding weight concerns and behaviors at a regular basis in a supportive and non-judgmental way (19,21). Cost- and time-effective measures such as the DEPS-R could be useful (12,21,24). As demonstrated in this study, attention should not only be paid to clinically elevated levels of DEB. Subclinical levels of DEB may also be an important warning sign as they can turn into problematic DEB (as seen in the Increasing DEB-group). Our cross-lagged analyses indeed demonstrated the predictive power of DEB across the whole spectrum of severity. Hence, in case of such warning signs, further assessment of DEB could be performed by trained mental health professionals (8). Ideally, all members of the multidisciplinary team (e.g., physicians, nurses, dieticians) would be provided with the opportunity to acquire the necessary skills to recognize warning signs of

DEB (14). Without any intervention, DEB and insulin manipulation may worsen and feed into substantial complications over time, given their prospective link with higher HbA1c-values (5,39). Diabetes management of youth with DEB can only improve until appropriate treatment begins for DEB (38,40).

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Table 1

Patient Characteristics at T1

	Patients participating at T1 and T2 (N=300)	Patients with HbA _{1c} -values at T1 and/or T2 (N=231)
Sex		
Males	43.2%	44.7%
Females	56.8%	55.3%
Age ^a	20.80 (3.31)	20.55 (3.46)
Illness duration ^a	7.56 (4.98)	6.89 (4.74)
Insulin administration		
Injection	75.2%	75.7%
Pump	24.8%	24.3%
Civil status (<i>more than 1 option</i>)		
Single	41.1%	43.9%
Living with partner/(re)married	14.8%	13.6%
Relationship (living separately)	35.0%	32.5%
Other	8.8%	9.6%
Work (<i>more than 1 option</i>)		
Student	66.1%	68.6%
Working	35.7%	32.9%
Unemployed	2.0%	1.7%
Other	2.7%	3.5%

^a Mean values with *SD* in parentheses.

Table 2

Pearson Correlations Among the Study Variables at T1 and T2

	1.	2.	3.	4.	5.	6.	7.	8.
1. Age	--	.15*	.13*	-.05	.04	-.04	-.12*	-.16*
2. Illness duration	.13*	--	.10	.04	-.01	.05	-.03	.08
3. BMI	.15**	.12*	--	.36***	.02	.08	-.08	.10
4. DEB	-.00	.01	.39***	--	.52***	.42***	-.39***	.35***
5. Diabetes distress	.05	.06	.10	.42***	--	.51***	-.31***	.24***
6. Depressive symptoms	.06	.05	.08	.39***	.40***	--	-.23***	.17**
7. Self-management	-.12*	-.05	-.17**	-.41***	-.26***	-.23***	--	-.27***
8. HbA _{1c}	-.13*	.03	.12	.49***	.16*	.16*	-.32***	--

Note. DEB = disturbed eating behaviors; BMI = body mass index. Values above the diagonal represent correlations at T1, below the diagonal correlations at T2. $N=300$ for all correlations, except for the correlations with HbA_{1c} ($N=231$).

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 3

Univariate ANOVA's and Post-hoc Group Comparisons Based Upon Tukey HSD Tests for the Four DEB Groups

Variable	Total sample	DEB groups				F-value	η^2
		Low	Increasing	Decreasing	Persistent		
<i>Time 1</i>							
DEB	13.19 (10.46)	7.59 (4.77) ^a	12.04 (3.85) ^b	22.82 (7.39) ^c	29.30 (8.19) ^d	234.270***	.704
BMI	23.35 (3.59)	22.56 (3.30) ^a	23.12 (2.98) ^a	25.52 (3.28) ^b	25.36 (3.88) ^b	12.675***	.118
Diabetes distress	0.86 (0.72)	0.64 (0.59) ^a	1.05 (0.69) ^b	1.12 (0.69) ^{bc}	1.47 (0.77) ^c	26.446***	.211
Depressive symptoms	12.48 (10.87)	9.66 (8.98) ^a	15.74 (12.03) ^b	18.36 (12.50) ^b	18.58 (12.12) ^b	15.150***	.133
Self-management	3.77 (0.51)	3.86 (0.48) ^b	3.90 (0.46) ^b	3.47 (0.46) ^a	3.54 (0.51) ^a	9.885***	.091
HbA _{1C}	7.42 (0.95)	7.17 (0.73) ^a	8.07 (1.41) ^b	7.68 (0.87)	7.99 (1.12) ^b	13.775***	.150
mmol/mol	58 (10.4)	55 (8.0) ^a	65 (15.4) ^b	60 (9.5)	64 (12.2) ^b	13.775***	.150
<i>Time 2</i>							
DEB	12.80 (9.92)	7.26 (4.54) ^a	24.17 (5.58) ^c	12.59 (2.99) ^b	27.26 (7.81) ^c	248.793***	.716
BMI	23.70 (3.50)	22.85 (3.01) ^a	23.92 (3.46)	25.87 (3.79) ^b	25.79 (3.84) ^b	14.764***	.135
Diabetes distress	0.82 (0.74)	0.63 (0.63) ^a	1.13 (0.81) ^{bc}	0.93 (0.69) ^{ab}	1.35 (0.78) ^c	18.726***	.160
Depressive symptoms	11.55 (9.75)	8.94 (8.26) ^a	14.84 (11.31) ^b	15.45 (9.09) ^b	17.68 (10.60) ^b	16.530***	.143
Self-management	3.78 (0.49)	3.88 (0.47) ^b	3.68 (0.35)	3.72 (0.45)	3.47 (0.50) ^a	12.131***	.109
HbA _{1C}	7.58 (0.95)	7.32 (0.70) ^a	8.34 (1.05) ^c	7.68 (0.90) ^{ab}	8.22 (1.28) ^{bc}	16.184***	.176
mmol/mol	59 (10.4)	56 (7.7) ^a	68 (11.5) ^c	60 (9.8) ^{ab}	66 (14.0) ^{bc}	16.184***	.176

Note. A group mean is significantly different from another mean if they have different superscripts. A mean without a superscript is not

significantly different from any other mean. Standard deviations are in parentheses. $N=300$ for all analyses, except for the analyses with HbA_{1C}

($N=231$). * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 1

Final standardized path coefficients of the cross-lagged models linking DEB to depressive symptoms (panel a) and glycemic control (panel b)

† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.